The epidemiology, politics and control of malaria epidemics in Kenya: 1900-1998

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Acknowledgements & background to the report

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1.1 Defining epidemics

Epidemics invoke fears of exotic and historical diseases, which capture political and global attention. Much of sub-Saharan Africa is exposed to stable, endemic *P. falciparum* transmission leading to high burdens of morbidity and mortality among young children (Murray & Lopez, 1997; Snow et al., 1999). In addition, the continent has witnessed several devastating malaria epidemics: during the early 1930's in South Africa (Le Sueur et al., 1993), 1958 in Ethiopia (Fontaine et al., 1961), and 1986 in Madagascar (Mouchet, 1998). These outbreaks in transmission followed clearly identifiable changes in climate favouring vector and parasite proliferation and were among non-immune populations. Various estimates from these epidemics indicate that between 1% and 14% of the respective populations died.

Just who is exposed to epidemic conditions and how best to define these conditions are much debated and often hard to quantify. Traditional approaches to measuring whether malaria transmission has the potential for epidemics have focused on the bio-mathematical relationships between the parasite and its primary host, the mosquito. These relationships have been quantified as the *Stability Index, Basic Reproduction Rate and Vectorial Capacity.*

Christophers' work on the Punjabi epidemics during the early part of this century were some of the first attempts to derive mathematical estimations of 'epidemic potential' (Anderson & May, 1991). Subsequent models focused on entomological parameters for an index of stability based on the malaria vector's human biting habit and its expectancy of life (MacDonald, 1956):

$$a / \log_e p$$

where "a" is the human biting habit or the average number of human blood meals taken by the female anopheles in a day, $1 / \log_e p$ the life expectancy of the mosquito and "p" is the probability of the vector surviving one day. Values of > 2.5 indicate stability and values of < 0.5 indicate instability.

The Basic Reproduction Rate (R_o) developed by MacDonald includes duration of sporogony, vector density and duration of infectivity in man. It is therefore a better indicator of stability. The R_o of a parasite is defined as the average number of successful offspring that it is intrinsically capable of producing (Anderson & May, 1991). It is expressed as the average number of secondary infections produced from one infected individual introduced into a non-immune host population. For the parasite to survive successfully R_o must be greater than 1 and transmission becomes unstable when R_o goes below unity. R_o combines measures of mosquito infectivity and survival and is calculated using the following formula:

$$R_o = p^n / p^n - s$$

where 'p' is the probability of the mosquito surviving through one day, n is the incubation period to infectivity in the mosquito and 's,' the proportion of mosquitoes that are sporozoite positive.

The Vectorial Capacity, a mathematical expression of risk of transmission, also includes anopheline density as a risk factor. It is defined mathematically as:

$$C = \frac{ma^2p^n}{-\log_e p}$$

where ‘m’ is the relative density of female anophelines, ‘a’ the probability that the mosquito will take a human blood meal during a particular day and ‘p^n’ the proportion of vectors surviving the parasites incubation period (i.e. p - the probability of vector survival and n – the number of days the vector lives). The probability of daily survival is key in determining endemicity levels. For *Anopheles gambiae* and *Anopheles funestus* an average daily survival rate of >60% has been shown to be associated with stable endemicity (Wernsdorfer & McGregor, 1988). The Vectorial Capacity is thus a reflection of the mean number of probable inoculations transmitted from one case of malaria in a unit of time and consequently linked to whether transmission in a given area is stable or unstable.

Climatic factors which influence the parameters defined in the formulae presented above for stability and transmission potential have been used in a series of recent models to define the limits of *P.falciparum* in Africa (Lindsay et al., 1998; Lindsay and Martens, 1998; Craig et al., 1999). These models have provided a series of observations which define geographical areas which constitute low risks of stable endemicity and consequently areas of increased risks of unstable malaria. Epidemic risks, however, may not always coincide with areas defined as unsuitable for stable transmission, or areas of unstable malaria do not always lead to epidemics. These issues are described for Kenya in Chapter 2.

We often classify areas according to whether transmission is stable or unstable. These two extremes are end points in a continuum of differing epidemiological scenarios. The term ‘stable’ implies equilibrium. Despite the mathematical simplicity of these concepts the formulae presented above do not provide us with any insights into their effects upon human populations. On the whole where malaria is stable, the prevalence of infection is persistently high and endemicity is relatively insensitive to environmental changes (Molineux, 1988). Variation in transmission is minimal over many years although seasonal fluctuations may occur and transmission can continue even with very few vectors. High levels of immunity develop within the population due to regular and often continuos transmission (MacDonald, 1956). Unstable malaria on the other hand is characterised by great variability in space and time. Collective immunity is low and there is a propensity for epidemics to occur. The disease is also characterised by recession and recurrence and periods when disease incidence is low alternating irregularly with high incidence periods (Warrell & Gilles, 1993).

MacDonald (1957) describes an epidemic as follows

> *“An epidemic is an acute exacerbation of disease out of proportion to the normal to which the community is subject. There is a proposal to restrict the term to the narrower sense of outbreaks in places where the disease is rare, but the writer [MacDonald] has found this a restricted definition unworkable in practice and prefers the wider and more colloquial term… Epidemics occur only in zones of unstable malaria, where very slight modification in any of the transmission factors may completely upset equilibrium, and where restraining influence of immunity may be negligible or absent, and they therefore show a very marked geographical distribution”.*

Many factors can influence the ability of parasites and vectors to coexist long enough to result in continued transmission. Several reviews have described the effects on transmission of environmental change, changes in agriculture and forestry practices and man-made construction (Hackett, 1949; Lindsay & Birley, 1996; Lindsay & Martens, 1998; Mouchet et

al., 1998). This report will not re-review these particular influences on malaria transmission. However, commentary will be made on the likely significance of small changes in the equilibrium of environmental determinants within the fragile conditions of unstable areas of Kenya.

The main aim of classifying malaria endemicity is to facilitate control and these classifications need to group epidemiological conditions according to common sensitivity to different control options. This review for Kenya will identify some of the weaknesses in our classical approaches to defining stability, epidemics and the need for a review of our classification systems to provide more appropriate public health tools.

1.2. The scope of the present report

Kenya provides an example of how extreme epidemiological patterns of *P.falciparum* transmission can coexist within a single national boundary. Kenya also has a well documented history of malaria encounters since 1900 across the country co-incidental with the rapid development of colonial, economic expansion and exploitation. We have used a series of archival material held by the Kenya National Archives to understand the clinical and political significance of epidemics between 1900 and 1970. Newspaper articles have provided the popular press's perception of epidemics and how these reports influenced Ministry of Health responses. The old, and often forgotten, literature from local and international journals have been sought to provide quantifiable descriptions of malaria epidemics as they affected different parts and different sectors of Kenya's changing society. And, finally we have visited 7 areas of Kenya affected by the unstable *P.falciparum* transmission to derive hospital admission data to express the temporal and secular patterns of clinically complicated malaria in each area. Combined, these reports, data and anecdotes have been used to build a picture of how epidemics have affected Kenya's population over this century. The epidemiology, previous attempts at control and their role within a changing health sector have allowed a series of observations and recommendations which should have significance for not only Kenya but those countries in the sub-region with similar ecologies.

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2. The distribution of epidemic malaria in Kenya with special reference to clinical patterns.

2.1. Overview of Kenya's topography and population.

Kenya covers an area of 582,000 square kilometres. It borders Ethiopia in the north, Sudan in the northwest, Uganda on the west, Tanzania in the south and Somalia in the east. It has 400 kilometres of Indian ocean shoreline. Lying between 3 degrees north and 5 degrees south latitude between 34 and 41 degrees east longitude, it is entirely within the equatorial zone and almost bisected by the equator. The country falls into two distinct regions, i.e., lowland and highland (uplands). This distinction affects the climate, patterns of human settlement and agricultural activities. Kenya has an unusually diversified physical environment - savannah, tropical, equatorial volcanic and tectonic. Approximately 80% of Kenya's land is arid and semi-arid and only 20% is arable. A large part of the arid and semi-arid zones have been set aside for wildlife conservation. Only 1.9% of the total surface area is occupied by standing water. The topography is varied ranging from sea level to over 5,500 metres. Combined, the physical environment varies from equatorial or desert to almost polar situations in the highlands (Ojany & Ogendo, 1988).

Kenya's national administrative boundaries (Figure 2.1) were derived for purposes of local government, resource allocation (including education and health) and population censuses. The main units form a five-tiered system from province (8) as the first level; gazetted districts (47); divisions; locations; and ultimately the smallest unit the sub-location. The boundaries are reviewed every 8-10 years and updated for the purposes of national censuses and local government although these changes more often involve merging or separating existing smaller units. Fifteen new districts have been formed since the last national census in 1989 (Koibatek, Keyio, Marakwet, Trans Mara, Mt. Elgon, Teso, Malava / Lugari, Butere/ Mumias, Rachuonyo, Suba, Kuria, Mwingi, Mbeere, Moyale and Thika) making a total of 64 districts. Although these new districts were not gazetted at the time of this report, topographical maps are available for each and these have been used to update the digital administrative boundary map of Kenya.

There are 43 ethno-linguistic groups in Kenya. The major groups are Kikuyu, Luo, Luhya, Kamba, Kalenjin, Mijikenda, Meru, Embu and Kisii. Kikuyus primarily live in central province, Luos inhabit the western part of Nyanza Province, Luhyas, live in Western Province, Kambas in the southern part of Eastern Province, Kalenjin in the Rift Valley Province, Mijikendas in the Coast Province, Merus in the northern part of Eastern Province and Kisiis in the eastern part of Nyanza Province. Christianity and Islam are the major religions although a significant proportion of Kenya's rural population continue to adhere simultaneously to traditional belief systems.

The enumerated population in 1989 was 21,495,716, a change of about 40% over the 1979 enumerated population (Government of Kenya, 1996a). It is estimated that the census in 1989 probably under-estimated the population by 7%. The average national inter-censal growth rate was about 3.4% per annum. The projected estimate of the population for 1997 is 28,311,112. Approximately 48% of the population are below 15 years of age. However, the population pyramids of Central Province and the districts of Nyeri, Murang'a and Kirinyaga suggest a major change in fertility rates. The evidence of a declining fertility is pronounced and it is expected to continue to decline, but the pace of the decline is much debated. Consequently the best guesses on changing fertility and expected impact of AIDs has led to population projections by the year 2010 of between 35.5 million to 42.9 million (Government of Kenya, 1996a).

Population density varies from 2 persons per Sq. Km. in Wajir and Marsabit districts to 2,469 in Nairobi. The projected population density distribution in Kenya during 1997 is shown in Figure 2.2. The total urban population was about 18% of the total population in 1989. 61% of the urban population reside in the 6 major centres (Nairobi, Mombasa, Kisumu, Nakuru, Machakos, and Eldoret). Nairobi alone accounts for 34% of the urban population. Population migration and mobility is high and 6% of the enumerated population in 1989 were found in districts different to their residence in 1988. Urban populations have increased from 2.3 million in 1979 to 3.9 million in 1989 (Government of Kenya, 1996b).

2.2. Climate and altitude determinants of malaria transmission in Kenya

There are seven principal climatic regions in Kenya (Onany & Ogendo, 1988):

1. *Modified Equatorial Climate of the Coast*: A very short or no dry season but two main peaks in rainfall (May and October) with high average temperatures and humidity throughout the year.

2. *Modified Tropical Climate of the Kenya Highlands*: High rainfall with two almost indistinguishable peaks and cool temperatures throughout the year.

3. *Modified Equatorial Climate of the Lake Victoria Basin*: No dry month and consistently high temperatures. Annual rainfall precipitation is often the highest in Kenya.

4. *Modified Equatorial Climate of the North Western Border*: An extension of the climate patterns of Eastern and Northern Uganda where in several areas higher altitude introduce a cooler temperature and rainfall patterns are more uni-modal with a peak between May and July.

5. *Tropical Climate of Narok and Southern Taita and Kwale areas*: Typical of mainland Tanzania with hills and mountains providing areas of cool temperatures and bi-modal rainfall peaks.

6. *Tropical Continental/semi-desert climate of Eastern Kenya*: This vast region receives less than 500 mm of annual rainfall and mean ambient temperatures are between 22-27^{o}C.

7. *Desert Climate of Central Northern Kenya*: This area traditionally receives less than 250mm of rainfall each year and is regarded as true desert. For example, between 1931 and 1960 Wajir had a mean annual precipitation of only 237 mm.

These variations in climate impact upon the distribution of malaria vectors. The effects of temperature on the transmission cycle are manifold, but its specific effect on sporogonic duration and mosquito survival is the most important (Onori & Grab 1980). The relationships are shown in Table 2.1. At the lower end of the scale the limiting factor is clearly the combined effect of n and p: below 18^{o}C transmission is unlikely because few adult mosquitoes (0.28%) survive the 56 days required for sporogony at that temperature, and also because mosquito abundance is limited by long larval duration. At 22^{o}C sporogony is completed in less than three weeks and mosquito survival is sufficiently high (15%) for the transmission cycle to be completed. So temperature below 18^{o}C is generally considered unsuitable, whilst above 22^{o}C conditions are suitable for stable transmission.

At the upper end of the scale *p* is the limiting factor, since *n* is less than a week. The potential number of infective mosquitoes (Table 2.1) reach a peak at 30.6°C, after which it drops off rapidly. Temperatures of above 32°C have been reported to cause high vector population turnover but also weak individuals and high mortality survival. Thermal death for mosquitoes occurs around 41-42°C (Jepson et al. 1947; Haddow, 1943) but *p* is zero at 40°C (Martens, 1997). So above 31°C, suitability starts declining, reaching zero at 40°C.

The relationship between mosquito abundance and rainfall is complex and best studied when temperature is not limiting. Studies have demonstrated the association between *Anopheles gambiae s.l.* abundance and rainfall (Molineaux & Gramiccia, 1980; Charlwood et al., 1995) but a direct, predictable relationship does not exist. *Anopheles gambiae s.l.* are observed to breed more prolifically in temporary and turbid water bodies such as ones formed by rain (Gillies & de Meillon, 1968) while in permanent bodies predation becomes important. *Anopheles funestus* in contrast prefer more permanent water bodies (Gillies & de Meillon, 1968). However, both temporary and permanent water bodies are dependent on rain. Rain is also related to humidity and saturation deficit: factors that affect mosquito survival (Molineaux, 1988). There is good reason for using rainfall to indicate the probable presence of vectors, their survival and the potential for malaria transmission. Although it is known that flooding often causes destruction of breeding sites (Molineaux, 1988) and a temporary reduction of vectors, it never eliminates the vector, so that very high rainfall was still considered optimal for transmission.

Table 2.1 Temperature effect on sporogonic duration (n) (Macdonald 1957; Detinova, 1962), daily vector survival (p) (Martens, 1997), percentage cohort survival against sporogonic duration (p^n) and larval development (Jepson et al., 1947).

T (°C)	Duration of sporogeny (days)	Daily vector survival (%)	Vector survival after period required for sporogony (%)	Larval development (days)
16	∞	89.3	0	47
17	111	89.7	0.001	37
18	56	90.0	0.28	31
20	28	90.3	5.9	23
22	19	90.4	15	18
30	7.9	88.1	37	10
35	5.8	80.8	29	7.9
39	4.8	38.9	1.1	6.7
40	4.6	0	0	6.5

Craig et al. (1999) combined temperature and rainfall to define the vector and parasite viability for transmission within fixed seasonal windows of time. Fuzzy logic models were used to define the suitability for stable malaria transmission across the continent at approximately a 5 x 5 km resolution. In this model, climate data were derived from weather station data from between 1920 and 1980 (Hutchinson et al., 1995) and interpolated into mean monthly temperature and rainfall surfaces. The model was structured to define distribution by setting the lower temperature cut-off at 18°C and assuming a saturation of the temperature

effect by 22°C; similarly rainfall values between 0 and 80 mm demarcate the range within which transmission is limited. Combined these features must coincide on a month-to-month basis for five consecutive months and a frost-factor (minimum temperature of less than 5°C) would eliminate transmission at any point in a contiguous period. In North Africa high temperatures combined with a rapid onset of a short duration rainfall allows for a limited transmission period of less than three months. The model was modified to allow for three-month conditions for North African areas. The model (Figure 2.3) provides fuzzy membership, or climate suitability values, ranging from 0 (unsuitable) to 1 (very suitable). Clearly there is an element of circularity in defining whether these models compare well with reality and one is often left with comparisons with "expert" opinion maps. In 1943 the Colonial Administration commissioned a series of maps for the East African Protectorates including a "best guess" map of malaria distribution (Atlas of Kenya, 1959). Butler and Nelson developed their map on the basis of whether transmission occurred less than 3 months, 3-6 months or greater than 6 months each year (Figure 2.4). Overall the fuzzy climate suitability model shown in Figure 2.3 compares well with this map (Figure 2.4). Malaria free areas as judged by Butler & Nelson represent fairly well areas considered to have a zero suitability for stable transmission. Furthermore the description of acutely seasonal or "malarious near water" represent areas with a fuzzy value of 0.2 or less. However, because the model is based upon long-term climatic averages, it provides a conservative estimate of stable transmission distribution and does not allow for epidemic potentials among areas where transmission is traditionally limited by either rainfall or temperature. Furthermore, comparisons with expert-opinion maps indicate that discrepancies in major river valleys result because the model uses only rainfall to predict water availability, while mosquitoes do survive along major river banks and flood plains, such as the Tana River.

A different approach has been taken by Hay and colleagues (1998) who examined the frequency and seasonal distribution of clinical cases against proxys for climate recorded from remote sensed images from satellites sensors. The predictions were made using relationships established between long-term data on paediatric severe malaria admissions and simultaneously collected data from the Advanced Very High Resolution Radiometer (AVHRR) on-board the National Oceanic and Atmospheric Administrations (NOAA) polar-orbiting meteorological satellites and the High Resolution Radiometer (HRR) on-board the European Organisation for the Exploitation of Meteorological Satellites' (EUMETSAT) geostationary Meteosat satellites. The remotely sensed data were processed to provide surrogate information on land surface temperature, reflectance in the middle infra-red, rainfall, and the normalised difference vegetation index (NDVI). These variables were then subjected to temporal Fourier processing and the fitted Fourier data were compared with the mean percentage of total annual malaria admissions recorded in each month. The NDVI in the preceding month was found to correlate most significantly and consistently with malaria presentations across the three sites (mean adjusted $r^2 = 0.71$, range 0.61-0.79). Regression analyses showed an NDVI threshold of 0.35-0.40 was required for more that 5% of the annual malaria cases to be presented in a given month. These thresholds were then extrapolated spatially with the temporal Fourier processed NDVI data to define the number of months for which malaria admissions could be expected across Kenya in an average year, at an 8 x 8 km resolution. In many ways these maps were easier to compare with those developed by Butler and Nelson (Figure 2.4) as they represented numbers of suitable months. The resulting map is shown in Figure 2.5. This map was better able to delimit areas close to permanent water bodies.

Before the precise relationships between temperature and sporogyny were well established altitude was the subject of much interest among earlier malariologists (Garnham, 1948; Schwetz, 1942; Hackett, 1945). Essentially altitude and temperature are intrinsically linked as every 100 metre increase in altitude roughly corresponds to a 0.5^{o}C decline in ambient temperature. Schwetz (1942) describe altitude-limiting levels for transmission in the Congo at approximately 1700-1800 metres. However, the varied nature of reports suggest that the limits of altitude vary considerably between the mountainous regions of Africa (Lindsay & Martens, 1998). However, Lindsay & Martens (1998) consider malaria to be absent above 1500 metres, which is contrary to early descriptions of malaria in Kenya made by Garnham who described transmission and epidemics at 1680 and 1950 metres (Garnham, 1948). In an editorial on highland malaria in Kenya, Rees (1994) regards malaria as unstable at altitudes greater than 1600 metres, whilst Oloo et al. (1996) refer to unstable malaria occurring at altitudes of 1700 – 2300 metres and malaria has been described as high as 2400 metres (Clyde, 1967; Garnham, 1948). Overall the use of altitude is confusing, nevertheless there is a tendency within the literature to refer to "highland malaria" in East and the Horn of Africa (Najera et al., 1992; Lindsay & Martens, 1998). To examine the relative distinction of altitude for the Kenya context we have selected an arbitrary limit for truly unstable malaria as 1800 metres. The available digital altitude map displays contours at 304.8 metre intervals (UNEP-GRID, 1992). Thus the closest contour we could select was 1828.8 metres. This was displayed in Mapinfo® and a contour polygon created. The resultant map can be seen in Figure 2.6.

2.3 Epidemic prone districts as defined by the Ministry of Health.

In 1992 a National Plan of Action for Malaria Control was developed by the MoH in consultation with local technical experts (MoH, 1992). The Plan of Action identified three priority districts regarded at that time as "epidemic prone" and based upon historical experiences and recent epidemics in 1988, 1989 and 1990. These districts were Uasin Gishu, Nandi and Kericho. These limited areas were first shown in the "malaria map" developed by DVBD in 1983 (DVBD Annual Report, 1983). This map was an extension of the map developed by George Nelson and Butler in the 1950's and later modified by Roberts in the 1960's.The map was attached as part of the National Plan of Action in 1992. By 1995 during the development of the National Guidelines for Diagnosis, Treatment and Prevention of Malaria for Health Workers (MoH, 1998), definitions of at-risk epidemic districts had changed and included Kisii, Nyamira, Nandi, Kericho, Bomet, Trans Mara, Kakamega (including Vihiga), West Pokot, Uasin Gishu, Trans Nzoia and Gucha. The guidelines also allude to the unstable conditions in the semi-arid areas of Turkana and Narok. Following the El Niño related epidemics in North Eastern Province during 1997/98 the subsequent development of the National Guidelines for Malaria Epidemic Preparedness and Response in Kenya included the districts in NEP (MoH, 1999). The current "unofficial" list of districts included as epidemic prone is shown in Table 2.2 and Figure 2.7.

These 21 epidemic prone districts cover a range of ecologies from areas traditionally regarded as "highlands", to areas of intermediate altitudes and ultimately areas where transmission is limited by low rainfall. National Census data for 1989 have been used in conjunction with district-level, intercensal growth rates (1979-1989) to project population estimates to 1997. These calculations have been performed on total population counts at the 4th administrative boundary level (location). The 1997 projected population data have been compared to altitude and climate-base models of stable transmission to provide a series of population-at-risk estimates shown in Table 2.2. Entire districts have been selected as the unit for sampling because epidemic preparedness and response plans are likely to be applied across an entire

Table 2.2: Districts with >= 75% surface area in zone defined as unsuitable for stable transmission by fuzzy probability model or altitude versus MoH definitions of epidemic prone

District	MoH Defined Epidemic Prone	District surface area	<0.2 fuzzy surface area (%)	Population in '97	% Pop. Located <0.2 fuzzy	Surface area or Population > 75% above 1828 m
Mandera	Y	28,420	100	140,143	100	
Wajir	Y	53,930	100	110,954	100	
Marsabit		76,230	100	138,127	100	
Garissa	Y	44,680	100	131,216	100	
Isiolo	Y	25,010	100	100,100	100	
Samburu	Y	21,190	100	141,804	100	
Turkana	Y	66,660	98.2	230,222	97.1	
Tana River	Y	38,660	92.7	179,564	73	
Kitui	Y	30,280	100	731,456	100	
Meru		7,328	100	1,104,425	100	
Nithi		2,282	100	323,898	100	
Embu		2,802	100	483,697	100	
Kirinyaga		1,488	100	503,386	100	
Narok	Y	15,130	94.1	458,287	97.6	Y
Nairobi		740	78.6	4,287,341	95.4	
Nakuru		7,887	100	1,276,450	100	Y
Kiambu		2,596	80.6	1,108,045	88.1	
Nyeri		3,372	100	739,453	100	Y
Laikipia		9,738	99	352,003	96.5	
Nyandarua		3,276	100	482,472	100	Y
Bomet	Y	2,379	100	458,297	100	Y
Kisii	Y	676	87.4	465,829	85.1	
Nyamira	Y	894	82.6	534,272	82.6	Y
Gucha	Y	673	98.4	436,428	86	
Trans Nzoia	Y	2,471	100	472,750	100	Y
Uasin Gishu	Y	3,386	100	620,357	100	Y
Kericho	Y	2,590	79.2	659,598	84.1	Y
Nandi	Y	2,884	53.9	608,035	52.1	Y
Trans Mara	Y	2,891	71.9	224,091	94.3	
Kakamega	Y	3,047	23.5	1,316,686	12.3	
Vihiga	Y	575	25.2	566,830	1.8	
West Pokot	Y	9,286	34.8	298,271	34.8	
Total		**473,453**		**19,684,487**		

district by District Health Management Teams (DHMT) unless sub-district targeting of resources can be developed.

Table 2.2. lists those districts for which greater than 75% of the land surface (and or population) are located within the fuzzy limits of stable transmission of 0.2 or less (see above). The Fuzzy Suitability Model (FSM) included Meru, Nithi, Embu and Kirinyaga districts located east of Mount Kenya; Nairobi and Kiambu; Nakuru, Neyri and Liakipia located in the central rift valley areas; and Marsabit located in the arid North. These districts have not been defined as epidemic prone through contemporary experiences of the Ministry of Health. Conversely districts regarded by the MoH to be prone to epidemics, such as Nandi, Kakamega, West Pokot and Vihiga, are not classified as being unsuitable for stable transmission. Table 2.2. also lists districts which have greater than 75% of their land surface (or population) located above 1828 metres. Clearly such definitions exclude the low lying arid communities but include Kiambu and Nyeri, two districts not classified as epidemic prone.

The total population regarded by the Ministry of Health as being at risk of malaria epidemics is 12,962,871 (in 1997) or 46% of the total population. 17.4% of the projected 1997 population were living above 1828 metres whilst 62% of Kenya's population live in districts classified as being unsuitable for stable transmission according to fuzzy logic climate models. Which ever ecological definition one uses a large proportion of Kenya's population reside in areas traditionally not thought suitable for stable transmission.

2.4. Clinical patterns and distribution of disease in selected "epidemic" districts.

One of the key purposes of this investigation is to provide a more detailed description of the clinical patterns (secular and temporal) of malaria in areas exposed to unstable or epidemic transmission. These details for clinical settings in Nandi, Kericho and Wajir are provided in Chapters 4 and 5. This section provides a description of malaria admissions at a variety of other epidemic prone districts in Kenya. In addition, Kirinyaga district has been included to provide a wider scope of information among districts not included in the MoH's list of epidemic prone areas. These data were abstracted from ward registers located at district and mission hospitals in collaboration with District Medical Records Officers. Monthly tallies were recorded of all malaria-related diagnoses by age groups < 1, 1-4 years, 5-14 years, children without age recorded and non-pregnant adults. Diagnoses including "chloroquine resistant malaria", "anaemia ? malaria", "unconscious ? malaria" were all classified as malaria admissions. It was not possible to identify the residence of each admission and admissions were likely to have derived from a wide catchment area. Nevertheless data from Kilifi district hospital on the Coast suggest that over 75% of malaria admissions to a rural district hospital are residents living within a 20km radius of the hospital. Hospitalisation with malaria represents a severe morbid event although it must be recognised that "severity" will range from a "clinical concern" to a potentially life threatening event. More often than not diagnoses were made without reference to microscopy and this limits the accuracy of the definitions used. However, it is more likely that in-patients have a microscopic diagnosis than out-patients and what ever the accuracy these patients are managed as if they were malaria.

Hospitals at Kirinyaga, Samburu, Nyamira and Eldoret were visited between March and May 1999. The monthly admissions for periods where data were available are shown in Figures 2.8 - 2.13.

2.4.1 .Kirinyaga district is located to the East of Mount Kenya where altitude ranges from 1200 metres in the south (Mwea division) to over 1800 metres in the north (Gichugu division). The district experiences two rainy seasons March-July and a short rains October-December. Mean annual temperatures range from 19 – 23^{o}C. The district's centre, Kerugoya, is the location of the district hospital and situated at 1524 metres above sea level. During 1990, 1994 and 1995 malaria accounted for 15.7% of all admissions to the hospital and 13% of the deaths occurring in the hospital. The irrigation scheme at Mwea has been the subject of much research (Ijumba et al., 1990; Mukiama et al., 1989: Rapuoda, 1995; DVBD Annual Reports 1978 & 1979) where *An. arabiensis* and *An. pharoensis* and *An. funestus* have been identified as the principal vectors. According to these surveys *An. arabiensis* accounted for 73% of vectors sampled, *An. pharoensis* 7% and *An. funestus* 3%. Among nine cross-sectional studies conducted in the southern part of the district between 1989 and 1990 parasite rates among children aged 1-9 years ranged between 0.7% and 5.4%. The seasonal nature of disease shown in Figure 2.8. Peak malaria presentations occur during the long rains which begin after March. The peaks in malaria admission vary year-to-year and sometimes the short-rains peaks are equally important. This temporal pattern is similar to other epidemic prone areas such as Nandi (section 4.4.); Kericho (section 4.6) and Nyamira (Figure 2.10). However the peaks observed in these other districts vary significantly more between years. Perhaps of greater significance is the present age-pattern of disease which appears to affect individuals below and above 15 years equally (52% of admissions were below 15 years between 1996 and 1997). Such a situation was not in evidence during the late 1980's (64% of admissions were below 15 years between 1988 and 1989). Co-incidental with a tendency toward admissions including older individuals there has been a continuos rise in hospital malaria burdens through the 1990's (Figure 2.9).

Figure 2.8: Seasonal pattern of malaria admissions of all ages to Kerugoya district hospital, Kirinyaga 1988 to 1997.

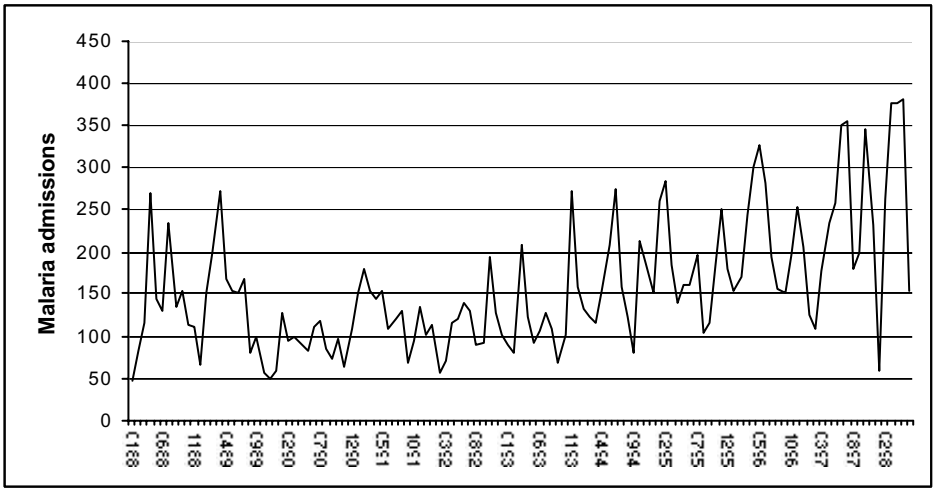
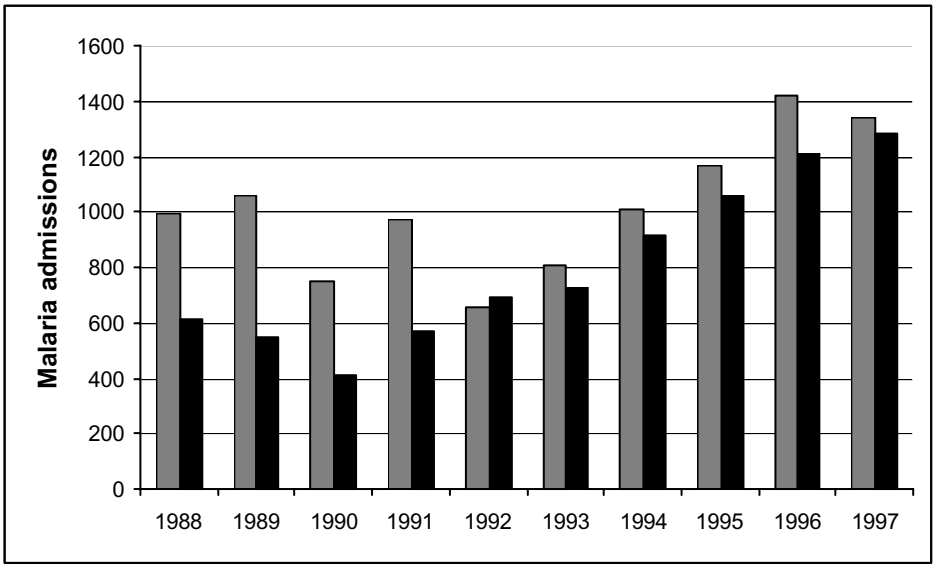


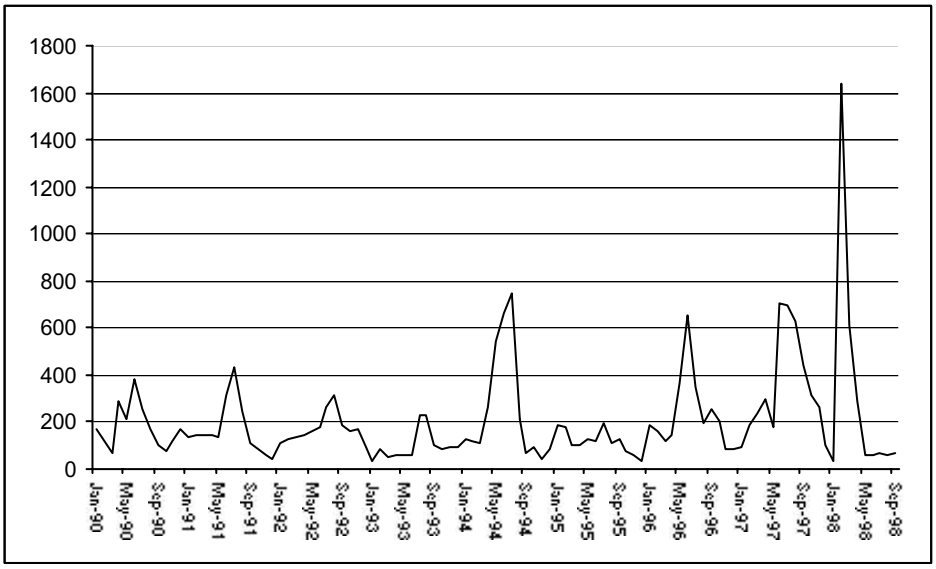
Figure 2.9 Numbers of malaria admissions to Kerugoya district hospital, Kirinyaga between 1988 and 1997. Grey bars are admissions below 15 years of age and black bars are above 15 years of age.



2.4.2 Nyamira district. Nyamira is located in Nyanza Province and has been defined by the MoH as epidemic prone. It borders Kisii district and was once part of this district at a time when the combined area was regarded as epidemic during the 1940's (Chapter 3). The district is divided into two topographical zones, the northern part comprising Nyamira and Ekerenyo divisions which lie between 1500 metres and 1800 metres and the southern half of the district which is mostly above 1800 metres. Rainfall averages 2000 mm per annum with the long rains occurring between March and June and a short rains between October and December. Mean maximum temperatures vary between 24.7^{o}C and 27.1^{o}C and minimum temperatures between 9.9^{o}C and 11.9^{o}C. Tea is a major industry in the area and five factories owned by the Kenya Tea Development Authority operate in the area alongside two private estates. Nyamira district hospital is located at 1,971 metres above sea level in the middle of the district. In 1995 and 1996 malaria accounted for 42% of all admissions to the hospital and 32% of the deaths that occurred at the hospital. The MARA/ARMA project has been unable to identify any parasitological or entomological data for Nyamira District. However, data from the neighbouring Kisii district suggests that parasite rates among children aged 6-14 years can vary significantly from 4.5% to 50.1% (Kaneko, personal communication). Vectors sampled by DVBD in Kisii suggest that *An. funestus* is an important vector, accounting for approximately 43% of anopheles sampled (DVBD, 1980 & 1981).

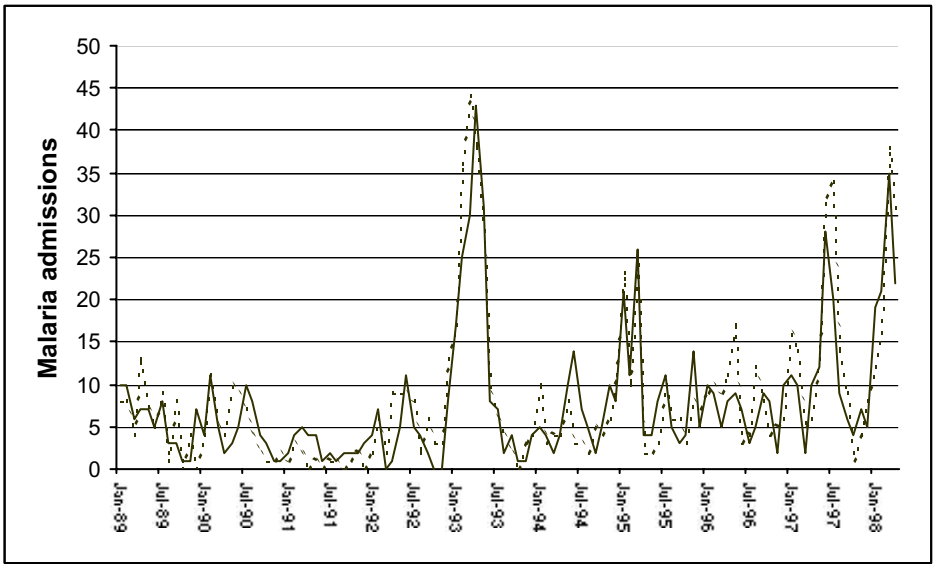
It was only possible to identify paediatric malaria registers at Nyamira hospital. However, summaries of all malaria admissions were available for the period 1990-1997. Comparing the ratio of childhood to adult admissions indicates that throughout the period admissions have been equally distributed between the two age classes with 47% of admissions being aged less than 15 years. Significant disease peaks among the paediatric malaria admissions are evident in June 1994, June – August 1996 and again during the long rains of 1997 and the exceptional peak following the El Niño related rains in January – March 1998 (Figure 2.10).

Figure 2.10: Malaria admissions to Nyamira district hospital from 1990 through to September 1998.



2.4.3 Samburu district is located on the northern interface between the highlands and the lowlands. The east of Suguta Valley is dominated by high level plateau's. Kirisia hill, rising 2000 metres above sea level forms the highest point. The high plains of the district (Elbarta, Sware, Bonyeki and the middle Waso Nyiro range from 1000 to 1350 metres).The Mathews Range and Ndoto Mountains are located in the central plains. The area comprises nomadic pastoralists of the Samburu. A catholic mission hospital is located at Wamba, on the Mathews Range at 1356 metres above sea level. Wamba is the biggest division of the district accounting for 24% of the land mass and 19% of total population. The rainfall is erratic. Data obtained from the meteorological office for Archers Post show an average annual rainfall of 605 mm. Long rains occur between April and August (although very variable) and there is often a short rains between October and December which lasts for only one month. Using monthly averages between 1936 and 1954 there appears to be no consecutive three-month period with rainfall always above 80 mm for each month. Overall the area is prone to drought and subsequent food insecurity. Mean monthly maximum temperatures vary between 31 and 33.4^{o}C and mean monthly minimum temperatures between 17.3 and 20.1^{o}C. No parasitological or entomological surveys have ever been conducted in this area however the climate suitability model indicates that the arid nature limits transmission across the district (Table 2.2). Admissions are few to this small hospital but microscopy is always used for diagnosis of all in-patient cases and records are well maintained. Sister Anne Lelesiit of the mission was kind enough to abstract the data for their hospital since 1989 (shown in Figure 2.11). A striking epidemic was observed during the early parts of 1993, 1995, 1997 and the El Niño related epidemic during 1997/98. Malaria admissions are equally distributed between those aged greater and less than 15 years of age.

Figure 2.11. Malaria admissions to Wamba Mission hospital, Samburu district since 1989: children less than 15 years (dotted line) and adult admissions (bold line).



2.4.4. Uasin Gishu district is located in Rift Valley Province north of Kericho and Nandi districts. Eldoret is the district's major centre and the location of the district hospital (2,133 metres above sea level). Rainfall is pretty evenly distributed across all months of the year with an average annual precipitation of 960 mm. The months of March and August tend to have the highest rainfall. The highest temperatures are 25^{o}C whilst the lowest is 8.8^{o}C. The average temperature in the district is 18^{o}C with the hottest month being February and the coolest June. During a study among school children at Ngeria and Towet locations parasite rates were 4.3% and the vector was *An. gambiae s.l* (no speciation done) with a sporozoite rate of 0.25% (Khaemba et al., 1994). One of the earliest recorded epidemics at Eldoret was in 1928 (Campbell, 1929). Furthermore, Eldoret was one of the earliest places during the late 1980's to record the "re-emergence" of epidemics or what was soon to be called "highland malaria" (Some, 1994). Nevertheless malaria has always posed a significant clinical burden to the district hospital. A review of the Medical Officer-in-Charge's annual reports to the Provincial Medical Officer between 1953 and 1957 show that malaria accounted for 27% of all admissions and that it accounted for 9.4% of the deaths occurring in the hospital. Case-fatality of malaria admissions between 1953 and 1957 was 2.2% (Pitt, 1953, 1954, 1956; Cathro,1955; Malherbe, 1957, 1958). Case-fatality in 1990 was 6.7% (Some, 1994). The annual reports during the 1950's also present monthly out-patient data (Figure 2.12) for all microscopically diagnosed malaria patients between 1952 and 1957 (the report for 1953 was missing the data on treatments). These data demonstrate both the seasonality of the disease and the large scale between-year variation. Malaria admission data were abstracted for the hospital at Eldoret for the period 1990-1998 (Figure 2.13). Data during 1996 were lost during a fire at the hospital and no data were recorded during the nurse's strike of December 1997 and January 1998. Despite these gaps, an epidemic during July 1991 is clearly evident as is the epidemic after the short rains in 1995. There was a peak incidence between July and August 1997 but toward the end of this year and the beginning of 1998 the epidemic associated with El Niño rains was not in evidence as was the case at other sites. Interestingly Some (1994) describes an epidemic during 1990 which heralded significant press coverage (Chapter 3), this "epidemic" whilst probably significant compared to the 1980's was far less significant than those described after 1990.

Figure 2.12: Ambulatory, microscopically confirmed cases of malaria at Eldoret hospital between 1952 and 1957

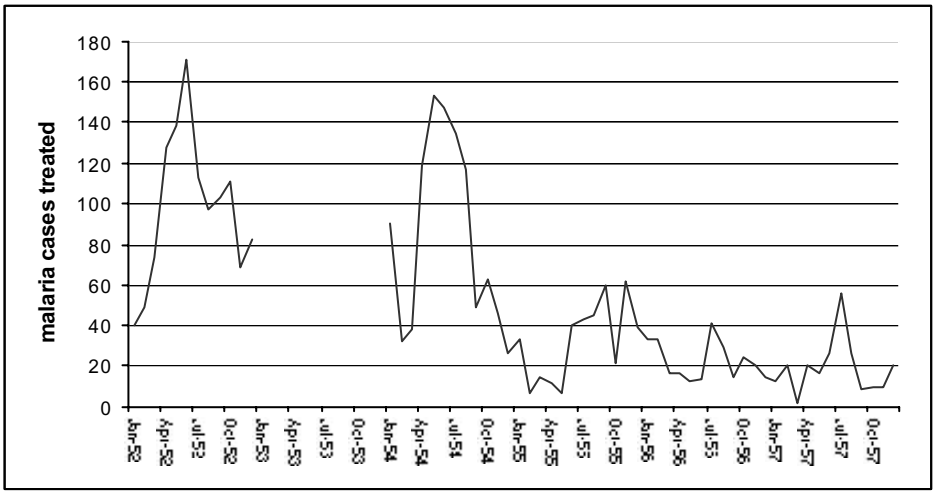
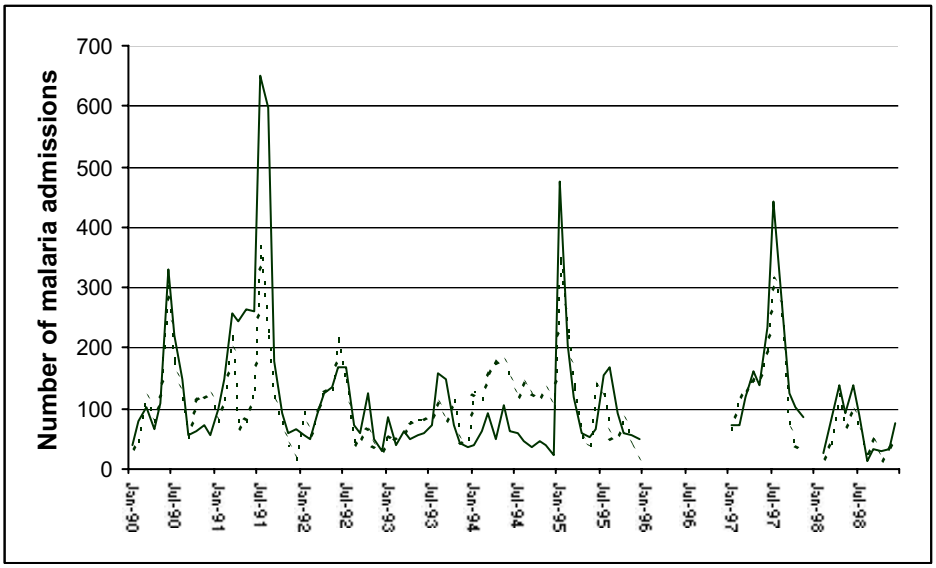


Figure 2.13: Malaria admissions to Eldoret district hospital, Uasin Gishu district between 1990 and 1998 (dotted line admissions below 15 years and bold line admissions above 15 years).



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3. The history of malaria epidemics in Kenya: their political significance and control.

3.1. 1900-1919: The medical services and malaria

The arrival of both the Imperial British East African Company (IBEA) in 1888 and the missions in the 1890s led to efforts on their part to attract trained personnel to Kenya, and resulted in a limited number of stations offering rudimentary medical services to the African population. The IBEA employed fully trained staff and, equipped them with the necessary materials and a hospital in the colonial town of Mombasa on the coast. For the most part the medical staff's services were restricted to the treatment of company members only. When the British government assumed control of East Africa in 1895, it gained control of the medical staff of IBEA. The medical services within the Protectorate were consolidated into a medical department consisting of a primary medical officer, seven doctors, three nurses, and seven hospital assistants by the year 1901 (Beck, 1970). In this way, the objectives of the IBEA's medical facilities were expanded from treating company members to ensuring the health of all Europeans planning to make East Africa their new home.

As the Colonial Office took over all government aspects of Kenya in 1905, administrators began to discover the magnitude of the effort required for malaria control in East Africa. This realisation may have been a factor in the painfully slow institution of control measures. Kenya's PMO, Milne, wrote in 1910 of only one significant effort - the work of a "*mosquito brigade*" of trained staff conducting rudimentary environmental control activities such as clearing bottles and tins, cutting harbouring bush, and filling in of borrow pits. The 1911 department report depicted "*gangs of convicts*" being employed to drain low-lying marsh and clean drains, and suggested such a programme be continued for a fortnight prior to the seasonal onset of rains. Noteworthy at this time was Milne's reluctance to advocate prophylaxis, his explanation being:

> *"The most effective measure for the eradication of mosquitoes has been bush clearing. Quinine is but irregularly taken by Europeans, and institution of any system of quinine prophylaxis or the free distribution of the drug, would, I am convinced, be attended with the most meagre results*" (Colony & Protectorate of Kenya, 1911).

Of the Native reserves, Milne had a different opinion:

> *"It is difficult to say that any but spasmodic efforts toward the prevention of the breeding of mosquitos have been carried out. The litigious temper of the inhabitants renders even ameliorative measures well nigh impossible .... [*malaria*] exists in places in which it should never have been allowed to get a hold, and yearly is a cause of a heavy mortality amongst Africans and Asiatics. The reason for this is two-fold; in the townships insufficient drains, not only public ones, but the almost total absence of domestic drains connecting houses with the roadside channels; and, everywhere, owing to the race for development, a tendency to disregard the unrenumerative expense that sanitary reform entails, and the unproductive waste of time involved in keeping compounds in order."* (Colony & Protectorate of Kenya, 1911).

In 1912, the anti-mosquito rules were made applicable in Mombasa as well as Nairobi and the enforcement of these laws among householders was added to the tasks of mosquito brigades in this and following years. Sanitary inspectors were hired, trained, and

dispatched to the three major towns of the colony - Nairobi, Kisumu, and Mombasa. Funds were freed to supplement the meagre routine schedule with experimental projects such as the introduction of larvivorous fish in Mombasa's water tanks, and the distribution of quinine to groups such as the native police officers. Also targeted were the workers of railways and their environs. An envelope of protection was soon completed, stretching from Mombasa on the coast, through Nairobi to Kisumu on the shore of Lake Victoria. The increases in success and opportunities for expansion were sufficient in remoulding Milne's views on how best to go about combating a general rise in malaria incidence.

> *"... reports from all provinces are unanimous in stating that malaria is on the increase; and energetic measures, combined with an intelligent and well supervised quinine prophylaxis, will be necessary as soon as opportunities present themselves."* (Colony & Protectorate of Kenya, 1923).

3.2: 1920 to 1925

John L. Gilks took over as the PMO of Kenya in 1920 and a major undertaking of his tenure was the land reclamation project along the shores of Lake Victoria, near Kisumu. Having commenced with preliminary grass cutting and clearing, filling in of ditches and n-dnor drainage, the project grew into a well-funded effort. By 1923, the Kisumu land reclamation project included major drainage works, the construction and maintenance of Partington's dyke, and the prevention of future land loss through agriculture (Colony & Protectorate of Kenya, 1915).

The grounds for justification had shifted somewhat from Milne's brand of argument based purely on efficacy to one considering economic benefit. The prospect of new arable land was itself a motivating force in the Kisumu project, one arguably more powerful than the hope of alleviating illness among the native population. Anti-malarial work of this kind, combining economic and public health interests, found considerable favour among administrators of several departments.

The epidemic of 1926 - deemed "*more alarming in extent and more serious in its consequences*" than any previously recorded - lent a sense of urgency to the quest for malaria control [Figure 3.1]. *Anopheles costalis* (*An. gambiae s.l*) was found to be highly adaptable to man-made breeding sites accompanying urbanisation. Thus, the case of the 1926 epidemic was employed by Gilks as a warning against reckless economic development. In his annual report, Gilks not only highlighted the more obvious sources of increased breeding in townships ("*borrow-pits, and other excavations, ill-graded earth drains"*), but also illustrated the avenues whereby the anopheline could spread into the rural, epidemic areas.

> *"That there have been no notable general alterations in the domestic environment of the natives of these reserves during recent years is true, but on the other hand it is to be remembered that in every direction roads, and to a lesser extent, railways, have been carried into and through these areas, and always where there are roads, artificial and undrained excavations are to be found."* (Colony & Protectorate of Kenya, 1926).

3.3: The epidemics of the late twenties

The epidemic of 1926 also led to a shift toward an attempt to plan and design a unified effort against mosquitoes. Investigations were made as to the conditions of anopheline breeding in various towns, and recommendations were submitted for "*taking adequate steps to secure the permanent elimination of breeding places*" (Colony & Protectorate of Kenya, 1926). The major step carried out here was that of recognition, a necessary precursor to policy change. The elimination of breeding sites had by this time been recognised as a priority. And although major, nation-wide anti-mosquito programmes will not be launched for years to come, the above shows a slight appreciation of the role extensive township programmes must play in public health policy. The only issue presented as a hurdle at this time was that of its feasibility.

In 1927, the government heeded the recommendations and granted the sum of £20,000 for anti-malaria work in the Nairobi area (Colony & Protectorate of Kenya, 1927). Nairobi's Municipal Corporation agreed to add an equal amount toward the project, and the Kenya and Uganda Railway Authorities accepted the financial responsibility for the eradication of breeding sites in the area under their control. With this influx of funding and commitment came the initiative to appoint an Anti-Malaria Works Committee, under the chairmanship of the Commissioner for Local Government and Lands, to discuss arrangements for operations targeting the anopheline breeding sites scattered through the Nairobi area. The seriousness of the malaria problem had been recognised, and the co-operation between health and non-health departments ensued.

However, the 1928 epidemic was not particularly concentrated in Nairobi; its effects were felt throughout the country, in both rural and urban areas. While the routine measures of oiling, filling in, and minor drainage continued in other parts of Kenya, the Nairobi area was selected, for various reasons, to not only be the centre of organised and funded works, but its sole site. This was perhaps a lapse in judgement that Gilks himself took to heart:

> *"Local authorities in general are taking an increasing interest in preventative measures in their areas, but the lessons of the epidemic of 1926 are to some extent already forgotten"* (Colony and Protectorate of Kenya, 1927).

1928 would provide Gilks with a series of tragic yet enlightening occurrences. Epidemics of malaria struck the Plateau and Trans Nzoia areas (Farnworth-Anderson, 1929-30), both centres of European settlement, as well as south Lumbwa (Kericho), Kisii, and Nandi districts. Campbell (1929) describes the epidemics in Uasin Gishu and Trans Nzoia. Approximately 2,500 Europeans had settled in the area beginning with the large migration of Afrikaners from South Africa after the Boer War. The area was thought to be malaria free until about 1919-1920's and some blamed the railway extension for its introduction. He noted that in 1927 there were only 60 cases of malaria from both districts treated at the Civil Native Hospital with three malaria deaths. In 1928 at Uasin Gishu alone there were 152 cases treated and 12 deaths. He further describes 179 European cases treated at Eldoret from Uasin Gishu and Trans Nzoia (representing an attack rate of 68.8 per 1000 population during 1928). Campbell (1929) summaries as follows:

> *"The very severe outbreak of malaria in 1928 began during the dry weather towards the end of seven months of drought and was probably*

largely due to physical and mental depression lowering resistance of people. To prevent a repetition of this outbreak it is necessary:

a) *For everybody to take personal protective measures against infection.*
b) *That the standard of living should be raised throughout the community.*
c) *That the production of vegetables, fruit, and diary produce be increased in the districts and that the towns co-operate by providing markets.*
d) *That native labour be better housed and better fed."*

Gilks was particularly expansive in his reporting of the effect epidemics had on government attitudes toward malaria.

"The position was undoubtedly serious and dislocation of businesses occurred, but although tragedies have to be deplored something good has emerged. There has been an acceptance of the fact that malaria exists and that it is a factor of economic importance. The population is now willing and anxious to adopt, and to spend money for, measures which will lead to the improvement of the public health generally of which malaria is but one manifestation. Hitherto, there has been a tendency to disguise or deny the presence of malaria. Government has alas realised that malaria cannot be dealt with as a matter apart from the other aspects of the public health problem and money has been voted not only for an increase during 1929 in the medical services in the areas more severely affected by malaria in 1928, but for a general increase throughout the Colony." (Colony & Protectorate of Kenya, 1928).

According to Gilks, the cost of malaria epidemics had now begun to include the loss of business as well as lives. Development required a level of political and environmental stability for it to continue at a considerable pace. An epidemic's effect on the availability of labour at various levels of business was a concern the Kenyan government as a whole could share. To disguise or deny the magnitude of the problem had become unacceptable primarily because the loss of income and progress was glaringly real. The connection between health and economics was both powerful and persuasive in the politics of government, a notion Gilks grasped and sought to exploit.

"It has to be remembered that economic prosperity and the state of the public health are so closely inter-related that they are inseparable. Without an improvement in economic prosperity it is very doubtful whether any considerable permanent improvement in the public health can be effected. The converse is also true. It follows that improvement can only come about as a result of a general policy of development in which every activity of Government is concerned." (Colony & Protectorate of Kenya, 1928).

Gilks' 1928 report can be seen as a documented attempt to render control a part of the fabric of Kenyan affairs.The Nairobi Anti-Malarial Works Committee first sat in January of 1928 and held meetings at intervals of a few weeks to sort out aspects of an effort being

viewed by many as a possible model for control in the rest of Kenya. The committee immediately voiced difficulties in having to negotiate with individual land owners prior to proceeding with control; it requested powers to demand priority for control issues; and it recommended experts such as anti-mosquito engineers be recruited as part of the project. The gradual formulation of anti-malaria policy in Nairobi influenced the success of financial requests from the medical department to Government at the end of each year. The estimates for 1929 mirrored the recommendations of the Nairobi Committee in making provisions for engineering projects in the more distant, less-populated towns, and in requesting the services of an expert for this purpose. Furthermore it requested the appointment of two farm medical officers who would be responsible for investigating conditions in the rural settled areas and carrying out personal propaganda with regard to the measures necessary for safeguarding the health both of European farmers and their employees. In addition requests were made as follows (Colony & Protectorate of Kenya, 1928):

> *a) The establishment of medical units and hospitals in three native reserves which are at present without medical attention.*
> *b) The posting of additional medical officers in four native reserves in which the provision of medical officers is at present inadequate.*
> *c) The provision of funds for propaganda with special reference to the improvement of housing conditions in the native reserves.*
> *d) The provision of additional facilities for entomological investigation.*

3.4: Malaria as social disease: a historical review

The year 1929 was marked by a generally low incidence of malaria. A series of malariometric surveys were designed and conducted throughout the country in an attempt to acquire a sense of disease distribution. Gilks summarised present knowledge on regional endemicity by dividing the colony into three groups (Colony & Protectorate of Kenya, 1929):

> *a) Areas in the highlands where malaria was absent whether in endemic or epidemic form.*
> *b) Areas in highlands where in recent years, malaria had become endemic or appeared as periodic epidemics.*
> *c) Great areas in the lowlands where malaria was endemic, where it undoubtedly takes a great toll of life and yearly is responsible for much sickness and disability.*

Among the latter Gilks was referring to the less sensationalised malaria deaths along the coastal regions. The coast of Kenya was densely populated but mostly by indigenous peoples. Less money and attention was given these areas due to both the ethnic make-up of their populations and malaria's more stable presence in the environs. Gilks drew much-needed attention to malaria in these areas, alluding to a future possibility of extending activity to areas populated exclusively by non-Europeans. Meanwhile, routine urban activity continued unabated throughout the country.

The major change in 1929 came in thought rather than activity. Gilks presented an important shift in the perception of malaria.

> *"Malaria is now being generally recognised as a 'social' disease, that is a disease which is dependent for its continuance in areas where it is endemic, on among other factors, a low standard of living among the bulk of the population; it follows that any general anti-malaria policy must take due cognisance of this fact and that all measures aimed at ensuring a general reduction of the disease must be based on this knowledge*" (Colony & Protectorate of Kenya, 1929).

That malaria need be treated as a product of societal conditions was a notion Gilks could have garnered from observing epidemics in surrounding Native reserves. However, it was most likely a result of a recent turn toward endemic areas, where malaria was perennial in nature. By 1929 anti-malaria policy had been in place for only five years. No such policy had been delineated before 1925 not, according to Gilks, because there was no problem to be had,

> *"but because at that time the only major problem presented was that of malaria in the Native Reserves and there, at that time as now, the need was for general development and the establishment of general health services*" (Colony & Protectorate of Kenya, 1929).

Control policy began as a product of an increase in funding provided toward that goal and the hiring of the medical department's first entomologist in order to obtain expert advice on ways control measures moulded by the "*knowledge of the part played by the mosquito*" might be developed (Colony & Protectorate of Kenya, 1929). The first organised anti-malaria policy came with the serious highland epidemics of 1926, when plans to eradicate breeding sites in the Nairobi area gained momentum. The second epidemic of 1928 struck in various other parts of the highland area and thus with a very different affect.

> *"... owing very largely to the fact that the districts affected were less highly developed than those previously attacked, and the European population less prosperous, the incidence of the disease among Europeans was greater and the effects more severe, and the importance of the disease as one which might affect a rural community was more widely appreciated"* (Colony & Protectorate of Kenya, 1929).

With the epidemic of 1928, colonialists began to realise the growing futility of isolating one's home from the rest of the land. The European settlements situated away from the urban centres became a concern for health administrators. Moreover the plight of malaria in Kenya entered an international arena following the much publicised speech made by the Prince of Wales on Armistice Day in Nairobi and reported in The Times (Anon, 1928). Because he saw the reputation of the Colony suffering due to this disease, and by extension its prosperity the Prince urged for the implementation of "*an intensive and scientifically conducted campaign*" the result of which would be to "*accelerate the progress of Kenya a hundred fold*" (Anon, 1928). This speech led to a flurry of activity among Colonial Office staff in London and the commissioning of an expert committee to examine the Kenya situation. This was eventually led by SP James (James, 1929).

3.5: The 1930s

The extended period after the 1928 epidemic did eventually have a dampening effect. Gilks again found the lack of epidemics a bit of a mixed blessing, complaining of a growing impression *"that the menace of malaria had been exaggerated in the past*" (Colony & Protectorate of Kenya, 1930). A prevailing economic depression did not help matters. While changes in control did not proceed at a pace some had envisioned, the shift toward a social perception of control remained intact, forming the basis in which preventative measures were being carried out. Improvement of sanitation and standards of living (health services, housing) became goals of departments and the primary strategy in rural areas:

> *"It is on these lines that malaria is being attacked in Kenya, and except in townships and other special areas this indirect method of attack would appear to be the only feasible procedure"* (Colony & Protectorate of Kenya, 1930).

AR Paterson replaced Gilks in 1932. Paterson's new tenure heralded an expansion of routine anti-larval measures. Kakamega, Meru, Malindi, Kilifi, Isiolo, and Kitui were added to the list of major towns such as Nairobi, Kisumu, and Mombasa that could afford a systematic control effort. The use of Paris Green as larvicide moved from its successful experimental stage of the late 1920s to that of routine measures often replacing oiling of breeding sites. By 1935, the Colonial Developmental Fund had approved substantial funding for extensive anti-malaria plans in Kisumu and Mombasa.

Early in 1935 a comprehensive pamphlet on the prevention of malaria was prepared by the Department, and issued free to the public. The pamphlet, titled "*A Guide to the Prevention of Malaria in Kenya" (*Paterson, 1935), was developed following the government entomologists and Paterson's warning that

1) *Local experience has also shown that in a year following on a period of severe drought malaria in Kenya may become epidemic, this happened in 1926, and again in certain areas last year.*
2) *Though malaria may not assume epidemic proportions this year, conditions at present time are so similar to those which preceded the severe epidemic of 1926 that it is not improbable that an epidemic may occur, and certainly highly probable that unless reasonable precautions are taken by individuals many avoidable cases of malaria will occur.*
3) *Malaria is avoidable, and neither you, nor any member of your family, need suffer from malaria this year if you will take certain simple and reasonable precautions now.*
4) *Reasonable precautions do not involve unreasonable expenses.*

The pamphlet was developed for two principle reasons:

> *(a) The less the natives, and especially the native children, on a farm suffer from malaria the less is the owner, or manager likely to suffer from malaria.*
> *(b) Malaria among native employees may, on occasion, involve the farmer in serious economic loss.*

The pamphlet advised Europeans to embark on a personal programme involving defence strategies, such as mosquito nets and boots, and the attack of mosquitoes with pyrethrum spraying. Thus, the heart of the programme lay in the interaction between mosquito and the European armed with a spray gun. In the pamphlet Paterson states:

> *"It would appear, therefore, that by means of spraying of huts provided only that the spraying were carried out efficiently and in a systematic manner any outbreak of malaria in the settled areas of Kenya could be prevented from assuming epidemic proportions and from completely disorganizing the labour forces concerned".*
>
> *"System and supervision would, however, be essential. Spraying boys would require to be engaged and trained, and it would be necessary to determine how many huts could be dealt with by one boy in a day, and to ensure that the huts actually were regularly and thoroughly sprayed. Very clearly, however, one boy could certainly spray at least fifty huts daily if they were not too far apart. Therefore, assuming that only four labourers lived in each hut, one spraying boy could deal with a labour force of 200 by daily spraying, or with a labour force of 1,200 by weekly spraying".*
>
> *"The cost of material and labour plus supervision required to give a hut a weekly spraying for a six months season worked out in South Africa at Sh. 3/5d. per hut last year."*

On treatment Paterson's comment's were as follows:

> *"Now the chief symptom of malaria is fever, that is a rise in the temperature of the body-"Homa." Malaria is common in Kenya that it is probable that on nearly every occasion when an African native complains of even slight fever or "Homa" in an area in which malaria occurs he is suffering from malaria. Therefore as a general rule whenever an African complains of fever he should be treated for malaria…What will it cost to treat your labour? Not so much as you think. Here are the figures:-The Government will sell you a pound of quinine for Sh. 32. One pound of quinine = 7,000 grains of quinine, or 466 15-grain doses or enough to treat sixty-six labourers for seven days at the rate of 15 grains of quinine daily for each man. It is unfortunately the case, however, that there are still people who appear to consider that an attack of malaria is of no great importance, and who refuse to take any precautions whatsoever. Such an attitude is unfortunately to a degree, not only for the persons concerned, but for the public as a whole, and for the reputation of the colony. Even a slight attack of malaria injures one's health, while repeated attacks of malaria impair health very seriously indeed, and most definitely impair efficiency, and ultimately both the individual and the colony lose thereby".*

The medical department soon had most of the European community protected from the impending epidemic of 1935/36. In Nairobi at least the epidemic when it did arrive in 1937 seemed to have a much reduced impact upon the European population than the African

population. Symes (1940) describes the clinical burden of malaria in Nairobi between 1925 and 1938. He uses various sources of information and it is not clear how these compare. Nevertheless the data for African out-patient treatments from the Railway's and General dispensary clearly show the effects of the 1937 epidemic relative to previous years (Figure 3.1). Among the Europeans the rates of hospitalisation remained unchanged during 1937 (Figure 3.2). Symes also described the changing pattern of endemicity in Nairobi during this period. Between August 1927 and August 1929 parasite rates among children ranged between 2.5 and 5%. In 1938 parasite rates among 2,418 school children aged 4-17 years was 26%. Symes invokes some crude estimates of the economic impact malaria has on European and African residents in Nairobi. He calculated that

a) The average number of days off duty for Europeans treated for malaria at the European Hospital during three recent years, was ten days (six days for Africans).
b) The average wage for Europeans, not including those receiving more than £540 a year is Sh 25/- a day (females Sh 12/50). (60 cents per day for Africans).
c) About 20% of people infected with malaria and seeking treatment go to hospital. The remainder are treated in their homes or at dispensaries. Hospital fees and accompanying additional expenditure for Europeans is Shs 30/- a day and for Africans is Sh 1/- per day and that private practitioners fees would be additional costs.

Using these crude estimates of disease burden and patient cost Symes estimates that malaria costs the Nairobi community approximately £4,000 per annum whilst only £1,000 is spent each year on its control. Here we have one of the first attempts in Kenya to equate costs, disability and intervention impact. Such approaches to defining problems and needs are today powerful public health approaches within the arena of malaria control (Goodman et al., 1999) and yet Symes used identical tools to raise awareness over 60 years ago in Kenya.

For the epidemic in 1937, the consequences, economic or not, were felt predominantly by the African population. Paterson's pamphlet may have contributed to a successful protection among Europeans but despite rhetoric on ways to improve the standard of living among the indigenous people, this same population were still regarded, as Gilks did in his 1930 report, *"the reservoir of the disease*". Paterson's pamphlet reveals the effect of this attitude among policy-makers and was never more clearly articulated in a government document.

Figure 3.1: Outpatient treatments for malaria as recorded at the General and Railways dispensary in Nairobi between 1925 and 1938 (Symes, 1940).

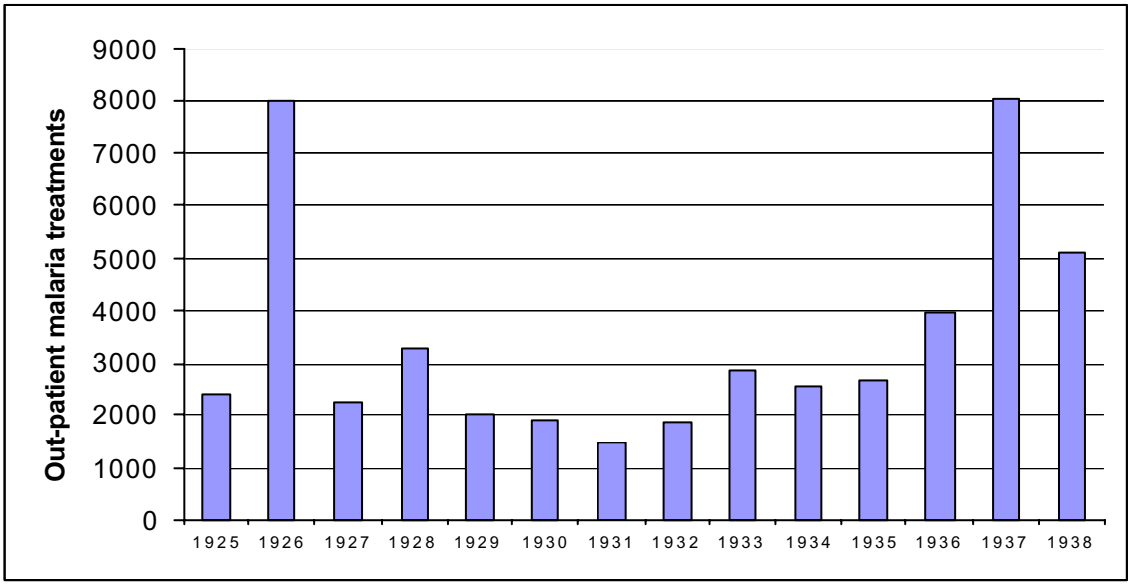
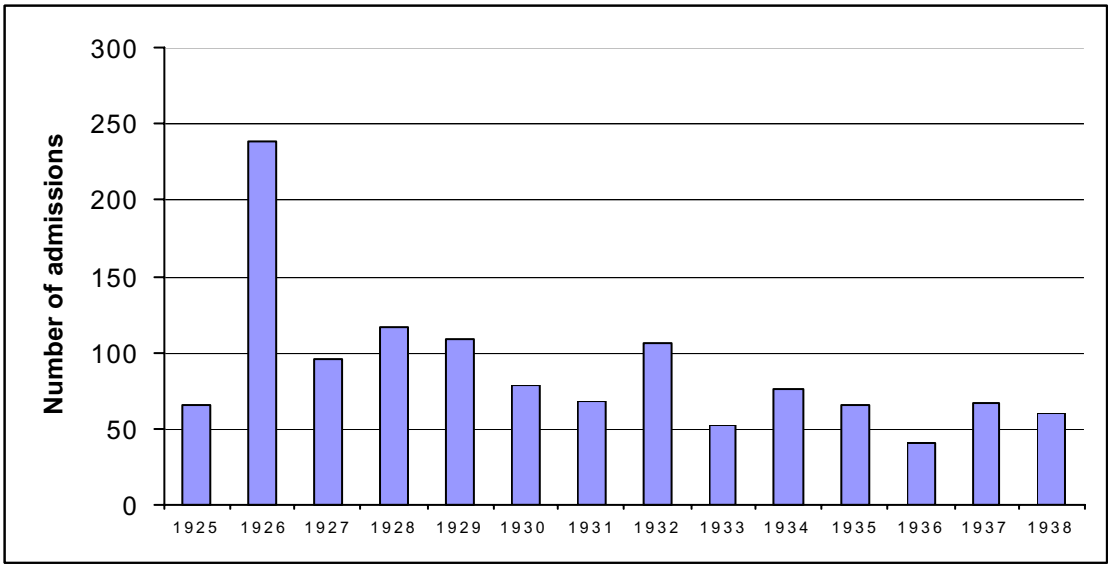


Figure 3.2: European malaria admissions to the European hospital in Nairobi between 1925 and 1938 (Symes, 1940).



3.6: The 1940s

The early warning of entomologists in Kenya would again be imporant during the 1940 epidemic. According to an abbreviated report (Colony & Protectorate of Kenya, 1940), the medical department had predicted the occurrence of an epidemic some time before, taking precautions among the European settlements and making the use of mosquito nets mandatory for all ranks of military troops, "*including Africans*". The result was a slight rise in malaria cases among settlements and troops, but devastating losses among indigenous populations. The Nairobi area was hit especially hard, partly because *'the experience of the past had gone unheeded*,' and the municipal anti-malaria effort was inadequate in increasing sanitation and living standards, tempering the dangers of development, and securing control over malaria in its Native reserves. De Mello (1947) documents the course of the 1940 epidemic in Nairobi using data from the notification system established by the Nairobi City Council in 1930 (Figure 3.3). De Mello (1947) describes the genesis of epidemics in Nairobi as being a function of three components:

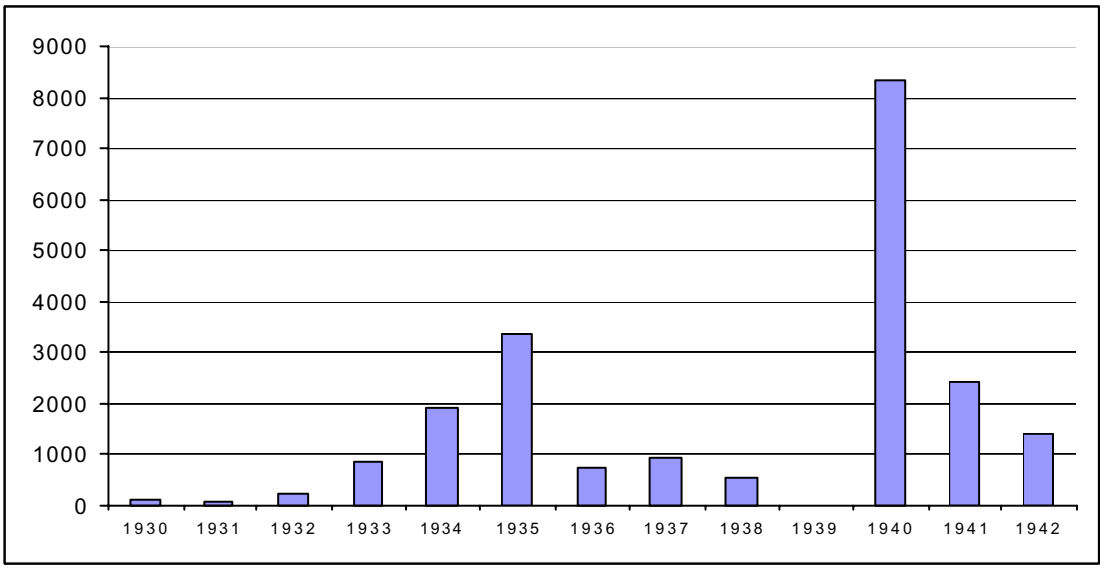
1. An increase in the density of vectors.
2. Lowering of the immunity in the local population due to 5 years of healthy period or 5 years of unhealthy period due to drought, famine or malnutrition.
3. Factor X. or “epidemic potential” due to relapses which lead to wave of primary infections among non-immune hosts.

Paterson took a more pragmatic view of the 1940 epidemic and attributed the loss of life to the inadequacy of anti-malaria programmes. Two specific reasons for a declared failure of Nairobi's control measures were provided. The first of which , ironically for Paterson, concerned a gross lack of research invested into the Native reserves.

> *“Firstly, the Department has never had sufficient staff to acquire a detailed knowledge of those topographical or entomological factors*

which govern the incidence of malaria in many of the great reserves, or sufficient to enable us to keep up to date in every area our knowledge of those environmental changes which may favour the spread of Malaria or result in its occurrence in epidemic form." (Colony & Protectorate of Kenya, 1940)

Figure 3.3: Notified cases of malaria reported to the Nairobi City Council (De Mello; 1947; Symes, 1940).



Paterson laid open to criticism the limited amount of research done on the conditions of malaria in Kenya. A majority, if not all, of investigations had been centred around areas not well inhabited by indigenous peoples. By this statement, Paterson also revealed a knowledge that malaria ecology may differ considerably in neighbouring areas, and that the reluctance to take this possibility fully into account was an omission from policy that cost lives. Also mentioned was the department's inability to react to an obviously epidemic situation.

"Secondly, ... the staff is inadequate to provide timely and adequate medical intelligence with regard to the incidence of unspectacular disease. The epidemic was most severe in the large native reserves of the central highlands, where ordinarily the incidence of malaria is low. We were, of course, aware at an early date that an epidemic was occurring, the evidence being provided by the rise in the number of hospital admissions, and arrangements were accordingly made for the distribution of quinine on a considerable scale, but as there was no staff available for outdoor work, and no machinery whereby deaths were automatically recorded, the severity of the epidemic was not fully appreciated at the time. When ultimately, extra staff became available a count was made in certain areas which showed that among one population of about 350,000 some 6,000 deaths had occurred from malaria alone during the four months of the epidemic." (Colony & Protectorate of Kenya, 1940).

Paterson cited as a fundamental problem the Department's indifference to the unspectacular or dramatic, pointing to the normally stable and low incidence of malaria in highland areas (in which Nairobi lies) as the reason why malaria was deemed a rather unexciting issue. However, he reported a difference in the precautions taken at the first indication of an epidemic, implying that reactions to a coming epidemic depended on the population assumed to be in danger.

1941 remained a year that followed the general form of the epidemic cycle, where a range of recommendations were made and activities were intensified. An anti-malarial organisation was appointed, with an anti-mosquito military unit in support. This time, though, the organisation wished to act on a nation-wide basis, seeking to establish or intensify control measures in "*every town and port of importance in the Colony, in all the larger trading centres, and along all the lines of communication*" (Colony & Protectorate of Kenya, 1941). Once again the bulk of the efforts were concentrated in urban, township areas with the department becoming slightly alarmed by the steady rise of malaria into the rural highlands, particularly the Masai reserve, Kericho and Kisii.

> *"A spread of malaria into parts of the highlands of the Colony which were formerly free from this disease has been going on now for some years, but without additional staff for investigation and control little or nothing can be done, and for the time being additional staff of the type required is unobtainable"* (Colony & Protectorate of Kenya, 1941)

Epidemics in highland Kenya, varying in magnitude, location, and effect, were to recur throughout the 1940s, as malaria extended its territory upwards from the lowland, hyperendemic regions to the areas of altitudes formerly thought impenetrable by the disease. For example, malaria epidemics were described in Lodiani (8000 feet) in 1941 and Timbora (8,300 feet) in 1944 (Garnham, 1948). The responses to this trend began to take on a characteristic form. In 1942, Paterson considered it

> "*probable that if staff could be detailed for a proper investigation into nature and cause, measures could be devised to both prevent further spread and clear recently infected areas of the disease*" (Colony & Protectorate of Kenya, 1941).

In 1943, FJ Carlyle Johnstone, the then acting director, expressed concern over the spread of malaria as did FR Lockhart, the acting director in 1944. NM McLennan, appointed director in 1945, referred to the spread of malaria as *"extremely disturbing"* and reiterates this concern, sometimes lifting the words directly from the previous year's report, till the end of the decade. It is apparent that Kenya's medical department had come to an impasse, and the 1940s were indeed marked by a searching quality. The department of the 1940s sought to deal with this impasse in several ways. One response was further compartmentalisation. The Division of Insect-Borne Diseases (DIBD) was set up in an attempt to concentrate efforts against Kenya's communicable diseases, of which malaria was merely one.

> *"Preventative measures against insect-bome diseases .... demand a high standard of special skill and knowledge. At the end of the year steps were taken to set up a Departmental Division of Insect-Bome Diseases and the whole of the organisation comprising research, field surveys, control was brought under the central direction of a senior*

> *specialist officer* [PCC Garnham]. *It is expected that by the co-ordination of all work in field greater efficiency economy will be effected and the machinery be made ready for expansion"* (Colony & Protectorate of Kenya, 1944).

DIBD (renamed the Division of Vector-Born Diseases in 197x) would soon become the main avenue for conducting and reporting all anti-malarial measures. During the year of its inception, DIBD took control of entomological investigations, doing field work in the major towns and other smaller ones such as at Londiani, Equator, Timboroa, Kapsabet, Thika, and Kiambu. DIBD's role in research cannot be overstated, as an enduring response to the dilemma of rural malaria involved the expression of hope that *"the discovery of new insecticides and the development of new methods of control in the field ... will permit of great advances being made in the post-war period"* (Colony & Protectorate of Kenya, 1944). DIBD's role in the development and adaptation of these new methods to Kenya's unique malaria ecology would prove vital in the colony's attempt at nation-wide eradication.

McLennan's first task was to tackle the recurring issue of rural highland malaria. In the 1945 report, he began by reiterating the dilemma that previous medical directors were faced with.

> *"Of the importance of malaria in relation to the general economy there can be no doubt, and the spread of malaria which has been taking place in the last few years into the Highland areas is now extremely disturbing. The area largely concerned is the mountainous country surrounding the Kano plains of the Nyanza province and includes both native and settled area. Of late the position has deteriorated and has caused much sickness among labour on tea estates and subjected the areas to increasingly severe epidemics as year follows year. The resultant economic loss is heavy"* (Colony & Protectorate of Kenya, 1945).

The economic argument was then evidenced by the loss of labour in the tea estates known to be crucial to Nyanza area and Kenyan economies. The specificity of argument uncovered more than a mere wish on the part of McLennan to be more persuasive. Rather, it also revealed the growth of understanding that had occurred over the years of expanding the malaria problem in Kenya. The medical department had graduated from Gilks' wish to make control policy a priority to McLennan's quest for a means to effectively and affordably attack malaria in the rural highland areas of Kenya. The use of insecticides against mosquitoes offered the most promise that the new director could uncover.

> *"In the face of these conditions, the institution of control measures is vitally necessary. Fortunately, unlike the control of malaria in localities more naturally subjected to the disease, control in these uplands is likely to have a fairly definite and more early chance of success. After consideration, therefore, it was decided that a proposal should be put forward for experimental control of the vector by DDT house impregnation on a wide scale. Should this prove successful in a limited area, the way might then be open to complete vector abolition"* (Colony & Protectorate of Kenya, 1945).

DDT and other insecticides were now being tested by both military and civil organisations, and information on their applicability to sections of Kenya's varied ecology was followed

with hope. The air-spraying of swamps near Kisumu was conducted during 1945 with success, rendering the technique a possible "*alternative to large and costly drainage works and at the same time ... a substitute for other temporary control measures"*. Ariel spraying at Dar es Salaam and Kisumu using 32 mg per sq. metre of pure DDT reduced anopheline populations by 98% (Wilson & Robertson, 1947). Other trials conducted by DIBD during this period included Pyrethrum dusting of rural households in Maragoli, Kakamega (Garnham & Harper, 1944). Further plans for large scale experiments using DDT on huts were submitted for the year 1946. Routine measures were conducted in the larger towns of Nairobi, which now included Nairobi, Kisumu, Mombasa, Malindi, Gedi, Kilifi, Fort Hall, Nyeri, Kisii, Kakamega, Maseno, Isiolo, Karatina, and Kitui.

The considerable experimental efforts of the military during this period in time led to the further discovery of applicable methods of control. As research rose in prominence, the knowledge of the factors of a given ecology became a priority, resulting in a series of studies concerned with vector behaviour at different altitudes and climate types. For McLennan, though, rural malaria remained the perplexing issue and while DDT experiments had proven satisfactory, further work had to be done to render the use of DDT fully effective and affordable. Along with the work on DDT, the development of new causal prophylactics and curative agents added a new dimension to control, making possible the consideration of mass measures combining mass chemotherapy and insecticidal spraying. Even by the end of the 1940s, still absent was a nation-wide, centrally organised policy, and in 1949, on the eve of the WHO-led Kampala Conference of 1950 (WHO, 1951), McLennan drew attention to this omission

> *"Control measures against this disease are on a reasonable sound administrative basis in the municipalities of Nairobi, Mombasa, Kisumu. Elsewhere these measures are somewhat fragmentary and the reasons for this have been investigated during the year. It would appear that no clear pronouncement has been made concerning the responsibility for the provision of finance for such measures. This has led to the institution of a number of uncoordinated and small enterprises throughout the country which have been severely crippled in scope by the uncertainty regarding finance, staff and direction*"
> (Colony & Protectorate of Kenya, 1949)

As a replacement of the current structure, McLennan called for an anti-malaria policy designed to "*eliminate these uncertainties*" by standardising key aspects of control efforts. He recorded moves toward making financing and everyday staff a local authority responsibility, and the administering of technical advice, entomological surveys, new schemes, and general supervision a duty of the central government and medical department (Colony & Protectorate of Kenya, 1949). It was an obvious summons to model a control programme after those carried out in other nations, and an accurate reading of the international atmosphere regarding malaria control - for many had come to believe that eradication was now possible.

3.7: The Eradication Era

EA Trim took over as medical director with the new decade and continued McLennan's approach to anti-malaria policy, albeit with a different result. Trim reasserted malaria's position as the most widespread disease of the Colony. He acknowledged the quarter century of work carried out in gathering accurate information on distribution and incidence, the result being that

> *"the areas where malaria is endemic and hyperendemic are known and the parts of the country which are subject to seasonal manifestations only can also be fairly accurately delineated*" (Colony & Protectorate of Kenya, 1950).

Information was seen as practically complete regarding the bionomics of Kenya's primary vectors, *An. gambiae and An. funestus,* and the data collected had now been thoroughly tested during large-scale insecticide spraying experiments. Trim suggested then, the practical, well-informed yet aggressive push to "*intensify and improve measures for the control of malaria in areas of high population density*" (Colony & Protectorate of Kenya, 1950). During 1951 an unusually heavy and prolonged short rains in October led to arrangements being made for wide-scale distribution of therapeutic drugs, particularly Mepacrine (Quinine based) for the native population in the rural areas (Colony & Protectorate of Kenya, 1951). Nevertheless Trim felt the areas for a concentrated control effort were areas of high population density, though, referred to townships, he felt less positive about control in other parts of Kenya.

> *"In rural areas the control of malaria is a much more complex problem. If we could consider this question in terms of eradication involving capital expenditure the economics of the problem would be greatly simplified. Unfortunately, however, as our malarious areas have no natural boundaries, which can act as barriers to reinfection, as in the case of an island, prevention would involve annual recurrent expenditure of considerable proportions"* (Colony & Protectorate of Kenya, 1952).

The reports of various organisations during this period repeat the enduring state of measures limited to urban sites "*where they are in the main effective"* (Colony & Protectorate of Kenya, 1960). Control policy in rural areas was discussed, but it became even more evident to administrators that such policy would "*be governed not only by financial stringency but also by doubts as to the feasibility of a regional malaria eradication scheme*" (Colony & Protectorate of Kenya, 1960). While eradication schemes were flourishing across the globe, local experts recommended policy

> *"along traditional lines: improvement of the efficiency of the existing organisation for control and treatment, regular review of peri-urban drainage works and a greater effort to frustrate increases in the already heavy load of man-made malaria in the region"* (East African Institute of Malaria & Vector Borne Diseases, 1961/62).

Thus, throughout Kenya, the pattern was of the intensification of routine measures including scouting for breeding sites, conducting entomological studies, oiling and larviciding of stagnant waters, filling in of excavations, even bush clearing. Some new methods were tested in various sectors of the country, notably scattered pockets of the endemic coast, where prophylaxis and residual insecticide spraying were combined, and certain communities living on the shores of Lake Victoria, where an experimental trial of prophylaxis alone was being conducted. The fragmentary nature of control that McLennan pointed out at the end of the 1940s continued, and the expressed pride in urban success would prove to be another trait of public health policy inherited from the past. Then in 1961

> *"The heavy rainfall late in 1961 also brought flooding to the mainland*

> *and gave rise to outbreaks of malaria in many areas that are normally healthy. These were particularly severe in Eastern Kenya and extended deeply into the hinterland. Nairobi has experienced several serious malaria epidemics in the not so distant past and a repetition of this calamity seemed almost inevitable. That a malaria epidemic in Nairobi was successfully averted is a reminder, that may be timely, of the continuing importance of traditional methods of malaria control, and of the efficient work of municipal malaria control organisations".* (East African Institute of Malaria & Vector Borne Diseases, 1961/62).

By the middle of the 1960s, the true role of DIBD emerged as a sort of conduit of information. As Kenya found itself witnessing both an ongoing global eradication effort and a traditional movement among local authorities, DIBD rose not only as a source of advice and assistance, but also as a co-ordinator of disparate activities of the nation. DIBD served this function as an international entity as well, where

> *"close contact is maintained with both international agencies, (i.e. World Health Organisation) and with institutes within the East African community who are also working on the problem of malaria within the three territories*" (DIBD, 1967).

> *"The principle of malaria eradication which was propounded by World health authorities during the era 1950-60 has proved to be too distant an ideal for national malaria programmes in the immediate future. However, the objectives of the present malaria programmes is to control the degree of malaria transmission so that the disease ceases to be of Medical and Public Health importance in Kenya. It can be seen therefore that there is still a long way to go"* (DIBD, 1969).

3.8: Malaria epidemics in from the 1990's.

In 1990 a series of "epidemics" occurred in Uasin Gishu, Nandi and Kericho, three contiguous districts in the economically significant Western Highlands. These epidemics were comprehensively covered in the newspapers and lead to a series of important changes in the way the Ministry of Health viewed malaria. On the 27th June 1990 The Standard Newspaper quoted the District Commissioner of Uasin Gishu's claims that malaria had taken 68 lives including a local assistant chief. John Ouma took over DVBD in 1990 and his first challenges were to establish control and responses to the claims by Uasin Gishu, then Nandi and Kericho districts for support and advise. Makeshift dispensaries were established and the community were advised to attend these as early as possible if they developed fever. The Standard article alludes to the fact that this epidemic was due to "drug-resistant strains" and research groups from the National Medical Research Institute (KEMRI) and the Universities were quick to respond to these new research needs. The epidemic was well described by Eliab Some the MoH at Eldoret (Some, 1994). Malaria admissions overwhelmed the services with over 1,200 admissions between June and July and 84 deaths at the hospital. Some (1994) maintained that over 92% of all the malaria deaths occurred at Turbo Rural Health Demonstration Unit which on average saw between 700 and 900 malaria cases per month between April and June (1985-1989), in 1990 the same quarter required the treatment of 1669 cases. Improved case management linked to public awareness was the main thrust of the campaign but also limited use of residual insecticides (Reskol), larvicides (Abate 500E) and adulticide (Cooper 25) were deployed. From the recognition of the problem Some was able to solicit support and organise

teams of mobile clinics within 6 days. Within 7 days of the campaign 13,820 people were treated. Some (1994) estimated that the additional cost to the district budget during the epidemic was KES 1.8 million for June alone.

During the 1950's and 1960's malaria incidence at Uasin Gishu was generally low (Figure 2.12). Pitt (1953) reports that this was due to a large extent because of preventative strategies aimed at oiling, the retention of a malaria specialist team at the district level and support from the East African Malaria Unit at Amani who provided "*malaria engineering*" surveys and reports. In addition the extension of programmes in Nandi and Kericho districts (Chapter 4) aimed at mass drug administration with Daraprim and Dieldrin residual house spraying all contributed toward a significant reduction in malaria in this area. Pitt (1953) goes on to explain how these preventative strategies were organised:

> *"The Turbo-Kipkarren Framers Association formed a Malaria sub-Committee to do the executive work for this scheme. The Plateau Wattle Company managed the finances and the secretarial work. The farmers paid at the rate of 20 cents per single dose. All costs including transport, with the exception of technical personnel, were met by farmers. There was a profit from the scheme of 214.75 shillings which is being put towards future control"*

The economic costs and administrative arrangements of prevention versus containment were clearly very different. The organised prevention was developed in the 1950's through a wider sector of the community at the district level whilst managing the epidemic in 1990 was essentially the province of the district health management team. Medical Officers reports up to 1971 suggest that malaria cases in the Turbo area were few and the mid-to-late 1980's were according to Some (1994) half those experienced during the 1990 epidemic.

The newspapers continued to report throughout the summer of 1990 on similar epidemics in Trans Nzoia, Nandi, Kakamega and Kericho districts. The following year sporadic claims of epidemics were reported in Elgeyo Marakwet, Kisii, Nandi and West Pokot districts. In 1992/93, following a period of drought, heavy rains preceeded claims of a yellow-fever outbreak in Baringo and Uasin Gishu districts (Okello et al., 1993). By mid-January 1993 cases of haemorrhage and jaundice were reported and investigations by international experts confirmed the presence of the virus. Nevertheless, Ouma noted that these isolated cases masked a far greater problem at the time in the respective districts, malaria.

During this period of increasing concern of emerging epidemics in the Western Highlands, the Government formed a National Malaria Control Programme. Its roots lay within the Division of Vector Borne Diseases, a division within preventative rather curative services. Dr Beth Rapuoda was appointed the manager and a series of National Action plans were developed in consultation with a wider sector of the control, NGO and research communities (MoH, 1992). The National Plan of Action reflected a battery of strategies to be promoted in Kenya, including insecticide-treated bed nets, residual house spraying, larviciding, environmental management, health education and prophylaxis for special at-risk groups (pregnant women, non-immune visitors and sickle cell sufferers). Despite this impressive check-list of preventative strategies the plan clearly articulates two key issues: 1) the emphasis on effective prompt treatment and case management, and 2) the need for intersectorial collaboration for malaria control to be effective. A significant departure from recommendations made between 1920 and 1960 was the recognition that malaria affects predominantly young semi-immune

Kenyan's and pregnant women. Gone were suggestions of eradication and strategies aimed at elite sectors of the population. Nevertheless, the complete reversal of interest toward stable endemic communities left recommendations for the special conditions of the non-immune populations located in the highland epidemic conspicuous by their absence. Despite the welcome attention now paid to Kenya's population living under endemic conditions epidemics continued to galvanise interest and political commitment to malaria as a wider public health problem over the next 6 years.

1994 heralded the next major confrontation with epidemics across the country. Districts affected included Nandi, Kericho, Uasin Gishu, Trans Nzoia, Kakamega, Kisii, Nyamira, Trans Mara, Narok, West Pokot and Turkana. In addition, Kuria district experienced at least 118 deaths between May and August, a district previously (and subsequently) unaffected by epidemics. During this epidemic it became evident that cracks were appearing in the overall health services, not simply by being overwhelmed by patients but also through a poor supply of essential drugs. Furthermore the only available therapy was chloroquine, for which there was mounting evidence of much reduced sensitivity (Chapter 6). At Kisii district hospital with a bed capacity of 35, approximately 80 children were being treated for severe anaemia with blood transfusions in August 1994 and there was an acute shortage of blood giving sets (Hill, 1994). Nyamira and Kuria districts were without functioning vehicles and were unable to maintain any out-reach services or health education. The Ministry of Health provided only between 50,000 and 110,000 shillings as additional support to Nyamira, Kisii and Kuria districts. Several reports indicated the rapid expansion in private, "unofficial" pseudo-medical services were operating a booming business during the crisis and capitalising on the breakdown in the formal health services (Hill, 1994). Pleas were made by District Commissioner's such as Harry Wamubeyi of Kisii for more funds and drugs (The Standard Newspaper, 26th July 1994).

The Ministry of Health sent a distress message to the donors to assist in the supply of additional drugs. The Government of the Netherlands responded by supporting costs related to case management, drugs and health education. The US$ 521,965 was managed by the UNICEF country office. Drug supplies began to reach the drugs by November 1994, after the epidemic wave had run its course. Other sources of funds were accessed from residual funds from the British Government's ODA support for operational research in Western Kenya (US$12,500) and drug sensitivity test kits from the WHO's office in Ethiopia. At best the situation was, fragmented, poorly resourced and probably resulted in the tragic loss of many lives. But as Gilks said in 1929 "*something good*" may have emerged from the crisis and that possibly was the recognition that Kenya's principal mainstay for therapy was no longer effective and concessions were made to deploy sulphadoxine/pyrimethamine during the highland epidemics.

Drugs procured by the Netherlands Government on behalf of the Kenyan Ministry of Health were stocked for wide spread distribution during 1995. On the whole there were few reports of epidemics during 1995 with the exception of an isolated report from Nandi District. Health facilities were adequately stocked with anti- malarials and combined with favourable climate conditions probably resulted in a reduced incidence of complicated clinical disease during 1995. Kisii and Nyamira were affected by epidemics again during 1996. At Kisii these were localised to Marani, Sameta, Mosocho, Keumbu and Ogembo divisions. Increased case loads at Kisii led to the authorisation of the re-deployment of clinical staff by the Provincial Medical Officer to Kisii.

The El Niño floods of 1997/98 greatly affected the entire country. Nandi, Nyamira, Kisii, Trans Mara and parts of Kericho districts were reported to be worst affected. The Daily Nation on the 5th February 1998 ran a headline "*Govt in alert over malaria*". In the article the Director of Medical Services, Dr James Mwanzia said:

> *"Reports by officers on the ground show a rise in malaria cases due to the increase in mosquito breeding places…..Kenyans should protect themselves by adopting a two-pronged approach: preventing the spread of the mosquito vector and seeking effective treatment"*

Ironically the Ministry of Health's Essential Drugs Kit, distributed to health facilities nation-wide still contained mainly chloroquine and only chloroquine for the most peripheral levels of the health service. Consequently the DMS's reference to "effective treatment" was at odds with what was available through the majority of the Government's formal health sector. The National Malaria Control Programme established an emergency malaria-specific drug kit. Its contents were developed on the basis of what was known about current drug sensitivity and the newly formed guidelines for the management of complicated malaria (MoH, 1998). The British Government's DFID provided £0.5 million as financial support for the crisis to cover extensive training, health education drug procurement and dispatch. The hospitals in districts such as Kericho and West Pokot were becoming overwhelmed, The Standard Newspaper reported Dr Hilary Mabeya at Kapenguria Hospital, West Pokot as saying that between 50 and 60 patients were being admitted daily. This headline in this same article read "*Malaria claims 118 more lives in Kericho district*". But unlike 1994 drugs effective drugs were delivered soon after the onset of the epidemic to the highland districts.

Conversely districts in North Eastern Province were pretty much neglected and it was the headline news from The Daily Nation on the 12th February that captured everyone's attention. The newspaper claimed that 1,500 had died of malaria. The newspaper reading population and Ministry of Health senior management had been used to dramatic figures for the highlands in the 100's not 1000's and the Nation report sparked much controversy. This debate, the response and outcomes of the epidemic as it affected the NE province are discussed in more detail in Chapter 5. Meanwhile, the Ministry of Health immediately convened a Task Force to deal with this escalation of the epidemic. The Task Force comprised of as many partner's and stakeholders as possible from within and outside of the Ministry. The Task Force met every week to every two weeks between March 1998 and April 1998 at the WHO offices in Nairobi and was chaired by Dr Gaturuku, the deputy DMS. It variously included people from medical supplies, health information systems, public health technicians, Mission groups, donors (USAID, UNICEF, DFID), AMREF, WHO's regional advisor from AFRO (Dr E Afari) and the research community. It was difficult to see what practically this collection of representatives actually achieved. The minutes reflect much discussion and deliberation of various approaches to control without pragmatic solutions to the present or future problems. What the meetings did identify was the fragmented nature of malaria control in the country, the lack of specific recommendations on the management of epidemics, the need for a better co-ordinated effort from within the Ministry and the growing need for accountability. As with the epidemics of the 1920's the El Niño crisis galvanised political and Ministry commitment to malaria control in a way that routine national statistics had been unable to achieve.

The epidemic ended in April-May 1998. Throughout 1998 political and logistic intransigence prevented major changes to Essential Drug Kit compositions which continued to include predominantly chloroquine. Recognising the lack of any formal policy on epidemic

preparedness and control the National Malaria Control Programme convened an expert meeting in February 1999 in Eldoret to develop such a document. This was assisted by the funds remaining from the British Government's support during the 1998 epidemic, and involved representatives from a broad spectrum of the research and control community. Much attention was devoted to "epidemic alarms", forecasting and early warning systems using climatic data and possibly new technologies involving remotely sensed images of the planet as derived from satellites (MoH, 1999). The Plan of Action defines the need for improved health information system data collection, which was recognised during the 1998 epidemic as having almost completely collapsed in many districts. Furthermore it recommends to districts most affected by epidemics to establish a District Outbreak Management Team (DOMT). The DOMT would review resources, ensure the mobilisation of resources not available, train staff, strengthen surveillance and sensitise communities. This concept was in concert with increasing moves toward decentralisation of health management although it was recognised that national technical support would be provided from the National Malaria Control Programme. The three major elements of the strategy are provided in Appendix A.

During the 1940's and particularly during the experimental preventative strategies of the 1950's and 1960's ownership of malaria control in towns and highland areas of Kenya belonged to civil administrative councils and employers. The health sector provided support within this wider intersectorial collaboration. Recommendations for responsibility for the management of epidemics into the next millennium lie firmly with District Health Management Teams. DHMT's are encouraged within the spirit of Health Sector Reform to solicit additional support for their depleted health budgets from wider sources within their districts (Owino, 1997). However, the guidelines offer no suggestions as to how this may be achieved and what the additional costs of mobilising the strategies outlined in Appendix A will be.

The health sector in Kenya is experiencing a general decline in financing and performance (Owino, 1997). In Kenya there were remarkable improvements in reducing infant and child mortality between 1960 and the late 1980's. However, the most recent Kenya Demographic & Health Survey shows a significant upward trend in both infant and child mortality across the country with marked sub-national disparities (KDHS, 1998). Reasons for this change may be multiple but many argue that a contributing factor is the lack of efficacious drugs to treat life-threatening malaria infections in the rural communities. Within this fragile health sector malaria continues to burden the over-stretched health services in Kenya. Malaria constitutes over 30% of all GoK clinic attendance's and hospital admissions. Such figures, however, do not reflect the burden to the population at large and recent estimates suggest that approximately 26,000 children under the age of five die each year from the direct consequences of malaria (Snow et al., 1998).

The recent Roll Back Malaria Initiative launched by the World Health Organisation is a renewed effort to develop a strategic plan for countries, such as Kenya, to reduce mortality by 50% by the year 2010. Through the brokering of international commitments RBM proposes to free up resources for malaria control in Africa. Malaria, they regard as a "pathfinder" which will strengthen improvements within the wider health sector. How this will operate in areas of Kenya where periodic waves of a single parasite can devastate and disrupt the entire health service remains to be seen.

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4. The Highlands: the example of Nandi district with reference to neighbouring Kericho district.

4.1: Location and population

Nandi district is situated in the Western part of the Rift Valley Province and lies between latitudes 0.111^{o} South and 0.561 ^{o} North and longitudes 34.737^{o} and 35.435 ^{o} East. It is bordered by the districts of Uasin Gishu, Kericho, Kisumu, Vihiga and Kakamega (Figure 4.1). Although the district boundary has remained the same since the last census in 1989, there have been boundary changes within the district in response to changes in population size. In 1948 a resident population of 79,800 people was recorded. By 1962 the population had risen to 119,000 (an annual growth rate of 2.75% p.a.). During the 1979 census the population of the district was recorded as 299,319 and the population during the 1989 was 452,018 (an annual growth rate of 3.7%). The population distribution across the district varies, in some sub-locations (5th level administrative units) population density is 8.61 per km^{2} and in others it is as high as 1201.9 per km^{2}. In 1986 the district had 26 locations and 101 sub-locations (there are currently 65 locations and 204 sub-locations). The population comprise mainly of Nandi people (74%), a sub-group of the Kalenjin who have resided in the area since the 17th or 18th centuries following migration from Southern Sudan via Mount Elgon (Mwanzi, 1977).

Kapsabet town is the largest urban centre and is also the district headquarters. Its location along the Eldoret-Chavakali road makes it accessible by tarmac road to the major towns in western Kenya. The other urban centre is Nandi Hills, while other smaller centres include Ndalat, Kipkarren, Kabiyet and Kapkangani (Figure 4.1).The roads are uniformly distributed throughout the district (Figure 4.1), except in the hilly areas of Tinderet division in the south eastern region of the District and the forest areas to the west and north western parts of the district (North Nandi and South Nandi Forests). Telephone services and water supply are not well developed aside from within Kapsabet town and at present the district does not have an adequate supply capacity to meet its domestic and industrial demand for water. There are 38 water supply schemes operated by government institutions, NGO's or self-help groups. Wood fuel is the principal source of energy for domestic use and for tea processing (Nandi District Development Plan 1997-2000, 1997).

The districts physiography can be divided into five distinct features; the Kapsabet plateau, the highlands and foothills of Tinderet volcanic mass, the Kingwal Swamp in the Baraton-Chepterit area, the rolling hills to the west and the Nyando escarpment at the southern border (Kenya Soil Survey, 1986). Altitude ranges from 1800 to 2500 metres. The soils in most of the district are deep, well drained and very suitable for crop cultivation thus agricultural activities form the backbone of the economy. Due to the adequate and reliable rainfall, the district has the potential to produce a surplus of diverse agricultural crops, including tree crops, horticultural crops, pyrethrum, cereals and fruit trees. Agricultural production plays a major role in the development of commerce, trade and services. The industrial sector mainly consists of agro-based industries such as tea and milk processing and saw-milling. The bulk of the formal employment labour force is in the public sector and the tea estates.

There are 443 primary schools and 57 secondary schools in the district. Level of school enrolment is the same for both boys and girls and in 1996 there were 135,635 children attending primary school. About 71,305 persons in 1996 had no formal education. 36.2% of these did not go to school for traditional or cultural reasons and 21.4% because of lack of interest. Kapsabet district hospital is the main Government Hospital and has 7 wards including 2 male wards, 3 female and maternity wards and 2 paediatric wards.

Infant mortality stands at 67/1000 live births and is similar to the national average (62/1000). The crude birth rate and total fertility rate for the district between 1979 and 1989 stood at 50.3 and 7.6 respectively. Expectation of life is 66.2 years for males and 64.7 years for women while the Crude Death Rate (CDR) is 8.8/1000 population (Central Bureau of Statistics, 1996). There are few reported cases of malnutrition (only 956 in 1995) since the district produces a surplus of almost all the basic food stuffs such as maize, beans and horticultural crops as well as milk.

4.2: The emergence of malaria in Nandi

The Nandi plateau was regarded as a prime area for early European settlers because it contrasted the malarious swamps of nearby Uganda and Nyanza. Indeed during the "pacification" campaigns between 1895 and 1906 medical officers reported not seeing mosquitoes and rarely reported on malaria among the troops stationed there (Matson, 1957). In 1901 Nandi was part of Uganda and was used as an area for convalescence for Colonial officials at a purpose-built sanatorium, because, in the words of Sir Harry Johnston, "*malaria does not exist in Nandi*" (Matson, 1957). Matson examined the colonial records of the area and experiences of the Nandi elders. Local traditional herbal remedies for fevers had not been developed to the extent that they had in other parts of Kenya. The Nandi word for fever "*cheptigonet*" was also used to describe East Coast Fever in cattle. Matson concludes that during the 19th century local transmission was either non-existent or negligible and that despite the presence of slave and ivory trade routes through the Nyando valley, Nandi was probably free from malaria until 1918 and protected from endemic areas by the high escarpment.

It is most likely that malaria was introduced following the opening of civil and military posts, building of Scatler's road to cater for the coast, passage and settlement of troops, police and traders to Uganda. Military encounters in Nandi involved between 1,000 and 4,000 non-Nandi troops from 1895 onwards. Increasing trade and transport led to major population movements from 1906 onwards. Trade caravans would often included in excess of 3,000 Ugandan porters. These economic, conflict and demographic transitions would have led to increases in epidemic potentials arising from new infectious reservoirs and the introduction of vectors.

With troop re-settlement in 1918-1919 following the First World War there was a malaria epidemic which followed the influenza pandemic. According to Matson (1957) when the Nandi returned from the War the word "*homa*" and "malaria" was introduced into local vernacular. The following years witnessed further development of this region including the development of the Ugandan railway from Mau to the intensely endemic lake side regions allowing increased "passage" of vectors. By 1927 the road between Kisumu, Aimosi and Nandi was completed. According to Matson (1957):

> "[Its during] *this period of great activity below the South Nandi escarpment that the vectors in this area became infective, and the reservoir of infection built up, from which, gradually, over the next 20 years, infective mosquitoes and malaria spread up on to the Nandi plateau."*

In 1928 an investigation of an epidemic involving 1,727 hospital cases was undertaken. Since then "*the malaria epidemic has become an annual event, and has caused a great deal of invalidism, reduced production, and probably had a considerable effect on progress, education and reproduction*" (Matson, 1957). Epidemics were reported in Medical returns during the years 1931, 1932, 1934, 1937 and 1940 (Roberts, 1964b).

4.3: Epidemic control in Nandi district.

In 1951 an intensive control (eradication) campaign was launched in the district to eliminate malaria as a serious health threat. This pilot project was launched and sponsored by UNICEF and WHO who viewed the project as a precursor to widespread eradication initiatives across the continent in concert with recommendations made during the Kampala Conference in 1950. As part of the Nandi project detailed household and health service maps were developed, baseline epidemiological and entomological work established, training of malaria control teams and community orientation through public meetings. These activities occurred between 1951 and 1953. The intervention began in 1953 and included two phases 1) mass drug administration using Daraprim (pyrimethamine) to the entire population; and 2) Dieldrin residual house spraying.

During the MDA phase (1953-1954) of the campaign epidemiological baseline data were used to schedule the timing of the two-rounds of population treatment and coincided with anticipated rises in infectious reservoirs within the population. It was estimated through censuses that among the target 83,000 people in the district approximately 79,000 (95%) people received curative doses of Daraprim. In addition a further 22,000 people living in the Nandi Hills Settlement, Uasin Gishu and Kiamosi district borders were included in the MDA programme. Roberts (1964a) estimated that the total cost (including drugs, equipment, transport and salaries was £640 each year or 1 ¾ d per person. Following the second round of MDA (May 1954) the parasite rate in the population was zero and rose to 7.7% by the end of the transmission season compared to an increase from 21% to 52% in a neighbouring control area (Roberts, 1964a). Both WHO and UNICEF had mixed responses to MDA studies elsewhere in Africa and appeared more in favour of the potential for phase II using insecticides. However, Roberts attributes the comparative success of the Nandi MDA project to appropriate organisation (financial and logistics), availability of trained staff and access to services. Indeed what is clear from Medical Officer annual reports to the Provincial Headquarters was that the entire district organisation (administrative and medical) and the community themselves were partners in this co-ordinated effort proving the necessity for well developed inter-sectorial and community partnerships for effective control.

The residual house-spraying campaign began in 1954 as a collaborative effort between WHO, UNICEF, the Kenyan MoH and the Nandi African District Council. Both the District council and the MoH were to share the cost of mounting the programme and were instrumental in its conception and design. The project was mounted at Nandi Hills, the Turbo and Kipkarren areas of Uasin Gishu and the Kaimosi farming area. The organisation was in the hands of the Medical Officer of Health with national MoH advisors and regional advisors from the Malaria and Vector Borne diseases Institute at Amani, Tanzania and the Colonial Pesticides Research Unit, at Arusha, Tanzania. The day-to-day running of the campaign was left to the District Health Inspector. Through allied government and local administrations, public awareness campaigns were mounted and 100 spraymen recruited on the basis of previous service in the police, army or local Government. The campaign took place between January and March 1954 prior to the onset of the heavy rains. Three groups were organised consisting of 10 spray teams, each containing three personnel. The dieldrin was provided at a dosage of 60 mg/sq ft. (constituted from 50% wettable powder) each year. However it was determined that the delivered dose was only 45 mg/sq ft for a variety of practical problems. Nevertheless, the parasite rates between 1954 and 1962 showed that the spraying operations were able to reduce the infection rate to below 3% between 1955 and 1962 (Roberts, 1964a). The MDA had left only a small residual level of infection (8%) without affecting vector abundance and it is

conceivable that the subsequent dramatic success of dieldrin on infection rates was a direct consequence of such a small initial infectious reservoir. The use of Dieldrin alone in tackling infection risks in endemic Nandi district in 1953 was not tested. The spray campaign virtually eliminated in-door resting *An.gambiae* and *An. funestus* and no resistance to dieldrin was noted. However, houseflies (*Musca domestica*) developed resistance after the first round of spraying.

The combined costs of implementing the spraying operations to cover 112,500 people for three years was as follows:

Dieldrin at KES 8/= per lb	£15,600
Transport (depreciation plus cost of maintenance)	£6,000
Staff costs (including advisory)	£7,000
Equipment (ancillary)	£2,000
Contingencies	£1,500
Total cost	£32,100

This was translated to approximately KES 1.6 per annum per head of population (1 UK shilling 7 ¼ d). Nevertheless as Roberts points out £10,000 per annum represents the total amount of money available to the medical and health services in the district (Roberts, 1964c) and he is quick to highlight MDA cost the district only £600 (Roberts, 1964a). The problems of aiming at eradication during epidemic control according to Roberts were two-fold:

1. Large population movement from endemic neighbouring areas,
2. Movement of Nandi peoples to neighbouring areas.

These problems of population mobility were echoed not long after the "project" phases of the spraying campaign had come to an end by the Medical Officer from Nandi Hills Hospital in his 1966 annual report (Anon, Annual Report 1967):

> *"malaria accounted for more notifications than all other infectious diseases put together".. "malaria continues to be a problem in the area. Many of the estate employees visit their relatives in Nyanza and bring back malaria with them."*

It is interesting to note that despite the "experimental" approaches and dramatic effects of house spraying there are few descriptions to such continued activity from the mid-60's onwards from either Annual reports from the District's Medical Officers of Health or the DVBD station heads. Far more emphasis appeared to be given to the use of chemosupression as a strategy but with mixed results

> *"Once again this year* [1965]*, with the help of 50,000 UNICEF chloroquine phosphate we carried out an antimalarial campaign on the estates* [Chemase]. *Judging by the results of random blood slides before and after we were only partially successful. In the view of the writer, it might be more economical to give a curative dose to the whole labour force three times a year and treat the families as and when they contract the disease. The movement of families to and from the reserves precludes any success for a comprehensive campaign."* (Roles, 1966).

> "*Chemoprophylaxsis* [at Chemase] *an average of 996 people received drugs approximately every fortnight. Distribution work, though well organised was very discouraging due to poor attendance and was purposively stopped at one point between June and July 1969*" (Esmail, 1970).
>
> And in 1972: "*A total of 4470 chloroquine drugs were distributed in Chemase Malaria Control Scheme in the first round, 4135 in the second round and 8,605 total*" (Patel, 1973).

These UNICEF sponsored campaigns obviously met with limited success. In 1972, 95/541 (18%) people who had received chloroquine were found to be positive for *P.falciparum* soon after prophylactic courses. And in 1973 DVBD screened 1,755 people at Chemase of whom 41% were found to be harbouring *P.falciparum*. To maintain weekly chemosupression among a widely dispersed and mobile population would have involved a logistic exercise far in excess of that required for single round MDA. Studies by DVBD demonstrated the failure of chloroquine chemoprophylaxis in the Chemase area of the District and the low levels of population infection achieved during the MDA and spraying experiments were never achieved again during the 1970's, 1980's and 1990's.

The epidemics in 1990's are described for the general communities located in the highlands in Chapter 3. The '*outbreak of highland malaria'* reported in June/July, 1990 was said to have been contained within Aldai, Mosop, Kilibwoni, and Tinderet Divisions. Control activities carried out during the outbreak included community health education, routine blood examination, spraying of homes and schools with Reskol and larviciding with HS oil. There are few documented reports of what proportion of the population were covered by vector control measures however it was reported that 12,513 people received full courses of anti-malarial treatment (DVBD, 1990). Between May and August 1998, after the El Niño epidemic an trial of ICON 10 Wettable Powder residual house spraying was used in the population (approximately 6,000) residing in 1,397 houses of an area served by Lelboinet dispensary and Mosoriot Rural Health Training Centre (Zimba, 1998). A control area was selected of approximately equivalent size and clinical cases were monitored between May (after spraying) and August. During the 11 weeks of this non-randomised trial 449 confirmed cases of malaria were described from the control area compared to 79 from the sprayed area. No comment is made about how these impressive results were to be fed into future control strategies in the district.

4.4: Changing disease patterns in Nandi district since 1950

Records kept at the Kenya National Archives in Nairobi were used to identify Medical Officer Annual reports to the Provincial Medical Officer from Nandi district between 1952 and the late 1960's. Particular attention was given to locating annual admission records for Kapsabet district hospital (KDH). These reports often reported separately the numbers of admissions with "sub-tertian malaria". These data were combined with those presented by Garnham (1948) who used abstracted records at KDH between 1938 and 1946. Medical Officer Annual reports were identified at a store in Kapsabet hospital which provided annual totals of malaria admissions during the 1970's and early 1980's. In April 1999 a more comprehensive investigation of all admission registers was undertaken at both KDH and NHH. All registers between 1986 or 1988 and 1998 were retrieved from stores and offices around the hospital. The early data series (1938-1985) represent discharge diagnoses. Conversely the more contemporary data represent admission diagnoses. It was not possible to establish discharge diagnosis from the ward books as these are recorded on the individual patient records and

only summarised quarterly by Medical Records Officers (MROs). Nevertheless a sampled series indicated that there was very little variance between admission and discharge diagnoses. Throughout the entire series it is not possible to gauge the reliability of diagnosis and whether microscopy was used to confirm clinical impressions. It is conceivable that clinical criteria may have changed over time and there is no way these changes can be allowed for. Furthermore the hospital data will reflect a wide range of patients and include those who are residents of the district but acquired infections elsewhere or a minority of patients from outside the district. What the data series do represent is the in-patient burdens treated as malaria at KDH since 1938.

The patient burdens to KDH were significantly reduced during the period of aggressive MDA and vector control, 1953-1961 (Figure 4.2). Nevertheless it was noted in the Medical Officer's report for the year 1953 that "*clinical cases of fever are rare – but they are often severe*". In 1955 the MO reported that "*malaria has this year been conspicuous by its relative absence*". From the late 1970's malaria admissions to KDH had returned to levels described during the 1940's and early 1950's. However, the pattern was one of a consistent patient burden between years. One must also remember that there had been a four-fold increase in population size during this period of comparison and it is difficult to provide a population-at-risk corrected patient burden estimate for these periods. During the early 1990's significant increases in patinent burdens were demonstrated, suggesting epidemics. Data for 1990, 1991 and 1995 show that malaria accounted for 23% (8586/37,961) of all admissions and 21% (345/1645) of all the deaths at KDH (HIS, MoH, 1999). By way of comparison with the period of aggressive parasite and vector control malaria only accounted for 2.1% (12/574) of deaths during the years 1956, 1957-58 and 1960 (MO Annual Reports). Perhaps of greater significance between the 1950's and 1990's is that the case-fatality of admissions has risen from 0.9% to 4%.

The acutely seasonal nature of the clinical burdens is clearly demonstrated. A large peak ("epidemic") was noted in June-August 1991. Despite mid-year peaks in disease during 1992 and 1993 it was not until 1994 that a large case burden reappeared and was followed by a post-short rains, exceptional peak in February 1995. Two mid-year seasonal peaks in 1995 and 1996 were followed by a much larger clinical case load during the June-July malaria season of 1997. The El Niño related malaria epidemic in January-March 1998, despite the absence of data for January 1998 due to the nurse's strike, 1998 was perhaps the most serious epidemic on record in this district. The patterns of disease were, for the most part, similar for both adults and children (Figures 4.3 versus 4.4) between 1986 and 1998. However the 1998 epidemic among the adult population was far less obvious. Of particular interest is the ratio of adult-to-childhood admissions (Figure 4.5). As discussed in Chapter 6 these ratio's should provide some insights into the communities immune status. The community served by KDH, for the most part exhibit an age-specific clinical pattern of disease consistent with little acquired immunity through constant parasite exposure (i.e. the Adult/Child admission ratio is often above unity). Where the ratio does falls below unity is after periods of clinical "epidemics". There is an apparent decline in the numbers of adult admissions toward the end of the 1990's implying an increased immunity among this population as epidemics and hence localised transmission becomes more frequent and intense.

For comparison we have also abstracted data for the years 1988 through to 1998 from Nandi Hills hospital located only 30km from Kapsabet District Hospital. These data are shown in Figure 4.6. These data are consistent with those of KDH with respect to an increased burden from the end of the 1980's however they differ in the frequency of well-defined epidemics. At Nandi Hills Hospital it would appear that the sharp seasonal peak in malaria is regular annual

event and that whilst children represented a larger proportion of admissions in the early 1990's the proportion is more evenly split during more recent years.

Figure 4.2: Admissions diagnosed as malaria between 1938 and 1998 at Kapsabet district hospital, Nandi district (all ages).

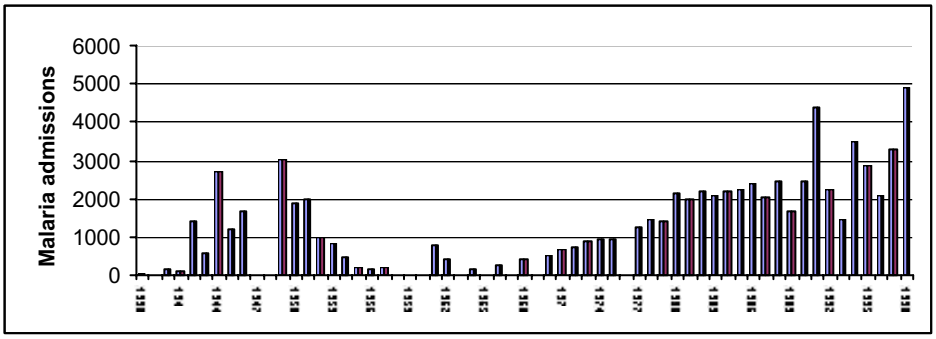


Figure 4.3: Monthly childhood (< 15 years) malaria admissions to KDH 1986-1998

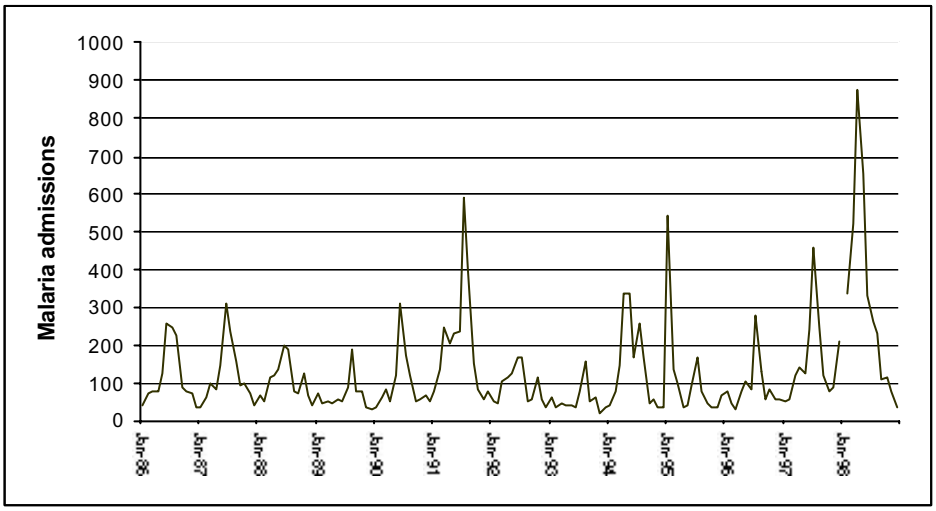


Figure 4.4. Monthly adult (>= 15 years) malaria admissions to KDH 1986-1998

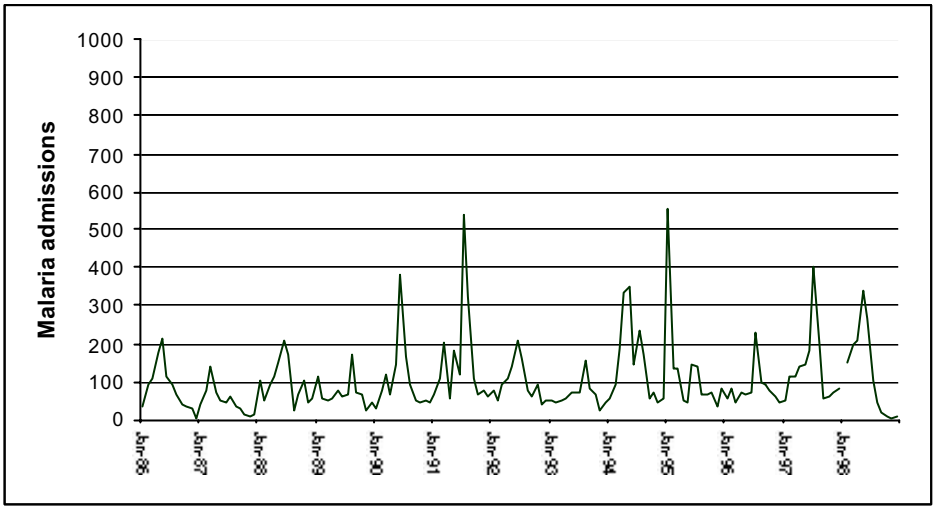


Figure 4.5: Ratio of adult-to-child malaria admissions at KDH 1986-1998.

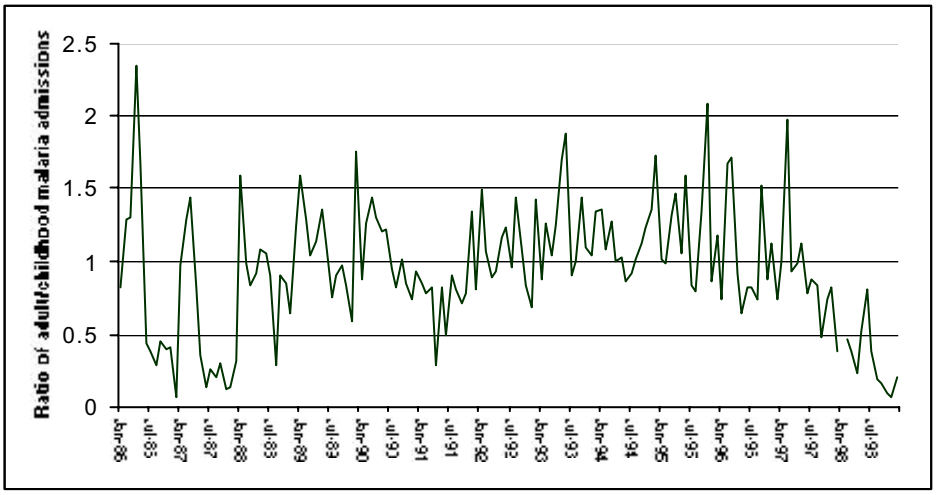
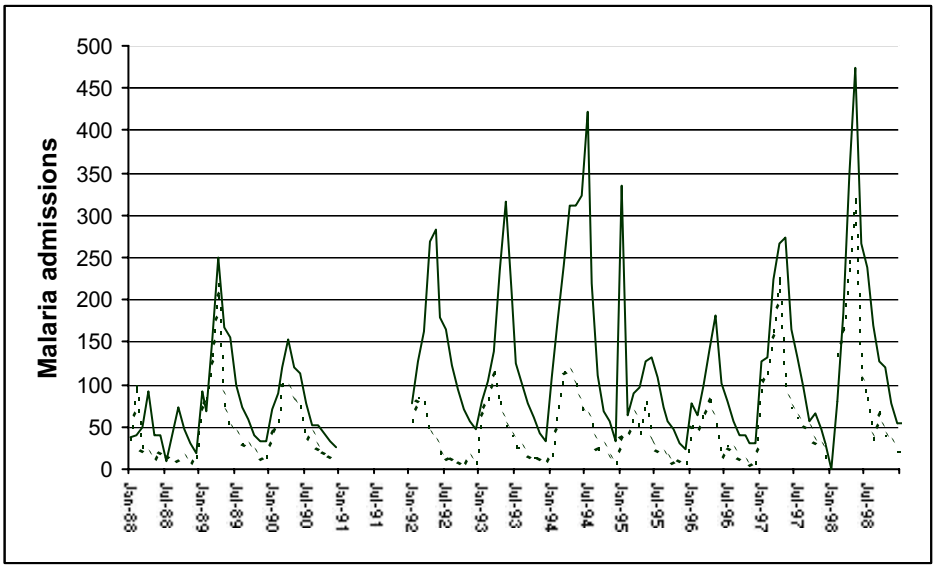


Figure 4.6: malaria admissions to Nandi Hills hospital between 1988 and 1998 (bold line admissions aged < 15 years and dotted line are adult admissions).



4.5: The Kericho example.

Kericho district (0° 22" S, 35° 17" E) borders Nandi district and is located between 1600 and 3000 metres. It is separated from the holoendemic areas surrounding Lake Victoria by a 600-800m escarpment. Malaria was thought to be introduced into Kericho District at the same time and for the same reasons as Nandi (Section 4.2) (Garnham, 1948).

The history and control of malaria epidemics between 1928 and 1970's.

The 1928 epidemic which swept the country was acutely felt in Kericho district among the residents of the Lumbwa reserve. Chataway (1929) who went to investigate the epidemic states that "*There has been no epidemic of the present widespread character within the memory of the present inhabitants*". He continues

> *"The present epidemic began early in February of this year.. there has been a combination of relative drought for the last six months of 1927 and a high*

*temperature for the first three months of 1928. No parallel exists in the detailed records since 1915…. The epidemic coincided with an apparent intense infestation of mosquitoes. It began in February and by the end of June was widespread gradually becoming less severe from then onwards… Once suitable mosquitoes had arrived, the large numbers of imported Kavirondo [*Luo's from Kisumu*] labourers, so many of whom harbour the malarial parasites, was probably of the utmost importance in the rapid spread of the epidemic through the population".*

In terms of the clinical pattern Chataway was unable to define the true extent of the problem but noted that "*employers of Lumbwa labour found themselves from 30% to 50% short handed*". He makes the important observation in relation to premunition among the varied ethnic groups of the area

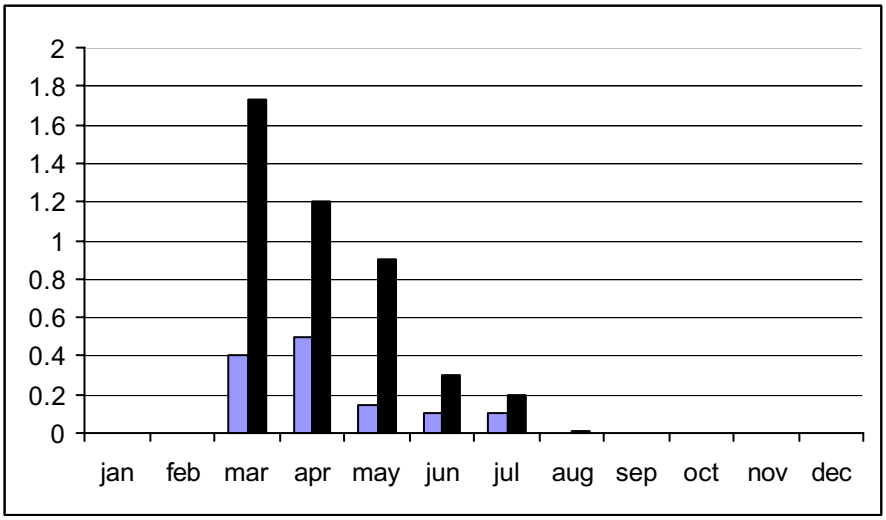
"Among the untreated there was a striking difference shown between the Kavirondo and Kisii imported labour, and the Lumbwa natives. The latter were very severely affected and died in large numbers generally of cerebral complications while the Kavirondo [Luo] *were only slightly ill and recovered without treatment in many cases".*

Chataway worked with the local administrative officers (including the police and community leaders and elders) to establish a control campaign. This campaign consisted of engaging chiefs, elders and headmen from the community for them to define members of their community to act as providers of quinine therapy. Quinine was weighed into appropriate doses and selected community members were trained in its constitution using whiskey bottles and dispensing according to the age of the patients. Using the community in the widespread delivery of therapeutic doses of quinine, Chataway regarded as the only possible means by which to contain the clinical epidemic using "*white men's medicine*". After one month 57,600 ten grain doses of quinine had been dispensed. Chataway (1929) concludes

"it would appear impossible to attempt a forecast as to the likelihood or otherwise of further widespread outbreaks of malaria in the Lumbwa reserve in the near future. It is believed that the natives are intelligent enough soon to learn to take full advantage of a hospital if it were possible to provide them with one."

Epidemics however were to become common in the area, Heisch and Harper (1949) describe the typical malaria picture in Kericho during the 1940's as typified by a "*severe annual epidemic which begins about June and reaches a peak in July or August and dies out in September*". During the two particularly virulent epidemics in 1946 and 1948 Heisch and Harper describe *P.falciparum* infection rates among the population at Buret location rising from 8% in May to over 36% by August. The significance of *An. funestus* was also described by the authors during the 1948 epidemic (Figure 4.7), with a sporozoite rate of 1.7% compared to none of the *An.gambiae* dissected showing evidence of sporozoites. During an epidemic in 1990, Ayisi et al. (1991) at Belgut Division in Kericho and showed that of the 94 vectors sampled in 4 villages at Koitaburot 60% were *An.gambiae* and 40% were *An.funestus* and the sporozoite rates were 3 and 8% respectively.

Figure 4.7: Average numbers of *An. gambiae* (grey) and *An. funestus* (black) per hut during 1948 in Buret location, Kericho district (Heisch & Harper, 1949)



A series of recommendations for environmental and insecticide management of malaria vectors in Kericho Township were made by the Colony's entomologist, CB Symes and others inlcuding Harper and Heisch. A large scale field experiment was conducted across 70 square miles of Kericho District involving DDT spraying of households and funded by the Colonial Development and Welfare Fund between 1945 and 1950. In the Director of Medical Services Annual report for 1950 he states that "*It has been demonstrated that a substantial measure of control can be carried out in an African rural area subject to seasonal malaria by an annual spraying of huts with DDT*" (Colony & Protectorate of Kenya, 1950). As with most experimental control approaches it was clear from a review of the Medical Officers annual reports during the 1960's and 1970's that the emphasis was rather on environmental management with varying degrees of enthusiasm. In 1967 the MO lists the members of the "Malaria Squad", nine in total, whose accomplishments during the year were "*weed clearing, oiling and drainage and channel clearing ..* [and].. *two others were posted to Londiani township as a result of complaints received that the incidence of mosquito breeding was high*" (Mutunga, 1968). It is hard to gauge the significance of malaria during the 1960's and 1970's (except for the estate populations see below) however a malariometric survey in two schools in Mombwo and Lebesonik areas of the district the MO reports that not a single positive slide was obtained (Fernandes, 1967). For the years where data were provided in the MO's reports (1966,1967,1970,1975 & 1976) there were 95 deaths among 5,686 malaria admissions to Kericho district hospital, a case-fatality rate of 1.7%.

4.6: The Brookebond tea estate populations at Kericho district.

The climate and soils at Kericho, like Nandi district, lends it to high agricultural potential and is economically significant because of the presence of large tea estates. The largest collection of estates (18) is owned and run by Brookebond who employ between 14,000 (1986) and 18,500 (1998) workers. Employees reside at the estate with officially (for an average employee) a wife and three children. The latter are a mobile and "dynamic" group between the employees natal home and the estate. These employees are recruited from highland (Kipsigis and Kisii) and endemic (Luo) areas of Western Kenya. Between 1942 and 1949 approximately 8,000 males were employed and housed on the estates with an additional 2,000

dependent women and children (Strangeways-Dixon, 1950). At these estates (located between 1,780 and 2,225 metres) a series of detailed investigations have been undertaken using clinical data from the estates medical services (Strangeways-Dixon, 1950; Malakooti et al., 1998; Shanks et al., submitted; Hay et al., submitted).

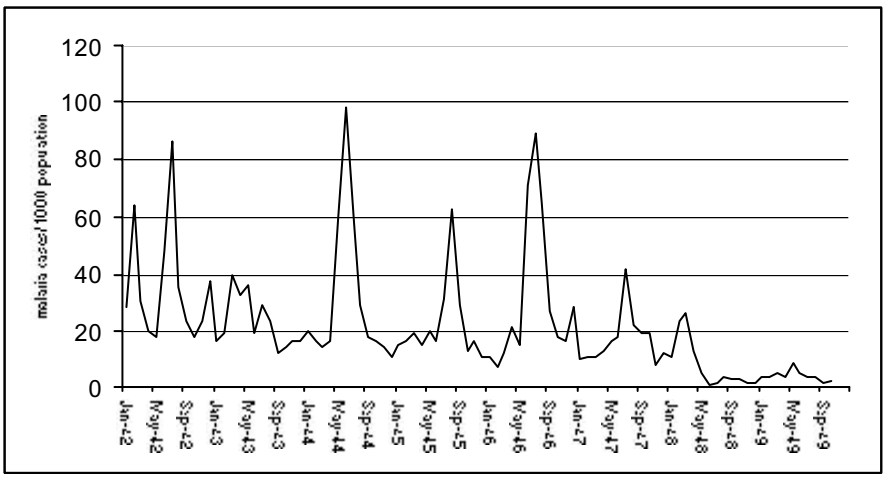
Since the inception of the tea estates by the African Tea Holdings Ltd in 1925 an extensive clinical service has been provided to the employees and their dependants. As early as 1931 Brookebond Kenya Ltd. were the sole providers of clinical care for its employees and their dependants. These services began with mobile clinics which, during the 1930's, were formed into a series of static health posts. Presently, there are 26 dispensaries and three medical centres. The company central hospital was opened in November 1955 to provide in-patient clinical care and has averaged 60 beds staffed by at least two physicians and two clinical officers.

The MO for the estates, Dr Strangeways-Dixon recorded the incidence of clinical cases of malaria between 1942 and 1949 (Figure 4.8). The advantage of this series of data (and subsequent contemporary data) is that case-burdens are expressed as risks per 1000 population-at-risk. The average annualised risk of a clinical attack among the estate population between 1942 and 1947 was 26.6 per 1000 people with peaks of 45 and 56 per 1000 people during the months of June and July respectively (Strangeways-Dixon, 1950). In March 1948 (prior to the recorded epidemic in the rest of the district) prophylactic Paludrine (Proguanil) was given twice weekly (a single 100mg tablet) to all employees. This strategy resulted in a significant decline in the incidence of malaria during June and July 1948 (0.5 and 2.0 per 1000 people respectively). In 1949 two rounds of DDT house spraying (March and June) complemented the prophylactic measures. During this period the incidence of uncomplicated malaria can be said to have been virtually controlled. Strangeways-Dixon (1950) estimates that bulk purchase of proguanil for the prohylaxsis of 10,000 people costs approximately Sh 1 and 10 cents per person.

More recently (1991-1997) at the same estate Malakooti et al. (1998) examined the out-patient records of microscopically confirmed cases of malaria and estimates the annualised risk of uncomplicated malaria to be 48.1 per 1000 population (range: 33.9 – 56.0). This represents an 80% increase in the burden of uncomplicated malaria in this population since the early 1940's and a significant rise following the descriptions by Strangeways-Dixon on the incidence of disease during active control on the estate. The contemporary rates of disease were described against a background of no significant preventative strategies (chemoprophylaxis, house spraying with residual insecticide or bed net distribution), and continued use of chloroquine as first line drug for unconfirmed cases but amodiaquine for confirmed cases (Malakooti *et al.*, 1998).

A unique series of data have been identified in collaboration with Dr Dennis Shanks (Walter Reed US Army Project, Kenya). Hospital admission registers were located in the attic of the Brookebond Tea Estate Company Hospital in Kericho. A complete series of registers from 1965 were identified and data on monthly admissions to the company hospital, by age were tallied through to 1998. Seasonal peaks of malaria admissions continued to typify the disease pattern from 1955 through to the 1980's. However the intensity of the clinical peaks increased during the late 1980's and the overall magnitude and severity of clinical disease among the estate population reached dramatic levels in the 1990's (Figure 4.9).

Figure 4.8: Rate of malaria cases treated during mobile clinics expressed per 1000 population between 1942 and 1949 at the Brookebond tea estates, Kericho. Paludrine provided to employees April-Jul 1948 and provided January-July 1949 combined with DDT house spraying in March and June 1949 (Strangeways-Dixon, 1950).



Records of the numbers of employees at the estate between 1986 and 1998 were provided by the company and used to express the risks of admission per 1000 employees during this period. If one assumes that the number of dependants has remained constant over the period the significant rise in 1990 in severe disease burden in this estate population is very evident through the 1990's (Figure 4.10). Between 1990 and 1996 the case-fatality of malaria admissions was 2.2% compared to 0.96% between 1955 and 1961.

Figure 4.9: Monthly malaria admissions to Brookebond tea estate hospital, Kericho 1965-1998 (Shanks et al., submitted).

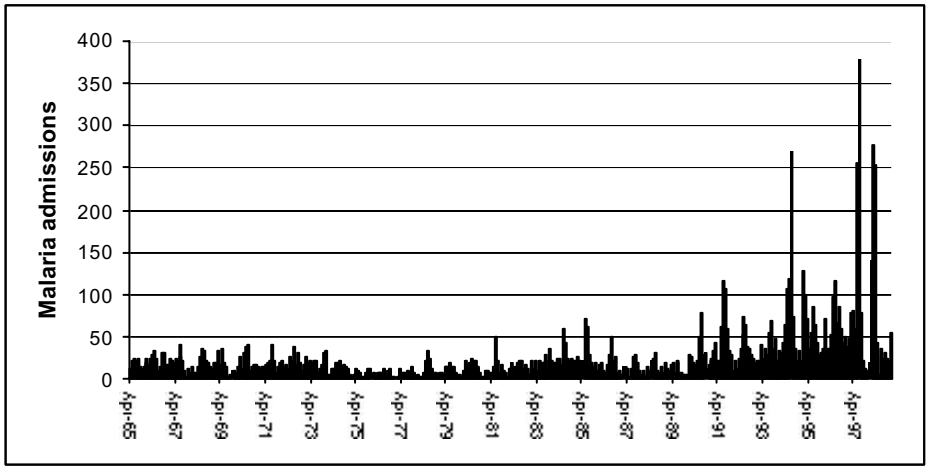
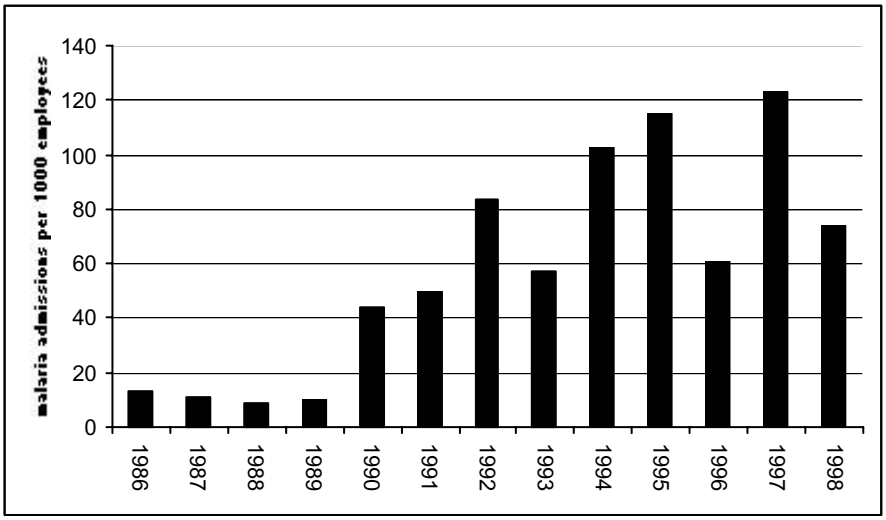


Figure 4.10: Rate of malaria admissions to the Brookebond tea estate hospital per 1000 employees between 1986 and 1997.



4.7: Climate data

Rainfall data and other meteorological variables were not consistently available from the meteorological stations in Nandi district. However, long-term, complete meteorological was available for Kericho District as a composite of data from the chief's office and the Bookebond Tea Estate. Annual rainfall between 1967 and 1997 averaged 2095 mm (Figure 4.11). In November and December 1997 there was 721 mm of rainfall. The temperate nature of this area of Kenya is exemplified by the monthly minimum temperatures (Figure 4.12). These cold climes limit parasite development in the vector. However, there is no obvious effects of "warming" in this area since 1967.

Figure 4.11: Monthly rainfall distribution between 1967 and 1997 in Kericho district

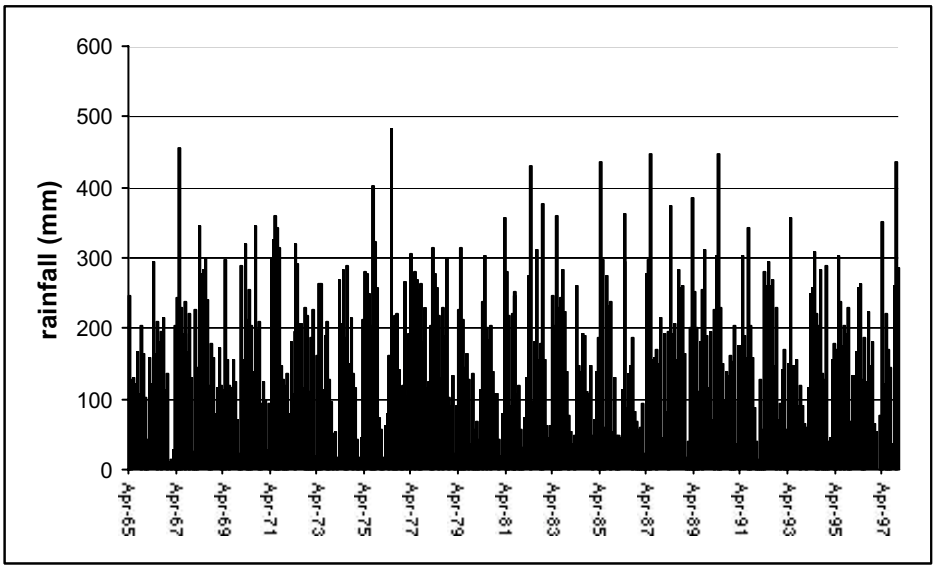
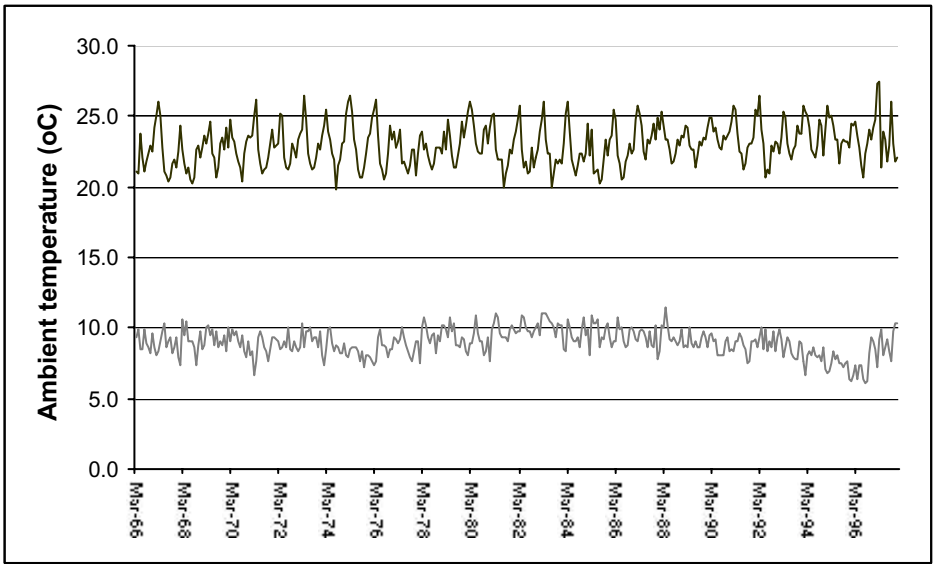


Figure 4.12: Maximum (upper) and minimum (lower) mean monthly ambient temperatures at Kericho district.



4.8: The distribution of infection and vectors in the district.

The Division of Insect Borne Diseases (DIBD), MoH, (the precursor to the present Division of Vector Borne Diseases) established a station at Nandi Hills in 1947. Entomological investigations during 1947 established that throughout the district *An. gambiae* (98%) was the principle vector with *An. funestus* (2%) playing a minor role. This observation is very different to neighbouring Buret Division in Kericho district and demonstrates the enormous heterogeneity in the transmission systems in the highlands over small distances. 78% of collections of *An. gambiae* were made during the month of July. The distribution of vectors identified through a series of surveys between 1946 and 1990 in Nandi and Kericho districts are shown in Table 4.1. and the location of the survey is shown in Figure 4.13. During 1996 an extensive search for malaria parasite ratio data was carried out covering all published surveys, surveys conducted by Non Governmental Organisations, University theses and Ministry of Health (DVBD) records, conducted in Kenya since 1929. 33 surveys were identified for Nandi and Kericho districts during this search. The data are predominantly school and community surveys of children aged between 0-15 years. These parasitological surveys conducted on children between 1948 and 1997 are shown in Table 4.2 and the position of the survey is shown in Figure 4.13. Even within a given location, e.g. Chemase, during the same year but at different schools parasite rates vary enormously ranging from 3-54%. The heterogeneous nature of infection rates is very evident. Using parasitological data among populations exposed to unstable malaria is difficult to interpret. Nevertheless, Figure 4.14. shows that cross-sectional parasite rates among randomly sampled populations are as high as 76% in areas defined as having a zero probability of stable transmission. Other areas classified as unsuitable for stable transmission (Section 2.2) have infection rates, which vary between 5 and 63%. Figure 4.15 shows the same data against altitude. Both distribution maps of endemicity are always difficult to interpret because surveys are often conducted only during epidemics.

Table 4.1: Entomological data available for Nandi and Kericho (number refers to location shown in Figure 4.13)

Number	District	Location	Survey date	Vectors identified
25	KERICHO	MUNICIPALITY	0446	A. gambiae, A. funestus
26	KERICHO	TECHOGET	'47	A. gambiae, A. funestus
25	KERICHO	MUNICIPALITY	0448	A. gambiae, A. funestus
4	NANDI	KABIYET	'53	A. gambiae, A. funestus
7	NANDI	KOSIRAI	'53	A. gambiae, A. funestus
24	NANDI	CHEMASE	'53	A. gambiae, A. funestus
21	NANDI	CHEMASE	'70	A. gambiae, A. funestus
21	NANDI	CHEMASE	'71	A. gambiae, A. funestus
16	NANDI	KEMELOI	0874	A. gambiae, A. coustani
18	NANDI	TERIK	0874	A. gambiae
21	NANDI	CHEMASE	0974	A. gambiae, A. funestus
21	NANDI	CHEMASE	1074	A. gambiae, A. funestus
21	NANDI	CHEMASE	0375	A. gambiae, A. funestus
21	NANDI	CHEMASE	0775	A. gambiae, A. funestus
21	NANDI	CHEMASE	0377	A. gambiae, A. funestus
21	NANDI	CHEMASE	1177	A. gambiae
21	NANDI	CHEMASE	0179	A. gambiae, A. funestus, A. coustani, A. maculpalpis, A. squamosus
21	NANDI	CHEMASE	0779	A. gambiae, A. coustani
21	NANDI	CHEMASE	1179	A. gambiae, A. funestus
21	NANDI	CHEMASE	1279	A. gambiae, A. funestus
21	NANDI	CHEMASE	0180	A. gambiae, A. coustani
21	NANDI	CHEMASE	0381	A. gambiae
21	NANDI	CHEMASE	0481	A. gambiae, A. coustani
11	NANDI	CHEMUNDU	0781	A. gambiae, A. christyi
11	NANDI	CHEMUNDU	0781	A. gambiae, A. cinereus, A. longipalpis
12	NANDI	CHEMUNDU	0781	A. gambiae, A. cinereus, A. christyi
21	NANDI	CHEMASE	0981	A. gambiae, A. coustani
15	NANDI	KAPKANGANI	1082	A. gambiae
9	KERICHO	SOIN	0690	A. gambiae, A. funestus

Table 4.2: Parasite ratio data available for Nandi and Kericho (number refers to location on Figure 4.13.)

Number	District	Location	Survey date	Age range (yrs)	Positive/Examined	%
26	KERICHO	TECHOGET	0548	1-10	n/a	53.9
26	KERICHO	TECHOGET	0848	1-10	n/a	9
10	NANDI	KAPLAMAI	0251	0-10	n/a	5
14	NANDI	KAPKANGANI	0251	0-10	n/a	12
8	NANDI	KOSIRAI	0351	0-10	n/a	3
16	NANDI	KEMELOI	0651	0-10	n/a	91
10	NANDI	KAPLAMAI	0851	0-10	n/a	63
1	NANDI	CHEPTERWAI	0951	0-10	n/a	76
2	NANDI	KABIYET	0951	0-10	n/a	10
13	NANDI	KAPKANGANI	0951	11-20	n/a	56
22	NANDI	KAPTUMO NORTH	0951	0-10	n/a	33
19	NANDI	MARABA	1051	0-10	n/a	47
6	NANDI	KOSIRAI	0452	0-10	n/a	10
19	NANDI	MARABA	0452	0-10	n/a	77
23	NANDI	KAPTUMO NORTH	0552	0-10	n/a	14
23	NANDI	KAPTUMO NORTH	1152	0-10	n/a	9
17	NANDI	KEMELOI	1152	0-10	n/a	34
19	NANDI	MARABA	1152	0-10	n/a	34
20	NANDI	KAPTUMO SOUTH	1152	0-10	n/a	15
21	NANDI	CHEMASE	0966	1-9	147/509	28.9
21	NANDI	CHEMASE	‘66	0-9	216/684	31.6
21	NANDI	CHEMASE	0775	4-16	105/177	59.3
21	NANDI	CHEMASE	0377	4-16	115/178	64.6
21	NANDI	CHEMASE	0779	4-16	9/116	7.8
21	NANDI	CHEMASE	1179	4-16	2/70	2.8
21	NANDI	CHEMASE	1179	1-9	102/190	53.7
21	NANDI	CHEMASE	0180	4-16	16/80	20
21	NANDI	CHEMASE	0381	4-16	18/100	18
9	KERICHO	SOIN	0690	1-9	236/379	62.3
21	NANDI	CHEMASE	0395	6-15	256/549	46.6
11	NANDI	CHEMUNDU	‘97	n/a	18/56	32.1
3	NANDI	SANGALO	‘97	n/a	23/71	32.4
5	NANDI	SANGALO	‘97	n/a	44/91	48.4

4.9: Summarising the evidence

It seems apparent that malaria was introduced into the highlands of Nandi and Kericho following the First World War and conditions were already beginning to develop for localised transmission through large scale population movement and development since the turn of the century. In many ways it seems inevitable that the geographical barriers between the intensely malaria endemic areas surrounding lake Victoria and the temperate, malaria-free areas of the highlands would sooner or later breakdown. The economic significance of these fertile areas of Kenya exposed the previously physically isolated communities (to both vectors and parasites) to a new disease. It seems reasonable to regard the epidemics described during 1918/19 and 1928 as true epidemics in the sense that these were new events for this community. What emerges thereafter is a situation best described as acutely seasonal, recurrent annual "epidemics". These conditions prevailed through the 1930's and 1940's. Authors continued to refer to these seasonal events as epidemics. Clearly the 1930's and 1940's galvanised efforts to control malaria in both districts and the term epidemic may have assisted in securing financial support in this rural area.

Toward the end of the 1940's through to the 1960's various experimental approaches for control were studied in both districts. These were not strictly community-randomised controlled trials as we know today but were more operationally-based investigations of methods involving thousands of at-risk people across wide areas of Nandi and Kericho districts. It was perhaps expected that the scale of these experiments would lend them more suitable to future, sustainable community-wide approaches. Perhaps the greatest and most "cost-effective" stratagems involved Mass Drug Administration with pyrimethamine (Roberts, 1964a-c) or weekly chemoprophylaxis with proguanil (Strangeways-Dixon, 1950). Both effectively achieved over 90% reductions in infection or disease risks and were regarded as within the financial realms of employer, medical department or district council budgets (approximately 1.75 Shillings per person). Despite these successes there was some international pressure to use residual house spraying. Whilst these also showed successes they were regarded as beyond the budgets of a district such as Nandi.

The ensuing period of the 1970's through to the 1980's witnessed a striking lack of any primary interest in malaria prevention or epidemic control in the highlands. There was certainly no evidence of any widespread use of targeted chemoprophylaxis, MDA or for that matter residual house spraying. Rather efforts continued toward environmental and disease management. At Nandi there appears to have been a period where disease incidence (as defined by hospitalisations, Figure 4.2) may have been considerably reduced compared to the 1930's and 1940's. This was certainly true for the Brookebond tea estate population at Kericho (Figure 4.9). By the early 1990's there was evidence that at Nandi and the tea estates in Kericho, malaria hospitalisations had increased significantly compared to the preceding two decades. The climatic variables shown in Figures 4.11 and 4.12 do not suggest any sufficiently gross changes over time to explain the rapid re-appearance of highly seasonal malaria of "epidemic" proportions.

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5. The arid areas of North Eastern Province: the example of Wajir district.

5.1: The location and population.

The North Eastern Province (NEP) is an arid region inhabited mainly by Somali Kenyans who live alongside other groups such as the Boran, Rendille, Galla and Samburu. Pre-independence, the area formed part of what was called the Northern Fontier District. NEP borders Somalia to the East and Ethiopia to the North (Figure 5.1). The inhospitable nature of the region did not encourage European settlement in the area. In 1909, Northern Frontier District (NFD) consisted of unofficial stations at Marsabit, Moyale and a police post at Uaso Nyiro (Archer's Post). In 1921 NFD was placed under military control with headquarters at Meru. Between the late 1920's and the 1960's the province underwent various administrative boundary changes and variously included Samburu, Turkana, Isiolo and parts of Tana River districts. By 1963 NEP was defined as Wajir, Garissa and Mandera with the provincial headquarters at Garissa.

Wajir district has neither perennial rivers nor lakes. However, seasonal swamps exist in the south including the Boji swamp in Lagh Bholol area and the Lorian swamp in Habaswein division (Figure 5.1). These swamps and their numerous drainage lines form important dry-season grazing zones and allow for some cultivation (Wajir District Development Plan, 1997). The area is classified as 100% arid, comprising thorn bush covered plains. There are a few scattered hills along the Ethiopian border. The district lies at an altitude of 150 metres in the South East to 200 metres at Buna in the North-West and 460 metres in Bute. Projected population data from the 1989 census indicates that in 1997 there were approximately 110,954 people living in 21 locations covering an area of 56,501 square kilometres. These population estimates are widely held to be under-estimates owing to the large-scale population movements across the borders during the Somalia civil war. The town of Wajir had a projected population of 17,550 people in 1997. There are only 51 primary schools and 5 secondary schools in the entire district. According to the 1989 census the life-expectancy at birth for males was 54.9 and for females was 51.9 years. Infant mortality rates were estimated at 69 per 1000 live births and crude death rates at 11.7 per 1000 population per annum (CBS, 1996).

The Somali of NEP form a linguistic and cultural continuum with populations in Somalia, Djibouti and Ethiopia and it is thought that the present day Somali of NEP settled in the area toward the end of the 19th Century. Kenyan Somali's are estimated to be approximately 300,000 and live mainly in the three districts of NEP (Garissa, Wajir and Mandera). They are predominantly Muslims of the Sunni sect who observe Shaafi'ite Islamic law (Lewis, 1965). The patrilineal society is organised around a clan system which operates at five levels: clan-family, sub-clan, primary lineage and the *dia*-paying group. The latter formed the fundamental legal basis of the wider community organisation (Dalleo, 1975). The basic survival and social organisation of the people of NEP demands an understanding of the economic and ecological conditions of the region. The environment, which is hot and dry (Chapter 2), has imposed an adaptive force on the community. Shelter, property, grazing and social institutions have all been affected:

> *"Nomadism is the prevailing economic response, and modes of livelihood and social institutions are tightly adjusted to the scant resources of an enviably harsh environment*" (Lewis, 1965)

> "*The intimate relationship between Somali nomads and their environment significantly affected their movement into northern Kenya. Because of inadequate supplies of water and pasturage, the Somali developed an*

expansionary outlook, and frequently fought with one another as well as with non-Somali people" (Dalleo, 1975).

The nomadic pastoralist Somali move regularly in search of water for their livestock. They sleep close to the animals, often in the open or in makeshift homes made from thorn trees and covered with mats. This survival strategy lends itself to increased exposure to potential vectors of the region.

5.2: Climate conditions in NEP

The environmental conditions are so fundamental to the people of this region that names have been given to the seasons. Rainfall is limited in the region, the long rains (*gu*-) last from March through to May and the short rains (*dir*-) occur between October and December. Monthly rainfall data were purchased from the National Meteorological Office from 1932 through to 1998 for the Wajir meteorological station. Complete years were not available for 1941, 1942 and 1959. The remaining data indicate three major "heavy rainfall" years since 1932 at Wajir: 1961/62, 1968 and 1997/98 (Figure 5.2). The El Niño (the little boy or Christ's child) Southern Oscillation is a phenomena which results from anomalies in sea surface temperature in the equatorial Pacific Ocean and effects global weather patterns. Its effects during 1997-98 in Eastern Africa (particularly the Horn of Africa) led to a rainfall precipitation 600 times greater than normal (Figure 5.3). International organisations such as the National Oceanographic and Atmospheric Administration (NOAA) and NASA were able to predict the onset of this event with a high degree of confidence and a lead-time of 6 months. La Nina is the resultant climate patterns observed following El Niño and often results in opposite patterns of rainfall (notably drought). The extent to which these "warnings" were conveyed to the health sectors in the region needs to be reviewed.

The mean annual rainfall for the period 1932-1998 was 304.6 (SE = 24.0) [Median = 256.7]. Temperatures are always high with very hot seasons (*hagai*) between June and October and between January and March (*Jilal*) (Figure 5.4). The area has one of the highest evaporation rates in Kenya leaving the region permanently arid.

Figure 5.2: Annual rainfall figures (mm) for the period 1932-1998

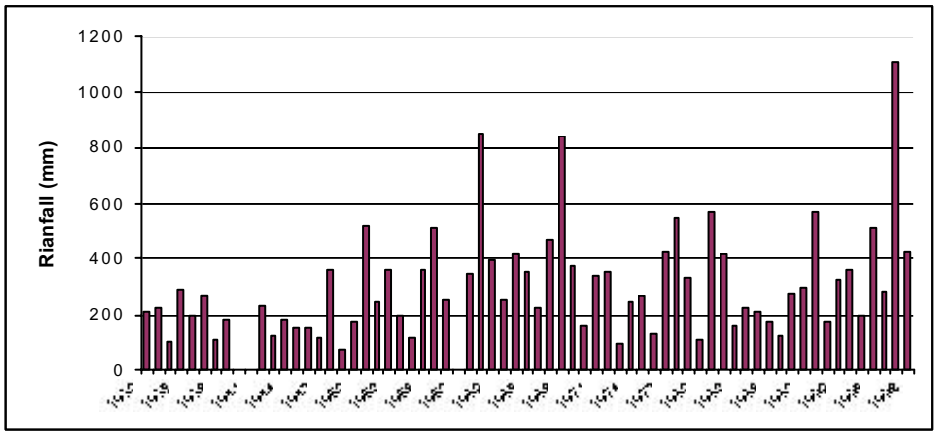


Figure 5.3: The monthly rainfall (mm) pattern at Wajir between 1991 and 1999.

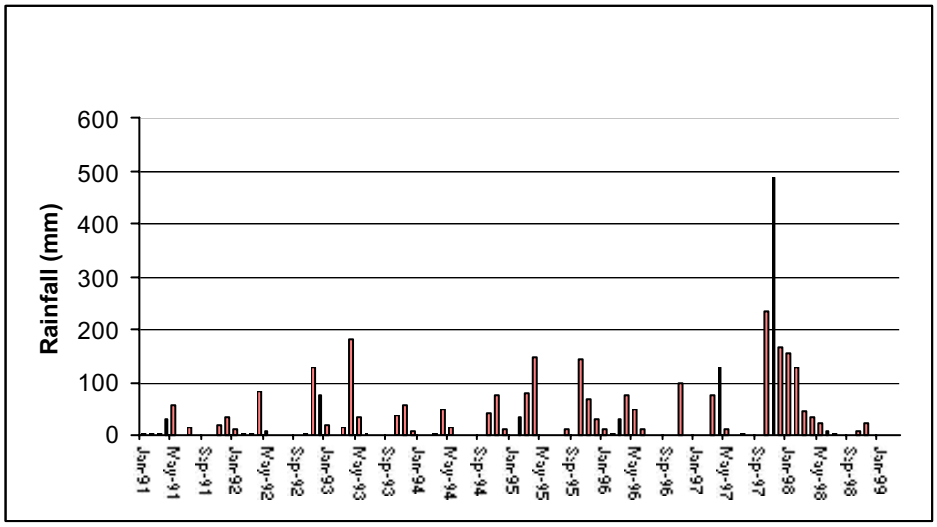
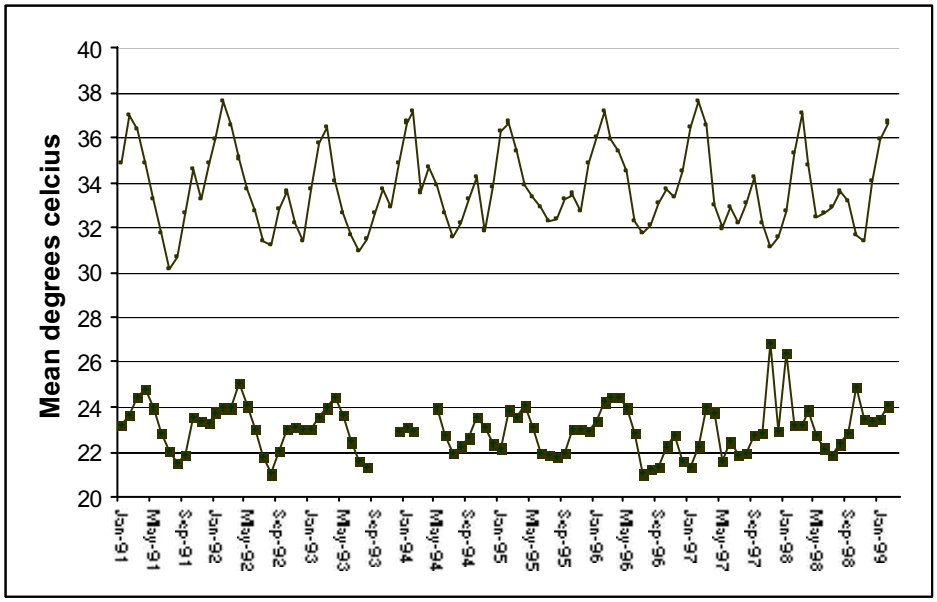


Figure 5.4. Mean monthly maximum and minimum temperatures at Wajir 1991-1999



5.3: Health services in Wajir district and the Province

The Government provision of medical services and facilities was poor throughout the colonial period. What efforts that were made in providing more sustainable health care were given to the settled communities or the Oromo, Boran and Rendille, the remaining population were viewed as

> "*Probably not very greatly in need of medical attention, and in any case they are too scattered to enable much to be done, but something should be done for the settled agricultural tribes on the Tana River*" (Provincial Commissioner's Annual Report, 1925).

By 1931 three medical officers had a responsibility for the entire NFD. In 1935, an attempt was made to better organise the medical structure of the NFD with a base in Wajir and responsibility for Garissa (which until then had no medical facilities). However, by 1940 "*the only civil medical officer in the NFD was removed, and the one sub-assistant surgeon was given a roving commission*" (Provincial Commissioner's Annual Report, 1940). During the 1940's and 1950's there was one Medical Officer and one sub-assistant surgeon. Dispensaries were run by African dressers who were visited by the MO once every three months. The nomads outside of towns had to travel days on foot or by camel to reach any formal health services. The development of the rest of the colony's health services through tax revenues and development funds were not applied to the NFD. Such central government neglect continued after independence. Mission activity was minimal in the area given the Islamic following by the majority of the population.

The first hospital in Wajir was built with the help of Italian Prisoners of War in 1944 under the direction of Dr R Heisch. Heisch spent 5 years in Wajir (1942-1949) and was of the opinion that the only way to generate confidence among the population was "*travelling amongst them and distributing medicines*" (Heisch 1947). Despite a national move in the 1950's toward providing rural health care through mobile health units and rural health centres this proved virtually impossible for NFD

> "*For a population of some 140,000 there is one Medical Officer stationed at Wajir, and one Assistant Surgeon stationed at Moyale. This is not exactly a high proportion particularly since it is understood that the average for Kenya is one Doctor per 10,000 head of population*" (Provincial Commissioner's Annual Report, 1955-57).

In 1961 African District Councils were formed in NFD to maintain Mobile Health Units. These were manned by health assistants, dressers and midwives and travelled the district in one of three vehicles provided by UNICEF. Nevertheless these must have made only a small impact on the health of the widely scattered populations of districts like Wajir.

The situation has improved little since the 1950's. Other than the district hospital at Wajir there are only 20 Government health facilities in the district mostly dispensary grade. One clinic is sponsored by the Africa Inland Church and 2 supported by NGO's . In 1996 there was only one doctor in the entire district (Wajir District Development Plan 1997-2000, 1997).

5.4: The malaria situation in Wajir district.

As demonstrated in Chapter 2 the NEP and particularly Wajir does not, on an average year, have sufficient rainfall to allow for stable *P.falciparum* transmission. These models (Craig et

al., 1999) assume that a minimum requirement for vector proliferation and survival to complete transmission is three consecutive months of rainfall above 80mm. Since 1932 this condition has not existed except for the 1997/98 El Niño rains. More recently models have been developed which assume a minimum of 60mm of rainfall within a sliding average of three months (Tanser et al., in preparation). Other than the recent El Niño phenomena these conditions only existed during 1951 and 1961/62. Despite constantly suitable temperature conditions for parasites and vectors rainfall limits the stable transmission of *P.falciparum* in this area.

Heisch was a self-taught parasitologist and entomologist with a keen interest in malaria. During his report on two years medical work in NFD (Hesich, 1947) he states:

> "*The disease has a wide distribution in the NFD but the endemic foci are often many miles apart. After the rains, which are short and erratic in character, there is often a great temporary increase in the incidence of malaria and epidemics are common. There is little immunity to malaria amongst the Somalis and the disease is severe in both adults and children. The malaria in the NFD is mainly seasonal, and large epidemics are confined to the rainy periods as is usual in a semi-desert country*".

During an ad-hoc malaria survey among residents of Sericho and Gubato (100km from Wajir) he noted that none of the 20 people examined had *P.falciparum* in September 1942 and 2/58 (3.4%) had *P.falciparum* in February 1943. During what Heisch calls a localised epidemic in March 1943 at Bulfya (120 Km from Wajir) 7/24 (29%) people were infected with *P.falciparum* and infection rates at Buna in February 1945 were 6% (3/50). For Wajir, itself he states that

> "*There is no malaria at Wajir and the only species of mosquito seen was C. fatigans which on several occasions appeared in large numbers after breeding in a leaking well. In April 1944, 182 children (1-10 years) and 32 young adults (11-15 years) were examined in Wajir; 9 per cent of the former and 12 per cent. of the latter had enlarged spleens. The people examined were Somalis who had settled in villages nearby, and most of those with enlarged spleens had recently visited malarious places. Although Wajir appears free from malaria the disease probably exists in the vicinity"* (Heisch, 1947).

The situation was quite different closer to Isiolo. Heisch (1947) describes the epidemic of 1943-1944 among Somali's who had settled there after the First World War and had lost their nomadic way of life. The epidemic began in January and reached a peak in February-April (Figure 5.5). Following the epidemic, in June, the *P.falciparum* parasite rates among 145 adults was 37% and among 182 children was 42%. *An. gambiae* was the only vector and although *An. funestus* did exist in the area it was at very low levels. During the 1997/98 epidemic the *An. gambiae* complex were described as predominantly *An. arabiensis* (DVBD, unpublished observations).

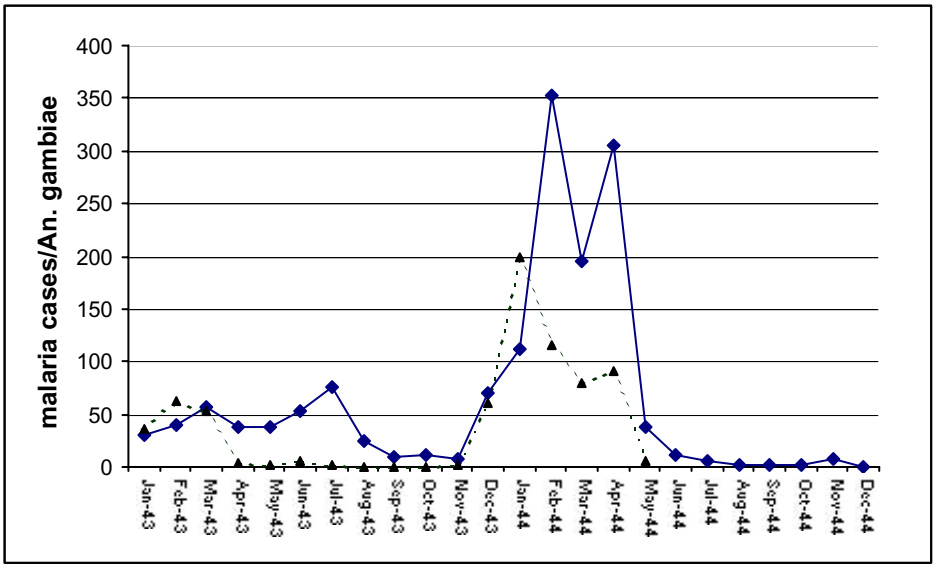
Charting the history of malaria epidemics in the region surrounding Wajir has proved difficult due to the scant reporting in the records kept by Provincial Commissioners, District Commissioners and Medical Officers. According to the Provincial Commissioners annual report of 1930 following "*heavy rains*"

"*everyone suffered severely from malaria. So serious was this in many areas that stock remained outside the villages because there was no one well enough to herd them… several hundred deaths must have occurred from it*."

Between 1940 and the 1960's, malaria control activities were sporadic and unfocussed. In 1947 DDT spaying was used in some parts of the province and in Wajir had formed part of activities undertaken by the military during the Second World War. There are several reports of DDT soaked balsa wood blocks being suspended in wells to reduce malaria vector breeding. Paludrine was routinely used by military personnel and civil administrators but not generally extended to the population at-large. However, there were occasional reports of the use of Chloroquine and Paludrine as chemoprophylactics among school children in Garissa during the late 1960's. Despite the obvious nature of breeding sites concerns were raised over oiling and larviciding activities in 1967

"*Throughout the whole province pans have been constructed as the source of water supply. It is difficult to treat these pans without causing harm to the animals and people who drink this water. In any case Garissa is the only town which has proper treated and piped water while Wajir and Mandera rely on shallow wells. In Wajir, rain water is used for drinking*." (Provincial Commissioner's Annual Report, 1967)

Figure 5.5: Monthly distribution of malaria cases (bold line) and *An.gambiae* (dotted line*)* recorded during the 1943/44 epidemic at Isiolo (Heisch, 1947).



5.5: The 1961-1962 floods

The climate data shown in section 5.2 clearly shows the heavy rains which began in September 1961. This exceptional rain affected the entire country and was particularly devastating in its flooding of the Tana River. A reporter for the Daily Nation (2nd October, 1961) reports of the Tana River

"*for the first time complete devastation which has overcome hundreds of native huts, and completely ruined crops in the area*"

Previous droughts across the country had led to famine in the area and relief food drops were already being organised by the RAF and funded through a national Famine Relief Fund. The Ministry of Health was prepared for a malaria epidemic. As reported in the Daily Nation on 21st November 1961 the Director of Medical Services (DMS) stated that "*the department is ready to either spray or provide anti-malaria pills in any area that is threatened in the next few months*". There are few clinical data available to cover this period in Wajir. However, the monthly return for December 1961 to the Provincial Medical Officer indicates that in Wajir there was a 70% rise in hospital admissions over the proceeding year. The DMS reported that during 1962 the malaria epidemic was a "*major public health affliction during the year*" and Wajir was one of the two worst afflicted areas. Free distribution of chloroquine tablets was initiated with a total of 205,000 being used in Wajir and Garissa Districts alone. He continues that it was

> "*impracticable to impose preventative measures.. as we do not possess equipment or insecticides for large-scale mosquito work*" (Government of Kenya, 1962)

There appeared to be no external assistance to the Province other than the previous donation of three vehicles by UNICEF to act as mobile health units (maintenance costs were the responsibility of the districts themselves).

5.6: The El Niño crisis 1997-1998

Between the end of the long rains of 1996 and throughout most of 1997 there was a drought in the NEP region and particularly in Mandera and Wajir. This led to crop failures and consequent malnutrition among the population. OXFAM and Medecins San Frontiere were working in the region providing basic nutritional and drought relief support. The unprecedented rains resultant on the El Niño phenomena began in October heralding the onset of the usual short rains. In October, 235 mm of rainfall was recorded at Wajir, close to the entire rainfall recorded during 1996. In November 1997, 489 mm of rainfall was recorded and this was followed by 166, 153 and 130 mm of rain during the subsequent months of December, January and February. By March 1998 rainfall was only 44.7 mm. This exceptionally heavy and protracted rainfall resulted in the devastation of roads, villages, crops and cattle. An estimated 80% of goats/sheep and 50% of camels were lost (Carrington, 1998). What basic infrastructure that existed in the area was ruined. The roads from Isiolo and Garissa to Wajir were virtually impassable with four-wheeled vehicles. Many of the Wajir community vouched that, in their living memory, they had never seen lakes where lakes now appeared and older members could only relate the situation to what prevailed during the early 1960's. Several key factors resulted in a desperate health problem:

- The population sought refuge from their devastated homes in drier areas and became very mobile and hard to locate.
- The Christmas period left many non-native Wajir administrative staff outside of the district and unable to return.
- Since December 1997 there was a general nation-wide Nurses strike which ended only during the second week of January and Nurse's of Wajir were unable to return to work if they were in Garissa, Isiolo or elsewhere.
- Medical supplies were limited and usual routes of supply were destroyed.
- National and International attention was focused on a possible outbreak of Rift Valley Fever.

With respect to the latter, the proliferation of mosquitoes in the area had led to several reported cases of a haemorrhagic-like disease among isolated communities. As usual these reports captured the nations attention with widespread press coverage. The exotic viruses, such as recent Ebola outbreaks in the Democratic Republic of Congo, were popular press cover and stimulated international concern. The months of November 1997 and December 1998 witnessed a plethora of international experts from CDC, Atlanta, the World Health Organisation, Geneva and the Institute Pasteur, Paris descending upon Nairobi and foraging out to the remote areas of NEP. Local research organisations such as AMREF, KEMRI and the MoH were called upon to assist these international teams in uncovering the mysteries of a handful of "interesting" clinical cases. What seemed to had gone unnoticed was the less exciting but more devastating effects of another vector-borne disease, malaria. The Daily Nation newspaper ran a headline on the 12th February 1998 shown in Figure 5.6.

Figure 5.6: Daily Nation, Thursday 12th February 1998


DAILY NATION

The newspaper that serves the nation

No. 11570, Nairobi, Thursday, February 12, 1998 Price KSh25/00 (TSh400/00)

Panic as 1,500 die of malaria

Epidemic killing 45 a day in Wajir

By MOHAMMED ADOW and HENRY NYARORA

The malaria outbreak in parts of Kenya has taken a heavy toll in Wajir District, where health officials report it has claimed more than 1,500 lives in the past two weeks.

BUNDUKI NI YA KUUNDA SIO YA KUUA

STOP

ONE PEOPLE ONE NATION

INSIDE

Issues behind the violence in Rift Valley

PAGE 15

Kenya for diplomacy in Iraq issue

BACK PAGE

Kabila snubs Jesse Jackson

PAGE 8

Latest results

The Medical Officer at Wajir (Dr Abdirizack Sheikh Mohammed) spoke to the Daily Nation by phone and claimed that "*there is no decrease in the number of patients. Indeed they are increasing every hour and right now I have more patients in the open yard than in the wards*". The article continues with comments for an opposition MP for Wajir West, Mr Aden Keynan

> *"The Government has done nothing for the people throughout the time they were battling with floods and now epidemics. Its surprising that Government Officials are only good at denying the misery and suffering currently affecting Wajir residents. I would like to point out that North Eastern Province Residents are no longer willing to be taken for a ride. The Government should act now to eliminate suffering"*

The national press continued with the malaria coverage in NEP. The following day the Minister for Health, Mr Jackson Kalweo, commented in the Daily Nation on the figures quoted for Wajir where he queried their validity. The Minister said that the international team of Rift Valley Fever experts based in Garissa had on Wednesday brought him "*encouraging reports*" on the public health situation in the province (Daily Nation, 13th February 1998). The

same article quoted the Director of Medical Services as querying the figures but the article pointed out that neither the DMS or Minister could provide any "correct" figures saying that they were still compiling "*a comprehensive report*". The disputed figures provided by the MO and NGO's are discussed below (section 5.7.). Meanwhile The Office of the President's National Disaster Operation Centre promised helicopter support to Tana River and "*will move to North Eastern Province sometime thereafter*" (Daily Nation, 13th February 1998). The disputed figures featured in subsequent newspaper reports and on February 26th 1998 the Daily Nation reported on the situation in Marasabit:

> *"Many patients are said to be in coma owing to anaemia and dehydration. The death toll has now reached 142, according to a community health worker based in Bubisa area, Mr Guyo Sake. Some of the dispensaries in the division lack clinical officers to prescribe drugs to the patients. "we treat more than 300 patients who are ferried to the dispensaries on camel back daily". Villages which are 15 kms from the trading centre are the worst hit as nurses are unable to leave the dispensaries unmanned. Councillor Sori Orto of Bubisa said lack of medical personnel had contributed to the high mortality rate."*

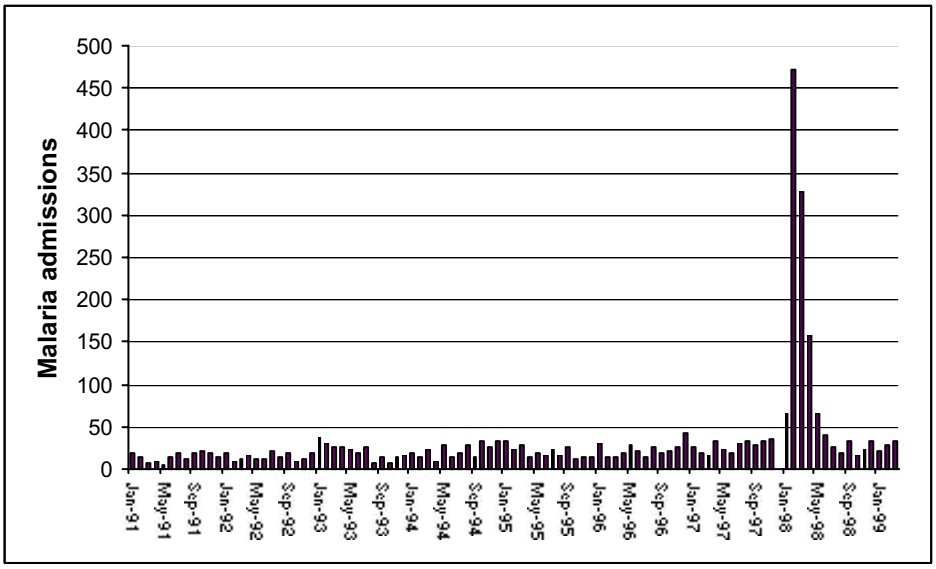
5.7: The malaria and mortality burden during the epidemic in Wajir

In February 1998 one of the authors of this report (RWS) went to Wajir district hospital to identify all the admission registers from varied locations around the hospital to build up a picture of the patterns of complicated malaria leading up to the epidemic. Ward admission registers were located for a complete period 1991 through to February 1998 with the exception of no information recorded during the course of the 6 week nurses strike in December 1997-January 1998. Tallies were made of all malaria and malaria-related admission diagnoses by age by month. Wajir District Hospital was re-visited in April 1999 to complete the admission data through to this period. These data are shown in Figure 5.7.

The dramatic rise in hospital admissions with a diagnosis of malaria followed approximately 3 months after the onset of the heavy rains. The clinical patterns among 89 patients seen by MSF doctors in March 1998 at a "special unit" within the hospital included 61% with severe anaemia, 28% with altered conscious levels and 6% in coma (note previous estimates were as high as 17% due to cerebral malaria, Allan et al., 1998). The case-fatality at this better equipped unit was 8.2% (Brown et al., 1998). As described in the 1940's by Heisch (1947) the clinical presentation included children and adults indeed the overall ratio during the epidemic was 2.1 adults per childhood admission implying that either adults were at greater risk of the severe complications of infection or had preferential use of the hospital facilities.

Between October and June 1998 the mobile clinics surrounding Wajir Town, operated by MERLIN attended to 68,021 consultations of which 82% were clinically treated as malaria (MERLIN, 1998). Similar proportions were described for hospitalisations during this period (unpublished data). Comparing a presumptive diagnosis (recent history of fever, in the absence of other causes) with thin film microscopy and Parasight F® among 148 patients seen at mobile clinics run by MSF showed that 49% had detectable antigen with Parasight F and 31% were positive at microscopy for *P.falciparum* (MSF, 1998). Combining these estimates one can estimate that during this period of the epidemic approximately 40% of illness episodes that sought treatment at clinics were probably a direct result of malaria infection.

Figure 5.7: Monthly malaria admissions to Wajir District Hospital (all ages) between 1991 and 1999.



MSF (1998) reported that between January and March 1998 the mobile clinics in the town of Wajir treated over 20,000 cases of malaria among an estimated population of 60,000. The population in the town and surrounding Bullahs had increased dramatically as people moved to safer territory following the flooding in the surrounding countryside. This represents an absolute minimum attack rate of 33 per 1000 population for these three months. Consultation data from MERLIN (1998) suggest equal attack rates between children less than 5 and the population aged greater than five years.

The controversial figures that stimulated press interest in the malaria epidemic in Wajir derived from a series of surveys conducted by OXFAM, MERLIN and MSF. These data were derived from a series of household investigations enquiring from elders of mortality within their villages over proceeding days or since Ramadan (Table 5.1). These five surveys represent an average overall mortality of 7.2 per 10,000 population per day. The reported annual crude death rate for Wajir District is 11.7 per 1000 population (CBS, 1996). Therefore mortality during the epidemic was probably 25 times greater than would have been expected in this area.

Table 5.1: Average daily mortality among selected communities during the epidemic in Wajir.

Dates	**Mortality per 10,000 total population per day**	**Location**	**Source**
19-25 Jan 98	8.0	Bulla Shahefey	Anne Waelbroek, Pers. Comm.
6-10 Feb 98	10.0	Wajir	Allan et al., 1998
14-27 Feb 98	4.7	Wajir	Anne Waelbroek , Pers. Comm.
31 Jan- 8 Mar 98	9.4	Wajir	MSF, 1998
26 –31 Jan 98	7.5	Mandera	Anne Waelbroek , Pers Comm.
Average	**7.92**		

Using the above data one can structure a series of conservative assumptions:

- The malaria epidemic lasted for four months (120 days)
- The epidemic affected the entire district (110,954 people)
- Risks of malaria mortality were equal for children and adults
- Overall mortality was on average 7.92 per 10,000 people per day
- Malaria only accounted for 40% of fatal events (according to proportion of true malaria cases among clinic attendance's)

Under these assumptions there may have been at least 10,545 deaths from all causes during this period or 9.5% of the population including 4,218 deaths due to malaria. Such calculation's are, by definition crude, but such an informed approach provides us with a minimum estimate of the devastating problem faced by Wajir during the first four months of 1998. The co-incidental risks of under-nutrition and malaria infection cannot be underestimated and overall the Daily Nation reports in February were probably very close to the truth.

5.8: Controlling the malaria epidemic in Wajir district.

Various NGO's provided immediate support and assistance when the epidemic reached public attention. These included OXFAM, MERLIN, Medecins Sans Frontieres, Belgium (MSF-B) and the Malarone Donation Programme (MDP), a Swedish Rotarian doctor at the hospital and the Catholic Mission.

MERLIN are a UK-based emergency and relief organisation and were asked by OXFAM (who were already in Wajir as part of a community-based health care (CBHC) programme training Community-Health Workers) to assist during the malaria emergency. MERLIN were provided with funds (UK£565,500) by the UK Government's DFiD emergency assistance programme. MERLINs activities included the use of four vehicles to provide out-reach clinical services outside of Wajir town and employing MoH nursing staff from the district hospital who worked with three expatriate clinical staff. The teams arrived on the 15th February and the mobile clinics began in March 1998. By October they had treated over 68,000 patients. These out reach clinics were additionally used to provide food supplied by the World Food Programme through OXFAM, provide measles vaccination and Vitamin A supplementation. In addition the emergency programme included the procurement of 20,000 insecticide-treated bed nets (ITBN) and their subsequent delivery to the rural communities. Nets were obtained from Siamdutch Ltd and impregnated with deltamethrin (AgroEvo ltd.). The programme estimated a ITBN coverage of approximately 80,000 people in the district. Several reservations were raised about ITBN efficacy in an area where the vectors were predominantly *An. arabiensis*, the potential for sale when food security was poor and household construction was temporary. However, surveys conducted by MERLIN in July 1998 showed a high retention rate of issued nets (98%) (MERLIN, 1998a). No surveys were conducted on whether the ITBN reduced morbidity. The team withdrew from Wajir in October 1998 (Allan et al., 1998; MERLIN, 1998a; Carrington, 1998; MERLIN, 1998a).

OXFAM continued their water and food security operations in the area throughout the epidemic and provided an important conduit between the NGO fraternity and the local community. Indeed the success of the mobile clinics operated by MERLIN was attributed to the well established trust in OXFAM by the wider community (Carrington, 1998). OXFAM, whilst not directly involved in providing a malaria service, did provide chlorination services

to wells etc. which probably removed the risks of cholera and diarrhoeal diseases during the malaria epidemic (Carrington, 1998).

Since 1992 (MSF-B) has supported a CBHC project at the Dadab Refugee Camp in Garissa district and provided ITBN to hospitals at the camp. MSF were operating in NEP prior to the malaria epidemic by providing emergency nutritional support in Mandera district. In January MSF-B sent clinical staff to support MoH staff at Wajir District Hospital. They developed a makeshift fourth ward at the hospital for malaria patients and employed the MoH nursing and clinical officer staff to man the ward and assist in 6 out-reach clinics in the Wajir town. To complement the out-reach clinics, 50 home visitors were employed to maintain a referral system for severe patients to the MSF ward using one of 21 donkey carts. MSF-B considered various options for vector control in and around Wajir including larviciding with Temephos, Ariel spraying (discounted because of cost: 68,000 US$ per single round) and residual house-spraying with deltamethrin. The latter was the selected option in April, after the epidemic had abated. 6,000 out of a targeted 10,000 homes were sprayed either indoors or if temporary shelters in-doors and at the bases of the "walls" (MSF, 1998).

Glaxo-Wellcome (Kenya) provided laboratory and basic medical supplies through their collaboration with the Malarone Donation Programme (part of the Carter Centre's Task Force for Child Survival & Development). The MDP had spent 12 months trying to establish a donation programme around atovaquone/proguanil (Malarone) in Kenya. Initial thoughts turned to the use of Malarone during epidemic control in accordance with its nominal *in vitro* gametocidal properties. The MDP used the epidemic as a means to get the drug registered (26^{th} February 1998) in Kenya and conducted *in vivo* comparative studies of the drug during the crisis in Wajir. These studies showed that Malarone was not efficient at clearing gametocytes but provided parasitological cure by day 3 all patients and performed as well as sulphadoxine/pyrimethamine. However, chloroquine failures were of the order of 10% by day 7. Despite the experimental nature of the MDP's activities, the Glaxo/Wellcome provision of basic supplies to mount out-reach services contributed to the clinical cover provided in the area.

The Catholic Mission, located in the town provided additional clinical services, and the Swedish Rotarians who provided laboratory and clinical support with two volunteers who worked at the hospital.

The Medical Officer in Charge, Dr Mohammed, was the first person to raise the public awareness of the plight of Wajir and continued to play a pivotal role in brokering the partnerships with donors, supplies and the community. His role was an unenviable one given the desperate nature of the staffing levels (always over 30% depleted since the strike) and the overwhelming patient burden. Nevertheless, three obvious features of the NGO activity in Wajir during the epidemic emerged:

1) a lack of local weekly planning meetings between the various partners and the District health Management team, perhaps related to a basic mistrust and competition for limited resources between the various partners
2) the payment for services provided by the already depleted and poorly paid MoH staff.
3) The absence of any MoH or WHO guidelines on appropriate preventative strategies or therapeutic protocols to assist a targeted and co-ordinated response.

It was interesting to note that despite the high rates of severe anaemia, MSF-B opted to withhold iron supplementation on the basis that this would exacerbate severe malaria progression. Furthermore, Brown et al. (1998) describe using pyrimethamine as treatment for uncomplicated malaria (probably a typographic error in the Lancet article or ignorance of appropriate management strategies). At the Dadab refugee camp in Garissa, CARE international continued to use chloroquine. A WHO consultant on vector control arrived in March and advised the MoH not to entertain residual house-spraying in Wajir four weeks before such a campaign was mounted by MSF-B in 6,000 households.

Despite the rapid increase in extended clinical services to the isolated communities of Wajir, residents of the town still relied significantly on the use of over-the-counter medications. Of 1233 patients seen at mobile clinics run by MSF-B between 6-9th April 15% had sought medication before being seen of whom 45% had obtained treatment at the market (Brown et al., 1998). Indeed it was argued by some that drugs and supplies air-lifted into Wajir for the NGO's and MoH found their way into the open market and supported a rapid proliferation in informal clinical services in the town. These informal services included the unusual practice of Fansidar injection. During the crisis there were 6-7 private clinics operated by MoH clinical officers and nurses in Wajir town (Carrington, 1998).

The Ministry of Health were able to re-deploy nursing and clinical staff in the region by mid-February, but as with other areas of the country, Wajir faced a serious shortage of drugs and essential medical supplies. Various organisations including MSF-France, CARE International, The Red Cross societies (who provided 12 tonnes of drugs and medical supplies) and DfiD provided funds for emergency drug supplies. The DfiD funds were able to procure essential malaria drug kits (Chapter 3) and these were provided to NEP through the Medical Supplies Co-ordinating Unit, MoH from Nairobi.

Co-ordination of the epidemic response was conspicuous by its absence during the initial phases immediately following the awareness of the problem by the Daily Nation. Many of the NGO's who moved quickly into the area did so without any central co-ordination. This was recognised as a weakness in the system and an Epidemic Task Force was established (Chapter 3). Despite irregular meetings between March and April 1998 this committee was unable to achieve any consensus view on how best to further prevent the epidemic but rather acted as a political advocacy body which ensured donor commitment to provide supplies for better curative services. There was no single reference document for either MoH or NGO staff to consult on standard protocols to be used. In Wajir the MoH was faced with a politically unstable area of people displaced by flooding, a food security crisis, no infrastructure, emerging from a nurse's strike and a limited clinical service completely unprepared for malaria.

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6. Conclusions: Combining the evidence

6.1 Defining epidemic malaria in Kenya

To understand the stability of infectious diseases direct measurements of parameters related to the basic reproduction rate of infection (BRR) are required. Deriving the necessary parameters for the BRR is labour intensive and subject to wide margins of error when measured in areas of extremely low vector abundance. Consequently several authors have defined *P.falciparum* transmission potential between secondary hosts and man using proximate determinants of parasite and vector development, such as ambient temperature and rainfall. Climate-driven models of transmission distribution in Africa have recently become easier to develop following technological advances in Geographic Information Systems and the availability of high-resolution climate databases for Africa (Lindsay & Martens, 1998; Lindsay et al., 1998; Thomson et al., 1996; Craig et al., 1999). Chapter 2 describes the application of the model developed by Craig et al. (1999) to describe the complex and diverse transmission conditions in Kenya. This model provides a crude approximation to the limits of stable transmission in Kenya, particularly among districts located in arid areas in North Eastern Kenya. However, the model fails to provide a reliable description of five of 13 districts in Western Kenya historically defined as being subject to epidemic malaria conditions. The complex nature of epidemic genesis among populations located at the fringes of stable transmission may require better models which capture the extent of inter- and supra-annual variations in climate; human and land-use factors; or the inclusion of higher resolution proximate determinants for permanent and temporary vector breeding sites.

Despite quantitative approaches to the definition of epidemic prone districts in Kenya historical and contemporary definitions have been driven as much by public health experience and political significance as by climate and vectorial capacity models. In many ways it seems more appropriate to define epidemic prone districts in accordance with the clinical and public health experiences of those charged with its control and management. Such people will determine resource allocation, investment in prevention and remain accountable to the communities they serve. However, reliance upon self-determined definitions lends itself to abuse and political appropriation. During the 1930's and 1940's epidemics were clearly articulated in areas of economic and political significance (Nairobi and the "White" Highlands). Conversely there was a blatant effort to play down the public health significance of the malaria epidemic that swept NE Province during the El Niño flooding of 1997/98. Consequently a balance is required to allow experience to guide the identification of at-risk areas whilst maintaining some credible, empirical basis by which the stability of transmission is defined at a national level. Climate-driven models may provide one such approach to defining the limits of stable transmission but require considerable refinement among these fringe areas. Developments in this area are focussed on the biomathematical associations between parasites and vectors of *P.falciparum* transmission (MARA/ARMA collaboration, 1999) rather than the public health consequences of infection.

In this review we have identified data on admissions to hospital with complicated malaria infections from a broad spectrum of conditions regarded as prone to epidemics. It has been possible to identify secondary data to demonstrate the continued presence *P.falciparum* parasites and infected vectors among the communities served by these clinical services. Parasite rates in asymptomatic hosts varies considerably within a restricted geographical area ranging from 4% through to greater than 70% during identical periods of observation. At each site, with the possible exception of Wajir, locally acquired infections requiring in-patient management occur every year within relatively short seasonal windows of transmission. Analysis of these data in Kenya suggests that epidemics (exceptional increases in case-burdens) were evident during the following months and years at the following sites:

Nyamira:	June 1994; June-Aug 1996; June-Aug 1997; Jan-Mar 1998.
Uasin Gishu:	July 1991; Jan-Mar 1995; Jul-Aug 1997
Nandi (Kapsabet):	Jul-Aug 1991; Jul-Aug 1994; Jan-Mar 1995; Jul-Aug 1997; Jan-Mar 1998
Kericho:	Jul-Aug 1991; Jul-Aug 1994; Jul-Aug 1997; Jan-Mar 1998
Samburu:	Jan-Mar 1993; Jan-Mar 1995; Jan-Mar 1997; Jan-Mar 1998.
Wajir:	Jan-Mar 1998

These "epidemics" have a tendency to overwhelm existing health services and available resources. The secular pattern is not consistent between sites located within the close geographical proximity of the highlands, however there appears to be "greater-than-average" increases in disease risks every 2-3 years at most sites. Such periodicity approximates to the frequency of epidemics described in similar communities during the 1930's and 1940's (Chapter 3). Nevertheless such frequency raises the appropriateness of the term "epidemic". In many ways it seems more appropriate to now define these highland communities as areas of low malaria risk but risks are subject to large-scale intra-annual variation. Among populations located in the arid areas of NE Province malaria "epidemics" adhere closer to the popular view of "epidemics". It is probable that the devastating effects of malaria transmission in 1997/98 in Wajir occur rarely. A consistent feature of all "epidemic" prone areas is the even distribution of cases with age. Populations exposed to a low risk of parasite challenge tend to develop functional immune responses slowly (Snow et al., 1997: Snow & Marsh, 1998). As transmission intensity declines and tends toward instability adults and children will remain at equal risk of severe complications of infection. This concept of immune acquisition according to parasite challenge is by no means new and well described in the old literature. Of interest is the direct use of these epidemiological patterns to define the unstable nature of transmission among "epidemic" prone communities. Defining epidemic potential on the basis of the ratio of adult to childhood disease risks may represent a useful public health tool. However, such definitions encompass wide areas of hypoendemic stable malaria conditions. Consequently it is probably worthwhile including the extent of inter-annual variation as an adjunct parameter. For example, a setting which exhibits a seasonal peak greater than two or three standard deviations over a 10 year mean and where adults and children are equally susceptible to the severe consequences of infection. These mathematical derivations of epidemic risk would require long-term data sets and an ability to remove long-term trends (see effects of drug resistance described below).

MacDonald (1957) describes three types of epidemics: long-term, periodic and irregular. Long-term epidemics were thought to be difficult to distinguish given their protracted effects but MacDonald (1957) provides the example of Italy, which was described as periods of prolonged waves of incidence separated by periods of "*salubrity*". MacDonald (1957) continues that

> *"The causes of these waves cannot be certainly determined and must largely remain the subject of inspired guesswork. It is certain that they were on the whole small, representing minor increases and decreases in the facility of transmission, which explains the difficulty in discerning them accurately."*

These long-term epidemics may apply to conditions in the highlands of Kenya. Conversely, the description provided by MacDonald (1957) of irregular epidemics due to the unusual introduction of parasite or vector into an area best describes the conditions prevalent in Wajir district during 1997/98. Despite a review of these classical descriptions of malaria epidemics, essentially derived from 17th Century Europe, South East Asia and the Americas, it remains the

case that much more work is required to provide better descriptions of the epidemiological features of transmission and disease among "epidemic prone" communities in Africa.

In summary, defining epidemic prone areas is pragmatically and conceptually difficult. Expert-opinion approaches may constitute the best possible means of defining epidemic risk districts for targeted control and resource allocation. However, these experience-based approaches should be developed in concert with a better public health definition of epidemic susceptibility. Combining the age patterns and secular variations in clinical disease may provide a useful rubric to identify at-risk districts.

6.2 Is there an emerging malaria epidemic problem in Kenya?

Since the late 1980's and through the 1990's much has been made about epidemic or highland malaria. Several authors have linked these phenomena to global warming and climate change favouring transmission of *P.falciparum* in areas previously free from malaria (Bouma et al., 1997; Loevinsohn, 1994; McMichael, 1997). Such epi-phenomena garnered popular interest among the wider scientific fraternity. During the 1980's the world was facing imminent doom due to population explosion and insufficient food to feed the global community. The scientific Nostradamus's of the 1990's cite ozone depletion, CO_2 emissions and global warming as the cause for pending global collapse. There follows a tendency to link all phenomena to popular scientific themes and this may also be true of malaria epidemics in Africa. What is clear from this review is that "epidemic" malaria was a common feature of many "newly emerging epidemic" areas located in Kenya's highlands between 1918 and the late 1940's. What appears to be true is that following concerted attempts to interrupt transmission during the 1950's and 1960's in Nandi and Kericho districts (Roberts, 1964a-c; Strangeways-Dixon, 1950), malaria risks declined significantly. These low public health risks of malaria were maintained throughout the 1970's despite any direct evidence of vector control or widespread chemoprophylaxis but widespread use of chloroquine for therapy and limited chloroquine use for prophylaxis. During the early 1990's at Nandi and Kericho there were several repeated exceptional increases in the clinical burdens of malaria in both districts, most notably in 1991, 1994, 1995 and 1997. It is always difficult to define populations-at-risk for analysis of case numbers presenting to hospital. The Brookebond tea estate hospital data from Kericho however provide a unique opportunity to control for changes in at-risk populations given that the catchment population is fixed, utilisation patterns have remained constant and data is available on the numbers of employees allowed access to the services. These data demonstrate the precipitous rise in malaria admissions in 1990. The Bookebond Tea Company only opened the hospital in 1955 but analysis of historical data for Kapsabet hospital in Nandi district suggests that disease burdens described in the 1990's were similar to those between 1942 and 1951. Consequently the historical appraisal of the public health significance of malaria among highland populations is better described as a re-emerging problem rather than a new, unprecedented, phenomena (Shanks et al., 1998; submitted). Previous attempts to link climate change to changing malaria patterns may not have examined a long-enough clinical time-series.

Clearly the genesis of epidemics in a population is multi-factorial. Conditions among the arid areas of North Eastern Kenya lend themselves to devastating malaria epidemics resulting from unusual climate patterns, particularly rainfall. Intermediary states of arid-versus-altitude limiting factors in Samburu also demonstrate the significance of exceptional rainfall patterns on disease incidence. The El Niño rains affected virtually every endemic and epidemic setting in Kenya including the highlands. These exceptional rains occurred during the traditionally short-rains in most highland areas, a period when ambient temperatures are highest. Combined these conditions led to a protracted period of suitability for transmission. However, the effects of long-

term global warming are less evident among these populations. Recent analysis of temperature data since 1965 at Kericho has been unable to identify supra-annual cycles of temperature which would explain the supra-annual cycles of clinical disease seen during the 1990's (Shanks et al, submitted; Hay et al., submitted). Rather these analyses suggest that other explanations must be invoked to explain the timing and frequency of epidemic waves and cycles in this area. De Mello (1947) and Campbell (1929) both suggested that inter-epidemic periods were characterised by periods of poor nutrition due to drought and linked to a waning immune response among the host population. Hay et al. (submitted) suggest that the genesis of epidemic waves may be linked to the build up of susceptible hosts within a fixed population and that these inter-epidemic periods may be decreasing in duration for reasons other than climate (Shanks et al., submitted).

6.3 Factors other than climate predisposing new disease patterns in the highlands.

6.3.1 Drug resistance

A striking coincidence exists between the onset of "new" epidemic conditions described for highland populations and the emergence of chloroquine resistance in Kenya. Throughout the 1960's until the late 1980's chloroquine was an effective, cheap and widely available source of treatment for uncomplicated malaria in Kenya. Fevers among rural communities, were for the most part, self-treated with over-the-counter purchased proprietary forms of chloroquine. Efforts were made during the 1980's to improve access to curative services by expanding primary health care services and Government dispensaries and clinics. Chloroquine was perhaps the most widely prescribed and consumed drug in the country. It probably continues to be so. It is difficult to gauge the extent to which rapid access to effective drugs was prevalent during the 1930's and 1940's. Evidently quinine was widely used to manage fevers and recommended by the colonial medical authorities for employers of Kenya's labour force (Patterson, 1935).

Quinine was not recommended for the management of uncomplicated malaria following the introduction of chloroquine in Kenya in 1950. The first case of chloroquine (CQ) resistant malaria was reported in a tourist visiting Kenya in 1979. The first case of indigenous CQ resistant malaria was reported in an infant in Kisumu in 1983. Between 1983 and 1991, 21 studies of CQ efficacy were conducted in Kenya. The results and methods used varied. The most consistent parameter was the failure to clear infections 7 days after administration of CQ. The results during this period suggested that between 1.9% and 96% of patients were unable to clear parasitaemia by day 7; 11 of the 21 studies showed parasitological failure > 25% of all patients. The results shown in Table 6.1 are summaries of the frequency of chloroquine failure among populations located in epidemic prone districts. In 1981, 15 isolates were tested *in vitro* for chloroquine sensitivity and it was accepted that inhibition results indicated that parasites from Nandi district were still sensitive to this drug (Masaba et al., 1985). The Table presents *in vivo* parasitological failures by day 7. Whilst there has been a significant move toward measuring clinical responses (WHO, 1994), non-immune patients who harbour treatment-failure infections must continue to be at-risk of severe complications. Between 1988 and 1998 between 1 in 10 and 1 in 2 patients treated with CQ in unstable areas of transmission in Kenya were unable to clear their initial infections.

Assuming all patients cleared infection before 1983 the present therapeutic conditions lend support to the rising consequences of prolonged infection to increasing disease rates in this population. The onset of increasing disease risks at most sites presented in this report is consistent with a rapidly emerging problem of first-line therapeutic failures. These areas, and those of stable hypoendemic malaria (e.g. Kirinyaga), may be more susceptible to the effects of drug failure than stable endemic areas where acquired immunity modulates the clinical effects of infection. Furthermore, case-fatalities of patients admitted to hospital with malaria during the

1990's have doubled compared to case-fatalities of the 1950's – 1970's (see Nandi, Kericho and Eldoret). Perhaps of concern during 1992 at Eldoret district hospital was the reports from clinical investigators from Moi University who recommended the continued use of chloroquine for the in-patient management of complicated malaria to reduce any unnecessary use of quinine (Nabiswa et al., 1994). Difficulties in providing easy access to SP preparations combined with a poor-evidence based clinical practice probably continues to provide the basis for extended chloroquine use in many highland populations.

Changes in the case-fatalities of severe disease over-time cannot be linked to climate change. The contribution to infectious reservoirs through unsuccessful treatments of primary infections may provide a basis to understand the re-emergence of supra-annual cycles of epidemics linked to the short windows of climate suitability for vectors. Among non-immune populations most primary infections will result in clinical disease. Whilst chloroquine was effective these primary infections would have been effectively cleared. As chloroquine began to fail to clear a significant proportion of clinical infections, the duration and size of the infectious reservoir may have increased. The period required to develop sufficient infectious hosts to pose a threat for an epidemic genesis is not clear but will also be a function of immunity acquired during the population exposed to a previous epidemic wave. Bio-mathematical models of transmission including infectiousness and immunity (Anderson et al., 1984) may be able to unravel some of these components and provide a testable framework for intervention.

6.3.2. Migration and population mobility

From the earliest description of epidemics in Kenya, population movement featured as a major determinant of emerging infectious reservoirs for new disease conditions. Matson (1957), Strangeways-Dixon (1950) and Farnworth-Anderson (1929) all describe the European migration for economic development to areas of the Western Highlands between 1909 and 1918. Prior to these migrations these areas were probably free from malaria. The Western highlands are located in close proximity to the Lake Victoria lake basin, an area intensely endemic for *P.falciparum*, with an estimated entomological inoculation rate of 237 infectious bites per person per annum (Beier et al., 1990). Consequently, people born under such conditions develop immunity quickly and enter adulthood with functional immune responses against the morbid and fatal consequences of infection. During the development of the railway and road networks people from "Kavirondo" (presently Kisumu district) were recruited as part of the labour force. Furthermore, Strangeways-Dixon (1950) describes over 30% of the labour force employed on the tea estates of Kericho as coming from the Lakeside (Luo), similar number of Luo's are employed on the estates today (Malakooti et al., 1998). "Immune" adults will acquire new infections, remain clinically well and consequently not access curative services, thus allowing patent infections to generate gametocytes and provide a constant contribution to an infectious reservoir. Mobility between endemic and unstable areas is high and associated with "home" leave and monthly visits to provide cash incomes to families. Chataway (1929) regarded this mobility as a major contribution to the genesis of the 1928 epidemic in Kericho and Roberts (1964) regarded mobility of people between Nandi and Kisumu as a major limitation to any attempts at eradication in Nandi district.

6.3.3. Changing land-use patterns

Linked to the large-scale settlement of Europeans in the highlands of Western Kenya was a major shift in the use of arable land for wheat in Uasin Gishu, tea in Nandi and forestry in Nandi and Kericho. This economic development within the agricultural sector led Gilks as early as 1928 to express concerns over the subsequent effects on changing malaria risks (see section 3.2). The expansion of towns and organised farms during the 1920's and 1930's led to the formation

of dams, irrigation schemes and drainage systems. This changing ecology may have favoured the development of permanent breeding sites. This was recognised early by the Medical Department of the Colonial Administration who organised extensive environmental management schemes aimed at reducing vector breeding.

A common anecdote provided in the historical and contemporary descriptions of epidemics in Kenya is their incredibly focal nature within a district. This phenomena suggests that local geographical and ecological factors may be significant. Khaemba et al. (1994) suggested that local man-made dams were far more significant breeding sites than swamps in Uasin Gishu during the epidemics of the 1990's.

We were unable to provide any quantitative or impressionistic evidence in support for or against major changes in land-use patterns among the highland populations. Nevertheless intensive agriculture forms the economic backbone of many of these districts and outside of commercial farming the small-scale farming practices of a rapidly expanding population may continue to pose increasingly favourable conditions to existing vector systems in the highlands. The agricultural sector in Kenya have high resolution GIS maps of land and soil use across the country and these need to be combined with higher resolution (1km) satellite images of the area to better understand the micro-epidemiology of the highland region. Studies are currently underway as part of a collaborative effort between KEMRI and University of Nimemegn, Holland, to examine the spatial ecology of malaria in relation to land use among the Western Highlands (Githeko & Takken, personal communication).

6.4. Control and prevention of epidemics.

6.4.1 Prediction

In 1937 and 1940 the entomologists attached the Medical Department provided warnings of pending epidemics based upon secular variations in periods of drought and heavy rain. No formal descriptions were provided of how these "warning" systems operated but they were important in soliciting finances and mobilising resources among the population (essentially limited to the European community). More recently forecasting, prediction, early warning and early detection systems have been recognised by Ministries of Health in the East Africa sub-region as fundamentals to their needs for epidemic management (Highland Malaria Meeting, Durban, March 1999). One of the limitations of climate forecasting in Kenya is that all meteorological data must be purchased from the Met. Office at great expense. However, there remain two fundamental issues pertinent to prediction or early warning:

1) What are the relative contributions of measurable factors, such as climate, upon the genesis of epidemic waves
2) How will early predictions and warnings influence how control is managed within a district.

There are various research efforts currently examining the temporal associations between climate variables and clinical malaria (MALSAT, Liverpool School of Tropical Medicine; MEWS, University of Oxford; etc). As described above these approaches need to recognise the complex nature of epidemics in areas such as the Western Highlands and how supra-climate variables can be captured within models of epidemic forecasting and early warning. Assuming breakdowns in effective health services and efficacious drugs are important determinants of epidemics of increased mortality and disability warning systems need to include the operational monitoring of drug supply and drug sensitivity.

Secondly, predictive models of changing disease frequency are only of value if these can be linked to a strategy for district-based control. The conditions prevalent in the highland communities presented in this report suggest that malaria is a clinical problem every year yet some years are considerably worse than others. Some may therefore argue that strategies should not be developed which focus only on a response to alarms from early warning systems. Rather strategies for control should be developed which aim to reduce the overall incidence of disease and that these approaches to control should be invoked every year.

There is a need to promote research aimed at understanding the temporal genesis of epidemic waves of clinical malaria in the highland's of Kenya. However, these models must be developed within the context of how responses will be mounted to this new information.

6.4.2 Organisation of malaria epidemic control.
Malaria has historically been regarded as an inter-sectorial responsibility in Kenya. The primary responsibility for disease control and prevention in Nairobi during the 1930's was left to the Municipal Council. Organised efforts to distribute treatments during the early epidemics in the highlands during 1928 required the mobilisation of local administrative officers and chiefs (Chataway, 1929). Roberts (1964) attributes the overall success of mass drug administration to its management by the town council and local employers. Therefore there is undisputed evidence that disease prevention or management during epidemics demands a collective ownership and responsibility beyond simply the health sector.

The response to the most recent epidemics of the 1990's has ironically demonstrated a far less district-based inter-sectorial approach than was evident during the first half of this century. Rather responses have been managed and co-ordinated centrally and operated almost entirely from within the Health Sector. New guidelines for the prevention of epidemics in Kenya (MoH, 1999) do not elaborate on how preventative strategies aimed at vector control can be developed within the wider sector of employers, civil administration, departments of education etc. Decentralisation, health-sector reform and inter-sectorial collaboration are widely used to describe new approaches to health in the developing world during the 1990's. McLennan in 1949 made financing, staffing and control of malaria a local authority responsibility, whilst technical advise and entomological surveys and general supervision were central Government roles (Colony & Protectorate of Kenya, 1949). Decentralisation is not a new concept but how epidemics can be better managed and prevented within this framework needs further investigation.

6.4.3 Effective case management
MacDonald (1957) claims the following

> "*There are only two radical means of dealing with epidemics; one is to recognise them in the very early stages, before any notable part of the population is affected and to prevent their further spread; the other is to stop them with drugs. Any measure of mosquito control must be secondary to drug treatment or prophylaxis once an epidemic is established if needless suffering is to be avoided. Even immediate imagicidal control leaves many incubating the disease, and larvicidal control cannot be expected to influence the epidemic for at least a month and perhaps longer. The most valuable drugs are those which can cure in the smallest possible number of doses, and which combine curative and preventative properties. For most circumstances the 4-aminoquinolines are the most appropriate* [Chloroquine & Amodiaquine]".

Early and effective management of clinical disease cannot be underestimated. Chataway (1929) argues that the epidemic of 1928 in Nandi district was abated following widespread therapeutic use of quinine. The recent rises in hospitalisation, case-fatalities and reduced intervals between epidemics may all be associated with failures to chloroquine. Currently there are several barriers to providing the presently recommended first line antimalarial at all available distribution points in a district, formal and informal. Assuming these can be overcome, it remains to be seen whether sulphadoxine-pyrimethamine (SP) will operate in the same way as cholorquine may have done by reducing infectious reservoirs within a community and therefore operating as both curative and preventative. Recent studies along the Thai-Burmese border have demonstrated that the combination of artesunate with mefloquine has not only extended the useful life-expectancy of the latter drug but has also significantly reduced local transmission (Price et al., 1996). It seems unlikely that mefloquine will ever be recommended as a first-line drug in Kenya, however there is a growing concern over the need to protect SP with artesunate. The transmission conditions on the Thai-Burmese border are not dissimilar to those of the Kenyan highlands. These areas may be some of the most appropriate sites to introduce combination therapy early in the development stages of this strategy. The effects of artesunate upon the sexual stages of the parasite combined with rapid cure offer great opportunities. Trials of combination therapy are currently being undertaken in The Gambia (T. Doherty, personal communication), an area of acutely seasonal low intensity transmission (as close to the highland areas as stable transmission can be) and the results of these trials should be followed closely.

6.4.4 Prophylaxis or MDA.

The results obtained from the experimental trials of Mass Drug Administration (MDA) with pyrimethamine (Roberts, 1964b) and weekly prophylaxis with proguanil (Strangeways-Dixon, 1950) at Nandi and Kericho districts respectively cannot be understated. Both approaches were thought to be "affordable" and resulted in impressive reductions in incident infections and clinical disease. Pyrimethamine alone is no longer an effective antimalarial and proguanil at daily doses would be beyond the scope of most conceivable district-level delivery systems. However, new primaquine analogues have been developed, tefanquine (WR238605), which effectively act upon asexual and sexual parasite forms and due to an exceptionally long-half life provides protection for up to 6 weeks. Limited MDA using SP and primaquine is current recommended practice in South Africa for household members of identified cases. Nevertheless, there is a widely held reluctance to advocate either prophylaxis or MDA in the rest of Africa. Reasons include problems of sustainable delivery, drug pressure for emerging resistance and impacts upon acquisition of immunity. The latter only applies to populations who live under stable endemic conditions. The special conditions prevalent among communities with no functional immunity demand a new conceptual approach. It was striking how control agencies would not consider MDA or prophylaxis during the Wajir crisis. In some ways the conditions that prevailed in Wajir were analogous to not recommending that European tourists take prophylaxis when visiting Kisumu. Unthinkable but demonstrates an apparent inconsistency in our understanding of risk.

6.4.5 Vector control

As MacDonald (1957) argues that most attempts at emergency responses to epidemics using vector control are like locking the gate after the horse has bolted. This was almost certainly the case of for the use of house-spraying by MSF-B during the Wajir epidemic. Consequently residual house-spraying, larviciding or the use of Ariel spraying would only be of value before the identification of significant rises in clinical case burdens. This goes back to structuring the purpose of epidemic forecasting versus early warning/detection. Experimental

preventative strategies through dieldrin residual house-spraying were effective during the 1950's and 1960's in Nandi (Roberts, 1964) and more recently was shown to be an effective preventative strategy in Nandi using ICON 10 WP (Zimba, 1998). Neither experiment was designed to use residual spraying during an epidemic rather they were targeted at times of known seasonal onsets of vector proliferation. The costs of these approaches have not been fully assessed, however, Roberts claims that to maintain the programme would have required at least of doubling of overall annual district health expenditure.

Insecticide-treated bed nets (ITBN) are currently being promoted across Kenya as a means by which households can take some responsibility for their own prevention. Populations residing in epidemic prone districts will receive similar messages and advise. This intervention is more passive than active and requires the establishment of sustainable net re-treatment services established by entrepreneurs, community pharmacists or shops. To-date no experimental trials of ITBN have been undertaken among epidemic prone communities. However it is "common sense" that the reduction in any infection risk must be a good thing for non-immune populations. The relative success of ITBN will, however, depend critically upon the expected demand for services and the meeting of this demand through innovative service providers. It seems unlikely that these services will be provided free of charge to households in the highland areas. However, during the Wajir epidemic MERLIN did provide free ITBN to a desperately poor and displaced population experiencing one of the worst epidemics on record. There was no evidence to support or refute whether this intervention saved any lives and nor is there any mention of whether these communities will receive, or need, net re-treatment services. Donors and NGO's are strongly supportive of ITBN as an intervention principal. Nets can be procured on mass, distributed and quantified. It seems most likely that this would provide important protection for long-term refugees or displaced people who find themselves within a new risk environment. However, clearer guidelines are required for situations such as those prevalent for the first time in 30 years for 5 months in Wajir and populations located in the highlands.

6.5 Recommendations for future research.

This review has identified several weaknesses in our understanding of the epidemiology and control of malaria. These are described below:

1. *Testing climatic, ecological, vector and parasite determinants of unstable malaria along the fringes of stable transmission in Kenya.* The definitions and determinants of epidemic malaria in Kenya require better descriptions. Clinically derived definitions of stability which accommodate the public health significance of acquired immunity and seasonal changes in risk should be explored further. This work is currently being undertaken as part of the MARA/ARMA collaboration and links with Dr Simon Hay, University of Oxford.

2. *Estimating the burden of disease and the economics of prevention versus disease management*. The review has identified a series of historical data sets which allow for population-based estimates of morbidity and mortality under conditions of low and high risk malaria epidemics. These data should be combined with an economic appraisal of potential intervention approaches to test the economic and public health significance of sustained prevention versus epidemic response management.

3. *Better descriptions of the pathological consequences of infection among Africa adults.* Recently there have been advances in our understanding of the pathophysiology of complicated *P.falciparum* infections among African children. These advances have guided the development of new therapeutic interventions and management strategies. We however, no very little about the management or pathology of malaria among non-pregnant African adults. This deficiency in our clinical understanding should be redressed so that more appropriate diagnostic and management guidelines can be developed for emergency situations managed by NGO's and routine case-management among highland populations. Furthermore, recommendations for intermittent chemotherapy or prophylaxis of multigravid pregnant women remains poorly defined within these populations.

4. *Improved understanding of micro-epidemiology of epidemics in communities*. Surprisingly few high-resolution epidemiological studies have examined the time-space determinants of clinical disease within epidemic prone areas of Kenya. The use of hand-held navigational systems linking spatial characteristics of vector distribution, infection and clinical cases may provide insights into local epidemic control.

5. *Large-scale community-randomised trials of epidemic prevention using drug or insecticide-based approaches*. Since the 1950's there have been few attempts at large-scale prevention of infection among the highland communities. These early studies were very successful and they need to be revisited with new tools and an improved understanding of costs and sustainable development within newly created health systems.

Table 6.1: Drug resistance studies conducted in epidemic prone areas of Kenya (parasitological failures by day 7).

Authors	Dates	Age ranges	Area	+ve Day 3	+ve Day 7	+ve Day 14
Chlorquine						
Keuter et al. (1990)	March 1988	Pregnant and non-preg women (89)	Kakamega 1219 meters	--	**14%**	23%
Khan et al. (1992)	June 1990	All ages (14)	Kakamega 1219 meters	--	**36%**	50%
Khan et al. (1992)	June 1990	All ages (9)	Uasin Gishu 1829 meters	--	**0%**	11%
Anabwani et al. (1996)	July 1993	All ages (32)	Uasin Gishu 1829 meters	--	**41%**	--
Rapuoda et al (1998)	June 1996	5-59 month (33)	Nandi 1981 meters	36%	**50%**	47%
Malarone Donation Programme (1998)	March 1998	All ages (39)	Wajir 152 meters	7.7%	**10%**	31%
Falaschi & Ansaloni (1997)	July 1995	3month-23 years (65)	Marsabit 610 meters	--	**39%**	46%
Clarke et al. (1996)	June 1994	1-40 years (39)	Turkana 610 meters	--	**8%**	18%
SP						
Keuter et al. (1991)	April 1987	Children (15)	Kakamega	--	**0%**	--
Keuter et al. (1990)	March 1988	Pregnant and non-preg women (90)	Kakamega	--	**7%**	8%
Khan et al. (1992)	June 1990	All ages (12)	Kakamega	--	**0%**	0%
Khan et al. (1992)	June 1990	All ages (8)	Uasin Gishu	--	**0%**	0%
Anabwani et al. (1996)	July 1993	All ages (44)	Uasin Gishu	--	**20%**	--
Rapuoda et al (1998)	June 1996	5-59 month (37)	Nandi	39%	**9%**	0%
Kaneko et al (1998)	Sept. 1997	All ages (70)	Kisii	--	**10%**	
Malarone Donation Programm	March 1998	All ages (47)	Wajir	0%	**0%**	0%
Falaschi & Ansaloni (1997)	July 1995	3month-23 years (54)	Marsabit	--	**0%**	0%
Clarke et al. (1996)	June 1994	1-40 years (29)	Turkana	--	**8%**	0%

6.6: References for Chapter 6.

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