C. The Diseases of the Country

Introduction: Source, Material, and Methods

Certain methodological problems are likely to be encountered if observed disease occurrences based on hospital or other medical statistics are interpreted in a spatial geographical analysis. Epidemiological surveys of good quality — taking environmental factors into proper consideration — are scarce and usually restricted to a few diseases of major interest or importance and consequently generally very specific as opposed to aiming at spatial coverage.

The most prominent problem is the lack of a *common spatial denominator*. For example, in order to examine the relationship between rainfall or temperature and disease, the two physical variables are demarcated by isohyets and isotherms, whereas disease prevalence available in terms of hospital records and medical statistics for notifiable diseases may be based on administrative units.

Hospital statistics or other health statistics suffer greatly from a number of systematic and mathematical biases, which can scarcely be overcome. Some epidemiologists consider them to be useless, but since there is usually no better information available they will have to be used for a long time to come, albeit with great care. Two sources of data are available and can be compared with existing epidemiological surveys and government reports on the disease distribution.

The most commonly used information is derived from *weekly notifications of infectious diseases* on a district basis, and set out in relation to district populations although very little can be said about the distribution within the district and the completeness of this information.

In English-speaking countries such as Kenya, *annual returns* from hospitals give a rather dense but dotted, non-contiguous coverage of a number of diseases according to the *International Classification of Diseases* (ICD).

By expressing the median number of diagnoses per year entered in a hospital record over a period of at least 8 – 10 years as a rate per 10,000 or 100,000 population, a Hospital Recording Rate for the hospital catchment area of, in the case of Kenya, 54 record-keeping Government hospitals becomes quite instructive about the spatial aspect of a disease and is open to careful interpretation.

$$\text{Hospital Recording Rate } \lambda = \frac{\Sigma\, x_i}{n_i}$$

The hospital recording rate is here defined as the frequency (λ) with which a certain disease (x_i) has been observed and recorded in a hospital (i) and entered in the annual return of diseases. It is assumed that the patients having these diseases came from a population (n_i) in general within the catchment area of the hospital. The population 'at risk' in this case and having a chance of being seen, treated, and recorded in the hospital, is the denominator, to which the reported diseases are then related. The observed distribution frequency is best approximated by a Poisson distribution and was previously ranked in a logarithmic scale. Studies by Hinz (personal communication, 1974) showed that a stanine rank transformation, classifying the hospitals according to an ascending order of the Hospital Recording Rate for each disease in 9 ranks differentiated (the hospitals) somewhat better and without an unjustified impression of accuracy:

Stanine Ranking of Hospital Recording Rates

1st = 4% 2nd = 7% 3rd = 12% 4th = 17% 5th = 20% 6th = 17% 7th = 12% 8th = 7% 9th = 4%	of all recording hospitals according to an ascending order of disease recording rate per population in catchment area

The hospital catchment area has been defined for 54 Kenyan Government hospitals according to a method described earlier (Diesfeld, 1969 a), whereby the population within the catchment area was estimated according to the Kenya population census 1962 (see Fig. 4). The median number of cases of each diagnosis was taken as the statistical median value for the effective number of years, for which annual returns were available. In general, the reporting period was the 10 years from 1963 to 1972. The median year was thus 1967, for which the population in the catchment area was estimated as three-quarters of the population growth between the census of 1962 and 1969. This was done by taking into consideration the change of administrative boundaries between the two censuses. In all it was possible to use 455 hospital/years, for the purpose of analysing the disease pattern according to the Hospital Recording Rate.

The advantages and disadvantages of this method have been discussed several times elsewhere (Diesfeld, 1969 a, 1973, 1974 a).

This method at least gives some semi-quantitative indications regarding the spatial distribution of certain diseases as they have been observed in Government hospitals over a timespan of 10 years.

Hospital statistics in Kenya on a larger scale have never previously been analysed, therefore such analysis of hospital statistics may well yield additional information.

Statistics from weekly notifications of infectious diseases and from the annual returns of Government hospitals over the past decade thus serve if not as an ideal then as a valuable basis for reflections concerning the spatial distribution of diseases. These reflections will be considered in the context of the scientific literature and other reports on the distribution of diseases in Kenya.

I. Diseases Transmitted by Arthropods

1. Malaria

(ICD 084) *

"Malaria is a general term applied to a group of diseases caused by infection with specific protozoa of the genus *Plasmodium* and transmitted by the infected female of various species of Anopheles mosquitoes." **

The following forms of malaria are distinguished parasitologically, clinically, and according to therapy, prophylaxis, and prognosis: Malaria tropica, caused by *Plasmodium falciparum,* Malaria tertiana, caused by *Plasmodium vivax,* and Malaria quartana, caused by *Plasmodium malariae.*

Malaria used to be the most significant single cause of mortality in the tropics and sub-tropics, until the WHO inaugurated its malaria eradication campaigns. Although half of the population—predominantly in Asia, which was formerly at risk is now free from malaria, there are still about 360 million people living in malaria-infected areas, the majority of which are in Africa.

But even in Asia malaria is once more on the increase—an indication in itself of the complexity of the problem of malaria control (Bruce-Chwatt, 1977).

According to the climatically-controlled chances of survival and reproduction of the mosquito vectors, malaria is transmitted either seasonally or all the year round. During transmission-free periods, the human constitutes the natural reservoir of the plasmodia, from which new mosquito generations can infect themselves and thereby maintain the transmission cycle. Nothing save continuous re-infection throughout the year can lead to a certain degree of immunity. In areas of year-round transmission, malarial mortality is particularly high among infants and young children who have not yet built up immunity—a matter of time. In contrast, in areas of seasonal transmission, occurring mostly at the beginning and towards the end of the rainy season, persons exposed to it develop a lesser immunity. The result is that not only babies and small children but also adults frequently succumb to clinical malaria. Seasonal epidemics may then arise, which can in their turn lead to higher rates of morbidity and mortality and, at times of intensive agricultural activity, to economic losses for rural families.

The distribution of malaria is determined by *human-ecological factors* such as the state of immunity, age occupation, the types of housing, sleeping-provision and settlement, the forms of agriculture, migration and the population density; set against these are the factors bearing on the *ecology of the transmitting mosquito,* comprising species of anopheles and their life habit, the blood sucking and manner of depositing eggs, their flight range, duration of hibernation and life, as well as their susceptibility to plasmodia. For both the plasmodia and the vectors, environmental factors like climate, temperature, relative humidity, altitude, and water types play an important role. Different species of plasmodia possess differing epidemiological characteristics. The state of immunity of the human host, the natural or acquired tolerance through total or partial immunisation, as well as premunition (=immunity built up and maintained only in the presence of parasites) influence the epidemiological situation of malaria in a given area.

To date no systematic malaria eradication campaign has been conducted in Kenya. In some parts of the country, malaria is still an important cause of illness (see Fig. 17, back of Map 9).

On the other hand, Kenya has played a significant role in the history of research into malaria, for it was P.C.C. Garnham, when working in the Division of Vector-borne Diseases in Nairobi in 1947, who elucidated the liver cycle of the malaria parasite.

Malaria has existed since time immemorial in the humid-warm coastal areas bordering the Indian Ocean and Lake Victoria. Before the arrival of the Europeans, altitudes above 1,500 m appear to have been malaria-free, for example, malaria epidemics being mentioned in Nairobi only from 1902 onwards (Symes, 1940; De Mello, 1947). Population movements brought about by World War I—such as recruitment from malaria-free areas, followed by assignment to malaria-infected areas and demobilisation and return to the home regions—contributed to the dissemination of malaria. Areas in Nandi and Uasin Gishu (Sirikwa County), which are situated at higher altitude, provide a case in point. After World War I the colonial development of the area by rail and road construction, the farm and plantation economy with its related labour movements, and the general increase in population mobility, all facilitated the further spread of malaria (Matson, 1957; Robert, 1964, 1974).

Indeed, John L. Gilks, a critical observer of the health situation during the colonial period, already considered malaria a social disease, which would not be successfully overcome without raising the standard of living of the public at large (Medical Report for 1929, p. 17).

It is possible to present the contemporary epidemiological situation in Kenya only in rough outline because large-scale malariological investigations are not available.

Figures from the hospital reports and the "Weekly Notifications of Infectious Diseases" from the district medical officers provide an inaccurate picture, since the true reference figures are unknown, and the diagnoses usually remain parasitologically unconfirmed. The diagnosis of "clinical malaria" often covers a multitude of other febrile illnesses. There is even a tendency to diagnose malaria more frequently as the season of expected malaria wears on. In those areas in which malaria

* This disease and the subsequent diseases in the following chapters are given the three-digit categories according to the Manual of the Internat. Statist. Classification 1975 Revision Vol. 1; WHO Geneva 1977.

** The definition of this disease and of the following diseases are cited after the list of the Council for International Organizations of Medical Sciences CIOMS: Communicable Diseases, Provisional International Nomenclature; Geneva 1973.

occurs frequently, parasitologically unconfirmed febrile conditions are naturally enough more often diagnosed as malaria than in areas where it is rare.

Over a period of time, original bias can thus simulate a pattern of distribution which will not necessarily stand up to parasitological examination. It is further promoted by the map of malaria transmission in the Atlas of Kenya, long since reprinted but apparently uncorrected, which takes its bearings essentially from contour lines. Thus it represents only the duration and to a certain degree the intensity of malaria transmission over the course of the year (see Map 7).

The most important sources of information concerning the distribution of malaria are the Reports of the Division of Vector-borne Diseases of the Ministry of Health, which in addition to the Nairobi headquarters maintains branches in several parts of the country. From these focal investigations into the distribution of malaria, control measures and even research are conducted.

Plasmodium falciparum is the most common agent of malaria; the other species of plasmodium also occur, but do not play a major role.

In Kenya the most important vectors of malaria are *Anopheles gambiae* and *Anopheles funestus. Anopheles gambiae* prefers temporarily sunlit, muddy pools, often resulting from human activities. The high temperatures generated in these pools, promote the rapid growth of the mosquitoes, often within five days. For this reason *Anopheles gambiae* generally occurs at the beginning of the rainy season. *Anopheles funestus* prefers clear and shallow water with vegetation, especially swamps rich in vegetation, quiet lakes or ponds fringed with vegetation, as well as clear streams. Areas of seasonal inundation serve as breeding grounds for both species, with *Anopheles funestus* enjoying a certain ecological advantage. In some places *Anopheles funestus* presents an alternating density, inversely proportional to precipitation, with the result that it alternates with *Anopheles gambiae,* thereby ensuring transmission all the year round (Fendall and Grounds, 1965 c; McCrae, 1968; Roberts, 1974).

In Kenya zones of varying malaria intensity can be distinguished. The first criterion is the duration of transmission in the course of a single year; this is determined by the seasonally-fluctuating density of the mosquito vector, which itself in turn is caused by the seasonal fluctuations of precipitation, temperature, and humidity. A rough distinction can be made between transmission throughout the year, as against transmission for 3 – 6 months of the year, and for less than 3 months a year. A fourth grade is found in the seasonal transmission occurring in the vicinity of streams in arid areas, and a fifth one is constituted by those malaria-free zones lying above 2,000 m, since the vector normally is no longer found there.

Another criterion is the age-specific spleen rate and the parasite rate in the blood of children aged 2 – 9. If an enlarged spleen or malaria parasites are found in the blood of more than 75% of children in this age group, the term holoendemic malaria is applied. A 74 – 50% rate of infection or enlarged spleen is termed hyperendemic, and a rate of 49 – 10%, mesoendemic; below 10% the term hypoendemic is applied. Using these criteria, different zones are distinguished in Kenya where malaria occurs in a characteristic manner (Fig. 17, back of Map 9; Table 23 and Atlas of Kenya, 3rd Edition, 1970 Map No 47).

Table 23. Malaria Epidemiology by Type and Area (from Roberts, 1974)

Classification/degree	Spleen rate age 2 – 9	Area
1. Endemic		
(a) holoendemic	>75%	Coast Province, coastal area; Tana River, Kano Plains, Taveta,
(b) hyperendemic	50 – 74%	North Nyanza, Bungoma, Busia, Shimba Hills (Coast).
(c) mesoendemic	10 – 49%	Machakos, Kitui, Thika; parts of North Nyanza, Murang'a and Embu below 1,300 m.
(d) hypoendemic	<10%	Meru, Pokot, Samburu, Isiolo, Baringo.
2. Epidemic	Variable	Highland over 1,600 m with high rainfall and dry areas with exceptional rainfall: Masailand, Nandi, Kericho, Kisii, NFD, Eastern Kitui, Londiani, Elgeyo.
3. No transmission (sometimes anophelism without malaria)	None	At altitude over 2,000 m: Aberdares, M. Kenya, M. Elgon (forest, moorland, plateaux).

The malaria situation in the Coast Province: The coastal province, including the lower course of the Tana, is considered a holoendemic malaria region with transmission occurring within a period of 6 month or more (Roberts, 1974). The continuous investigations and selected controls in some key projects by the *Division of Vector-borne Diseases* (DVBD) of the Ministry of Health have succeeded in considerably improving the malaria situation without, however, in the least affecting the overall situation in the coastal area. Thus in the coastal settlements, especially Mombasa and Malindi and the important tourist-oriented seaside resorts malaria has receded markedly due to drainage and insecticides.

Indoor spraying since 1949 has been combined with fortnightly chemoprophylaxis among selected population groups since 1966 and has achieved the virtual eradication of the parasite among children under 15 in the Malindi urban district; there has also been a lowering of the parasite rate to 2% in the periurban district, whereas more than 10% of the children of the surrounding locations still have malaria parasites. Prior to the introduction of controls, the parasite rate stood at 48 – 75% (Ministry of Health Annual Report 1962; DVBD Annual Reports, Nairobi 1969 – 1973). In Faza (Lamu) the use of insecticides has likewise succeeded in lowering the parasite rate to about 3%. In Kilifi, Mombasa, and the coastal section between Mombasa and Msambweni malariometric investigations carried out in 1973 showed

a parasite rate of 6 – 8% (DVBD Annual Report, Mombasa 1973).

For years considerable efforts have been made in the various resettlement and irrigation schemes to reduce what was originally an extremely high malaria infestation. In the Shimba Hills Resettlement Scheme, where 70% of all children had malaria parasites in 1959, fortnightly chloroquine medication of the more than 7,000 settlers, combined with the use of larvicides, succeeded in lowering the rate to less than 5% (DVBD Annual Reports, 1965, 1972, 1973). Because of its originally catastrophic malaria situation, the eventual success of this project had been in doubt.

The irrigation scheme at Hola (Galole) on the Tana River and the Ramisi sugar estates near the Tanzania border have been subject to larvicidal and chemoprophylactic treatment since 1963. Here too the parasite rate among the settlers could be kept at a low level, falling to 6% at Ramisi and even below 1% at Hola (DVBD Annual Reports, 1969 – 73).

Western Kenya (Western and Nyanza Province and Sirikwa County): Western Kenya, humid and warm in the vicinity of Lake Victoria Basin, at an altitude of 1,100 – 1,500 m above sea-level, is the second most important of the holo- and hyperendemic malaria zones in the country. When Lake Victoria was experiencing high water levels at the beginning of the sixties, flooding carried the intensity of malaria transmission from the coastal strip far into the interior of the country (Ministry of Health Annual Reports, 1960, 1962).

The DVBD branch in Kisumu and lately also that in Kimilili (Western Province) controls mainly the Kano Plains with their rice and sugar cane plantations. In Kisumu, where the larvicidal activities in the urban district are supported by the DVBD, occasional chloroquine prophylaxes are carried out in the peri-urban district, where in 1965 the parasite rate was still five times higher (2.4%) than in the urban district (0.5%). However, a study group of the WHO and the Medical Research Council recently noted far higher rates of infection (DVBD Annual Report, 1973).

In some locations in the Nyanza Province—in Vihiga, Khwisero, and in the Kisii District—the introduction of fishponds to increase the production of protein has also considerably increased the mosquito density, particularly that of *Anopheles funestus.* More than 80% of the thousands of newly constructed fishponds were found to be infected, and this caused a considerable increase in the chance of malaria transmission and a correspondingly increased malaria morbidity (DVBD, Annual Reports 1965, 1973).

In *Nandi District* (Chemasse, Nandi Hills) and in the Uasin Gishu District (Turbo, Kipkaren, and Lugari Settlement Scheme) at altitudes between 1,500 and 2,000 m a very unstable malaria epidemic had spread in the course of agricultural development in the early fifties, striking a non-immune population. The same holds true for the Londiani and Molo townships on the railroad in the direction of Kisumu at altitudes of up to 2,500 m. Insecticides, in combination with chemotherapy or without it, were able to lower the originally high parasite rate to 8 – 10%, and in some places to below 3.5% (Roberts, 1956, 1964, 1974).

In *Kericho District* Garnham's first experiments with D.D.T. in 1946 were successful in a very unstable malaria situation with a parasite rate of 37%, practically stopping malaria transmission to the present day (Roberts, 1974).

In *Central Kenya,* in a malaria situation very much dependent on the particular altitude, the control measures of drainage and larvicides are carried out only in the larger settlements. The extensive irrigation installations at Mwea Tebere (Embu District) in a mesoendemic area are again successfully controlled by the DVBD.

In *Machakos District,* usually a mesoendemic malaria zone with transmission during 3 – 6 months of the year, malaria epidemics occurred in 1951 and in 1961; there was a renewed outbreak in 1972 after remarkably few outbreaks over the intervening years. It was possible to contain this outbreak by massive application of chloroquine (DVBD Annual Report, 1972).

Apart from a few exceptions at the valley openings to the west, near Kitale, the high altitudes (above 2,000 m) of the *Rift Valley* are free from malaria. In the southern Rift Valley, in the Kerio Valley, and further north in the semi-arid areas, malaria occurs only occasionally and epidemically along the stream courses. In the Perkerra Irrigation Scheme at Lake Baringo, the DVBD carried out control measures. In the semi-arid areas of the north and north east the chances of malaria transmission are rather reduced, because scanty precipitation and a high level of aridity do not encourage the vector populations. Following extraordinary rainfall there may be local malaria epidemics to the extent that a sufficiently large reservoir of plasmodia is available in humans. Such a situation is favoured by the increasing mobility of the population.

As Gilks stressed as early as 1929 (op. cit.), the control of malaria is a question of raising the standard of living of the majority of the population. This still applies even though highly effective chemotherapeutics and insecticides are now available. In spite of the increasing development of the basic health services, that represent the backbone of malaria control, systematic malaria control is not possible without massive application of logistical, organisational and financial means, and includes above all the necessary cooperation of the entire population. Today, almost 50 years after Gilk's statement, these prerequisites are still not present in sufficient degree, although the population's preparedness to prevent malaria is clearly on the increase.

2. Trypanosomiasis

(Sleeping Sickness, ICD 086)

Trypanosomiasis in tropical Africa is an infectious disease found and caused by haemoflagellates of the genus *Trypanosoma* and transmitted by *Glossina* species (tsetse flies).

Parasitologically, epidemiologically and clinically two types of sleeping sickness occur in Kenya; Gambian trypanosomiasis caused by *T. gambiense,* and Rhodesian trypanosomiasis, caused by *T. rhodesiense.*

The vector of the former are tsetse flies of the palpalis group, the latter of the morsitans group, each having its own distinct biology, which thus influences the epidemiological pattern of the disease. In recent years *Glossina palpalis fuscipes* has also been found to transmit *Trypanosoma rhodesiense.*

The history of the distribution of human trypanosomiasis in Kenya and its present extent cannot be discussed without a short reference to the larger framework of sleeping sickness in East Africa.

The agent of sleeping sickness, *Trypanosoma gambiense,* which first spread epidemically in East Africa, was probably imported from the Congo Basin and the southern Sudan to Uganda in the course of increasing social, economic, and military mobility at the end of the nineteenth and the beginning of the twentieth century. The 1889 expedition of H. M. Stanley in search of Emin Pasha certainly played no more than a symptomatic role (Langlands, 1967).

The vectors of the *Glossina palpalis* group of tsetse flies were densely dispersed in the gallery forests of Uganda and Kenya, along streams, and in the coastal areas of Lake Victoria, so that the imported trypanosomes met an ample reservoir of vectors and thus with conditions favourable for the distribution of sleeping sickness among the population.

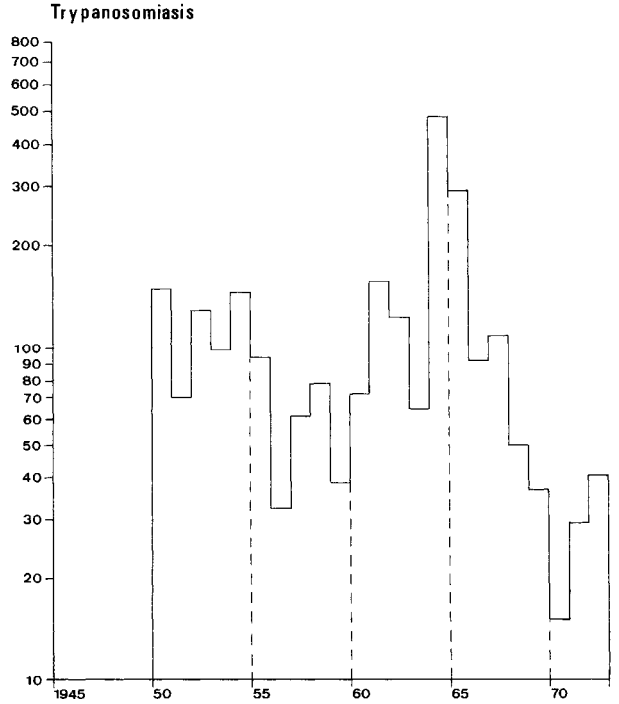

Figure 5. Trypanosomiasis, number of annual notifications 1950 – 1972 (Source: Willett 1965, Annual Reports EATRO, Bulletin of Infectious Diseases, Ministry of Health, Nairobi, 1962 – 1972)

Since the start of the twentieth century sleeping sickness has spread in several waves from Uganda to Kenya. The islands of Musenga and Mageta were the first to be affected, and from them it moved over to central and south Nyanza on the mainland (Onyango, 1974; see Fig. 15, back of Map 7).

In the course of the ensuing sixty years, Kenya experienced several epidemics, the first one of which lasted from 1902 to 1908. Emigration of the population allowed the disease to die down, so that a relatively low grade of endemicity was attained between 1911 and 1920. The deserted areas were left to revert to a natural state, increasing the density of Glossinae in the process. After 1920 the population increasingly returned to the deserted areas, thus enabling a second epidemic wave to rise between 1920 and 1930. However, at this time chemotherapy and bush clearance were already available as control measures, and the epidemic could be contained within certain limits. In a third dissemination wave from 1940 to 1950 the three districts of Kadimo, Alego, and Samia in the Western Province were particularly affected, together with the Nyando Basin west of Kisumu and the Kuju-Migori river basin in southern Nyanza. Massive control measures—above all the spraying of DDT in the gallery forests along the rivers—proved successful in suppressing sleeping sickness in these areas during the early fifties (see Map 7).

Until then it had largely been sleeping sickness caused by *Trypanosoma gambiense* and transmitted by *Glossina palpalis fuscipes.* Since 1940, however, Tanzanian migrant workers moving to the sugar cane plantations of Busoga in Uganda have brought with them *Trypanosoma rhodesiense,* which in turn encountered the vector *Glossina pallidipes* in these coastal areas which had reverted to a natural state, following the exodus of the population. Here this species had spread considerably, along with *Glossina palpalis fuscipes.* During the following decade and a half, particularly since the return of the population to the formerly deserted areas following the independence of Uganda, sleeping sickness once again increased. On this occasion it was caused by *Trypanosoma rhodesiense.* In the course of this spread a renewed importation of the sleeping sickness occurred from the west in Kenya.

Lesser outbreaks of *Trypanosoma rhodesiense* sleeping sickness, transmitted by *G. pallidipes,* occurred in the Sakwa and Mutonga Districts in 1953/54, in Samia in 1957/58, and in Yimbo in 1961. In 1958 Heisch et al. were able to identify the bushbuck as the animal reservoir. The rise in the surface-level of Lake Victoria caused by several years of abundant precipitation and the subsequent increase in humidity far into the hinterland led to a great increase in the Glossina population, again especially among *Glossina palpalis fuscipes.*

The years of 1964/65 saw an explosive outbreak (Fig. 5) in the Alego District with a total of 650 known cases. For the first time in the history of trypanosomiasis, the vector of *T. rhodesiense* was *Glossina palpalis fuscipes;* under favourable climatic conditions this has peridomestically spread from the immediate vicinity of streams and

shore into the farmyard hedges and the thickets surrounding the fields and become very dense in numbers (Willett, 1965). This has demonstrated and confirmed certain earlier assumptions that *Glossina palpalis fuscipes* is able to transmit trypanosomae of the *T. gambiense* type as well as those of the *T. rhodesiense* type. In addition it proved possible to establish that the different transmission cycle by way of the *G. morsitans* or the *G. palpalis* group, and their respective preference for a host, already determines the virulence of a *T. rhodesiense* course or a *T. gambiense* course (Willett, 1965). Beyond this the assumption was confirmed that not only wild ungulates but also Zebu cattle may serve as potential reservoirs for the agents of trypanosomae pathogenic to humans (Onyango et al., 1966).

In southern Nyanza, the second focus in Kenya, the last outbreaks of the *T. gambiense,* transmitted by *G. palpalis fuscipes,* had been terminated in the Kuja-Migori area in 1958/59, due to very intensive anti-*glossina* measures on land and water (see Map 7).

Since 1962, however, there have been epidemic occurrences of sleeping sickness of the *T. rhodesiense* type over a limited area of about 500 farmsteads in the Lambwe Valley resettlement areas. In this fertile grassland *G. pallidipes* of the morsitans group was identified as the vector. A zoonosis was evidently encroaching upon a population recently immigrating within the framework of an agricultural resettlement project.

This focus is still not under control, whereas the old areas of sleeping sickness in the Western Province have largely been brought under control by application of insecticides, combined with bush-clearance, and planned agricultural utilisation with the close cooperation between the Division of Insect-borne Diseases of the Ministry of Health, the Tsetse Control Division of the Veterinary Department of the Ministry of Agriculture, and the recent support of the United Nations Development Programme (R. J. Onyango, 1974). The development of sleeping sickness in Kenya, particularly over the past 20 years, has once more demonstrated first, that new outbreaks of sleeping sickness arising from ecological shifts on the part of nature or man can occur at any time in areas long known to be infested by *glossinae,* and secondly, that these factors warrant special attention within the framework of agricultural and animal husbandry development projects. Correct planning with regard to certain ecological rules can arrest the danger, whereas wrong planning only conjures it up again. Although the tsetse fly, and especially the potential vectors of sleeping sickness, also occur over large parts of Kenya (see Tsetse Distribution Map, Kenya National Atlas, 3rd Edition, 1970), sleeping sickness has never left its original foci in central and southern Nyanza, a fact surely largely attributable to decades of efforts resulting in their control.

3. Leishmaniasis
(ICD 085)

Leishmaniasis is a group of infectious diseases, which occurs in many countries and is caused by protozoa of the genus *Leishmania* and transmitted by sandflies *(Plebotomus spp.).* Of the three distinct clinical conditions only visceral Leishmaniasis (kala azar) occurs in Kenya; diffuse cutaneous Leishmaniasis has so far been found only in a few locations.

a) Visceral leishmaniasis (kala azar): An acute or chronic infection caused by *Leishmania donovani* and transmitted in Kenya by sandflies of the Synphlebotomus group. The clinical manifestation includes irregular fever, anaemia, leucopenia, splenomegaly, hyperglobulinaemia, finally, kachexia, resulting not infrequently in death.

Before World War II visceral leishmaniasis was a disease only sporadically observed among the nomads of the north. During the British Army's Ethiopian Campaign, between 1941 and 1943, an outbreak of kala azar occurred in the vicinity of Lake Rudolph which affected 135 soldiers (Fendall, 1952). Wakamba troops from the northern part of the Kitui District were probably responsible for its introduction at home, the first cases occurring there in 1948. Due to conditions especially favourable to transmission, a serious epidemic developed between 1952 and 1954, which spread further south along the eastern border of Kitui District and north among the Tharaka on both sides of the Tana river.

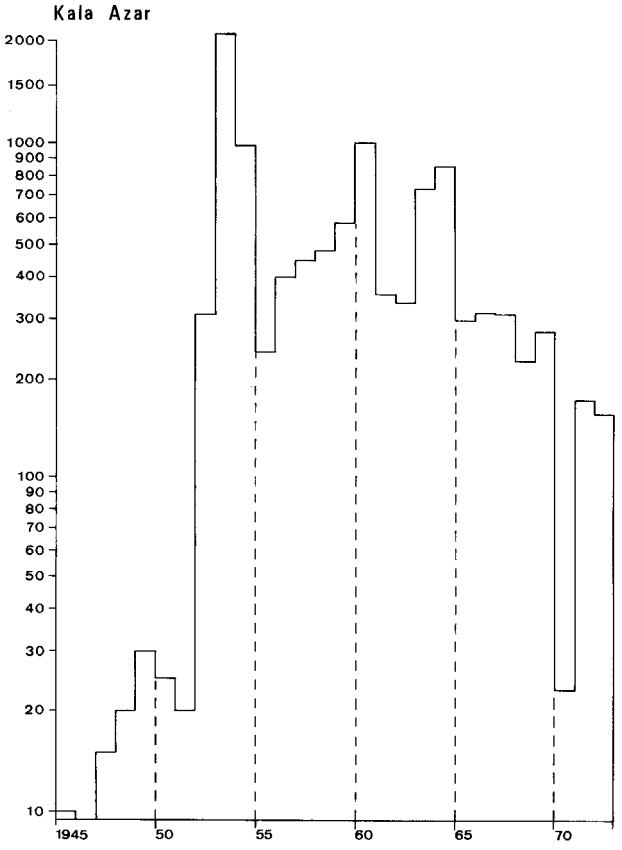

Figure 6. Kala azar, number of annual notifications 1945 – 1972 (Source: Fendall and Grounds, 1965 c, Bulletin of Infectious Diseases, Ministry of Health, Nairobi, 1962 – 1972)

The figures reported in the period 1954 – 1964 are essentially due to the development of this epidemic (Fig. 6). The ensuing slow decline in numbers is indicative of the self-adjusting endemic state.

The regional distribution of cases of kala azar is reflected in the analysis of Annual Returns of Diseases for the years 1968 – 1972; the following average values per 100,000 inhabitants in the catchment area of the hospitals are detailed below:

Marsabit	54	Tambach	12	Meru	6
Kabarnet	49	Lodwar	11	Kitale	6
Kapenguria	41	Machakos	9	Nakuru	6
Kitui	16	Garissa	7	Embu	3

Besides this relatively recent area of sedentary population, where the infectious condition has stabilised in the meantime in the sense of becoming endemic, investigations undertaken on account of these results showed further endemic foci of kala azar in the northern offshoots of the Rift Valley, in the Kerio Valley (Elgeyo Marakwet), in the Baringo and Samburu District, and in the West Pokot and even among the semi-nomadic herding population up to Uganda (Amudat) as well (McKinnon and Fendall 1955, McKinnon, 1962 a; Fendall 1961; Wykoff et al. 1968, 1969).

A recent investigation was carried out with the aid of the Montenegro skin test using the *Leishmania tropica* culture antigen in the well-known endemic area of the North Eastern Province with positive results in the three divisions of Tseikuru (29.5%), Gatunga (17.8%), and Nuu (20.2%). There was no positive case among 97 persons tested in the northern Rift Valley and only 5 positive outcomes among 116 persons tested in West Pokot (Mutinga and Ngoka, 1975).

Epidemiology: The epidemiology of the disease is essentially determined by the biology of the transmitting sand flies *(Phlebotomus martini)* and part of the Synphlebotom complex (Wijers and Minter, 1962, 1966; Wijers, 1963; Wijers and Mwargi, 1966). These sandflies exist in arid and semi-arid areas, mainly below 700 m, although they can still be found at an altitude of 1,000 m in West Pokot and Baringo. They hide in the ventilation shafts of *Macrotermes belicosus* anthills and only leave these on humid-warm, and calm evenings at the beginning and end of the rainy season. They have a very short flight radius. Indeed, it is only when man stops in the vicinity of anthills that he runs any risk of contact and transmission (Wijers, 1974). Here nomadic herding people come into sporadic contact when pitching their tents in the vicinity of the anthills. Although all age groups are affected, the greatest frequency occurs among children and younger adults (McKinnon, 1962 b). Among sedentary people those families dwelling near such anthills are the main ones to be affected; owing to the large number of anthills in this area, epidemics occurred between 1952 and 1954, in 1957, 1960 and again from 1963 to 1964. So far no animal reservoir has been discovered (Heisch, 1954, Heisch et al., 1956, Wijers, 1974).

As the endemicity increases, the peak of infections shifts to younger age groups. In the Kitui and Taraka areas, kala azar has by this time become a disease of young children (Southgate and Oriedo, 1962; Wijers, 1971). In some parts the disease has affected 70% of the population (Wijers, 1966). Wijers (1974) stresses that the infection rate is particularly high in hunger-stricken areas, among which the north of Kitui District and the Tharaka Division are to be counted, and that well-fed persons evidently appear to offer better resistance to this disease.

In some cases the disturbances in this area in 1965, which were connected with attacks by bands of robbers (Shifta), caused the population to leave the area, and the number of infections decreased, only to rise again after peaceful conditions were restored and the population returned to the area.

b) Cutaneous leishmaniasis, caused by *Leishmania tropica,* does not seem to occur in East Africa, although a few cases have been observed on the southern slopes of Mount Elgon in the Bungoma District since 1968 (Mutinga, 1975 a, b).

An investigation based on the Montenegro skin test showed a positive reaction among 16.5% of the 327 persons tested at Kapsokwony (Mutinga and Ngoka, 1975). Apart from several nonanthropophile phlebotomes, this new humid and relatively temperate area (in contrast to the hot and dry areas of occurrence of visceral leishmaniasis) was found to accommodate *P. pedifer,* which transmits cutaneous leishmaniasis in Ethiopia and the Sudan. It must be assumed that this species of phlebotome transmits cutaneous leishmaniasis here as well, but the experimental proof is still lacking.

Leishmaniases have been isolated from rock and tree hydraxes and a giant rat, and there is some suspicion that these animals may act as reservoirs (Mutinga, 1975 a, op. cit.).

The case of a cutaneous leishmaniasis contracted by a health worker trapping sandflies in the caves of the suspected animal reservoir has proved the transmission cycle recently (Arap Siongok and Birgen, 1976).

4. Arthropod-borne Virus Diseases
(ICD 060 – 066)

Arthropod-borne virus diseases (Arbovirus diseases) are a group of infectious diseases transmitted by arthropod vectors (mosquitoes). A number of arboviruses are known to be present or are assumed to occur or to have occurred temporarily in Kenya. Some viruses have been isolated, for others serological evidence was found (Metselaar, 1974 a).

Arbovirus infections are of public health interest in East Africa, where a very widespread epidemic of *Aedes aegypti*-transmitted chikungunya fever occurred in Tanzania in 1952 – 53 (Robinson, 1955), and was also observed in Kenya. A vast epidemic of o'nyong-nyong-

fever, transmitted by *Anopheles spp.* raged through Uganda, Kenya, and Tanzania in 1959 – 1960 (Haddow et al., 1960). In both epidemics the infection rate was very high, yet fatal cases were not reported.

In a large multipurpose serological survey (1966 to 1968) in three selected districts: Malindi (Coast Province), Kitui (North Eastern Province), and Siaya (Nyanza Province), a total of 1,500 randomly selected sera were tested as to the presence of arbovirus antibodies (Geser et al., 1970). The sera were tested for antibodies against 3 Group A arboviruses (chikungunya, o'nyong nyong, sindbis), 6 Group B arboviruses (zika, yellow fever, West Nile, Banzi, Wesselsbron, dengue-1) and Bunyamwera virus. Chikungunya and o'nyong nyong infections, immunologically not distinguishable, showed the most dramatic geographical variation. Fifty per cent of all the people examined had antibodies in Siaya (Central Nyanza) and Malindi (Coast), whereas this antibody is virtually absent in Kitui, a fact perhaps explained by the dryness of this area and its low population density. The presence of antibodies even in the age group 0 – 4 indicates that this infection is still present in the population. This is supported by the observation of Bowen et al. (1973), who found that in the Nyanza Province south of Kisumu in an area on the coast of Lake Victoria proposed for an irrigation project, over 60% of schoolchildren had antibodies against the chikungunya -o'nyong nyong complex. The authors considered the irrigation project to be a severe threat to the population, as well as constituting a breeding place concentration.

Bunyamwera virus antibodies were isolated in 26% of samples from Malindi, only 5 – 6% of sera from Kitui and Siaya were positive. The same was observed for dengue-1 antibodies; 47% of the samples were found to be positive in Malindi. There is no clinical evidence of dengue fever. Metselaar, Henderson et al. (1974) have isolated from 147,000 mosquitoes in 7 areas of Kenya a number of viruses known to cause arbovirus infections in man as well. Among these were Semliki Forest virus (group A), Banzi virus (group B), Bunyamwera and Beliefe virus (Bunyamwera group).

As far as yellow fever is concerned, there was only one case in 1943 in Langata Forest in Kenya (Mahaffy et al., 1946). Kenya has never experienced yellow fever epidemics of the sort that have occurred in the neighbouring countries to the north—in the Nubian Mountains of the Sudan in 1940 and in south west Ethiopia between 1960 and 1962. In the course of years isolated cases of protective yellow fever antibodies have been found repeatedly in monkeys and bush babies. Similar investigations in Uganda have, on the other hand, disclosed that more than 30% of the monkeys are in possession of protective antibodies (Haddow, 1952).

Although Geser et al. (op. cit, 1970) in their sample survey found antibody reactions against yellow fever virus in more than half of the serum tests carried out in Malindi District, they were of the opinion that these were either cross-reactions to other arboviruses or the result of yellow fever vaccinations. However, the serious epidemic in Ethiopia yielded proof of yellow fever protective antibodies in 7.5% – 15% of all human sera tested in Marsabit, Moyale, Lodwar, and Lokitaung in the northern districts of Kenya. The relatively high proportion found in Marsabit is attributed by Metselaar to the importation of yellow fever by members of the Ethiopian Burji tribe, who live there but now and then travel back to their home region, which was affected by the yellow fever epidemic in 1960 – 62. In the environs of the isolated, wooded sugar-loaf mountain of Marsabit, 150 km south of the Ethiopian border, neither protective antibodies among monkeys and bush babies nor yellow fever viruses among mosquitoes were to be found (Metselaar et al., 1970, 1974 b).

The problem of the potential transmitter of yellow fever is not unequivocally solved. In Kenya the classical transmitters *Aedes aegypti* and *Aedes simpsoni* do not appear to be particularly anthropophile. *Aedes africanus,* the vector of yellow fever, which lives in treetops, was not found in any of the three areas of Kenya in which yellow fever virus had evidently circulated some time ago. *Aedes simpsoni* was not found in the Langato Forest or in Marsabit, but only on the coast where it is not very partial to stinging humans; nevertheless, all three species of Aedes must remain under consideration as potential vectors of yellow fever, even in Kenya. According to Metselaar, the ecological balance may presently be such as to permit in principle the encroachment of yellow fever viruses wherever it seems to be present in primates, even if at present, according to all appearances, the probability is too restricted. Moreover, some aspects of the epidemiology and epizootology of yellow fever in Kenya are still not elucidated, so that constant vigilance and readiness are recommended to facilitate action in the event of any sudden requirement for yellow fever vaccinations.

5. Diseases Caused by Bacteria and Bacteria-like Organisms and Transmitted by Arthropods

This group of infectious diseases includes two important diseases or disease groups, in which rodents constitute an important natural reservoir for human infections.

For this reason these infections often disappear from the human context for years until they suddenly recur through contact with the animal reservoir. The occurrence of human cases is frequently preceded by unrecognised epizootics. In the case of plague, the observation of such an epizootic infection is of the greatest importance to prevent a threatening plague breaking out among the population.

Further typical representatives of this group are the endemic *tick-borne spotted fever* and the *endemic tick-borne relapsing fever,* which occupy a special position in so far as ticks act simultaneously as reservoir and vector, due to their characteristic ecological and biological behaviour. The epidemic infections of rickettsia and relapsing fever

transmitted by lice make use of man himself as a reservoir and generally occur only in mass situations or catastrophies accompanied by widespread louse infestation. For years now these forms have scarcely been seen in East Africa and a description may therefore be omitted.

a) Plague (ICD 020): Plague, primarily an infectious disease of rodents, occurred in the past even in pandemic form, but outside its enzootic areas it is now reduced to mere foci in a few parts of the world. Large forest areas and highlands are ecologically predisposed to it. The agent *Yersinia pestis* is transmitted by rodent fleas such as *Xenopsylla cheopis* within the rodent population, as well as by the human flea *Pulex irritans.*

The natural reservoir exists in rodent populations, from which the agent is transmitted to semi-domestic and domestic rodent populations, and from these it passes to man.

Four clinical forms occur among humans: the bubonic plague, pneumonic plague, septicaemic plague, and abortive forms. Of the three pandemics during the Christian Era, the first Justinian plague of the sixth century is presumed to have affected East Africa (Pollitzer, 1954).

In 1697, during the Arab occupation, a plague epidemic occured in the then Portuguese Mombasa.

In the course of the third pandemic of the 19th and 20th centuries, the plague probably spread on the then customary trade routes across the entire African continent. During the closing decades of the 19th century, the various Christian missions thus repeatedly reported outbreaks of plague in East Africa. In Kenya it was mainly the construction and running of the Mombasa to Kisumu railway that played the important role.

Massive outbreaks occurred between 1900 and 1914, particularly in the vicinity of larger settlements (Nairobi 1902, Nakuru and Kisumu 1904, Mombasa, 1912). Nairobi experienced numerous outbreaks, the last one in 1941 with 781 cases (Roberts, 1950). In the year 1912, a plague epidemic occurred on the southern outliers of Kilimanjaro, which was described in great detail by Lurtz (1913), who already suspected at that time that it was not a matter of importation from outside via the domestic rat or by humans infected by plague, as was commonly assumed, but of encroachment from a local plague focus among the wild rodent population. As a result of the outbreak of war, this important observation was lost and only revived forty years later by Heisch et al. (1953).

From 1920 onward the rural foci of plague moved more to the fore: Rongai and Solai, north of Nakuru in the Rift Valley on the outliers of the Aberdares and Mount Kenya (Kiambu, Murang'a (Fort Hall), Nyeri, Kerugoya). No plague occurred there before 1914, whereas between 1921 and 1930 more than 2,000 cases were reported annually.

After World War II plague occurrences receded rapidly. A remarkable outbreak at Rongai in 1953 was carefully investigated and once again diagnosed as having been caused by wild rodents such as *Avicanthus niloticus* providing the reservoir for *Yersinia pestis* (Heisch et al., op. cit., 1953). In Kenya the last small outbreak of five cases and one death took place near Murang'a in 1963. A number of smaller outbreaks in Tanzania near the Kenya border, at Mulu on the Kilimanjaro foothills in 1968 (Msangi, 1969), and near Arusha (1970 and 1972) also indicate the presence of enzootic sylvatic plague, which can at any time encroach upon the human population, a possibility underlined by the investigation of Davis et al. (1968).

Applying the microhaemagglutination test to over 20,000 rodents of different species from different parts of the country, they discovered in 2.5 – 10% of the sera significantly high antibodies against fraction I of the kapsel antigen of the plague agent *Yersinia pestis.*

In its enzootic and epizootic course, plague is a typical example of a nesting disease, with a constant latent threat of encroaching upon human populations. Particularly the geography of Kenya with its secondary mountain ranges, the intermediate altitudes of the great mountain massifs, and the saddle of the Rift Valley are peculiarly conducive to the flourishing of rodents. Precise observation of the migratory movements and mortality of wild and semi-domestic rodents and rats in the urban districts is necessary and is carried out by the health authorities in order to recognise a sylvatic rodent epizootic in time to prevent the flood- or drought-conditioned transfer of a sylvatic rodent epizootic to an area which happens to be settled by humans.

b) Rickettsioses (ICD 080 – 083) Rickettsioses are defined as infections with various species of Rickettsiae —bacteria-like micro-organisms, which are transmitted by lice, fleas, or ticks. With the exception of the epidemic, louse-borne typhus, which occurs only in man and is caused by *Rickettsia prowazeki,* all rickettsioses have an animal reservoir, from whence fleas or ticks can transfer it to man. Neither louse-borne typhus nor flea-borne typhus (murine typhus), which is caused by *R. mooseri,* has been reported in Kenya so far, although the natural reservoir of murine typhus is at least known.

Tick-borne rickettsial diseases appear to be relatively rare in Kenya at the present time. Systematic epidemiological investigations have not been carried out in Kenya since the first observations among Europeans in the Kenyan Highlands were reported by Gilks in 1920 (cit. from Craddok, 1974 a) who then had discovered cases also among the African population. But, at least in former times, there evidently existed some immunity, acquired during childhood, which prevented the occurrence of clinical manifestations at a later stage (Craddock, 1961).

Since an exact diagnosis can be made only after substantiation derived from serological investigations, hospital statistics under the prevailing conditions mean little—in so far as the diagnoses of suspected cases are recorded at all (see Fig. 7).

In East Africa rickettsioses are anthropozoonoses (Heisch, 1957, 1960), for ticks and their hosts constitute the natural reservoir of the rickettsiae. In East Africa

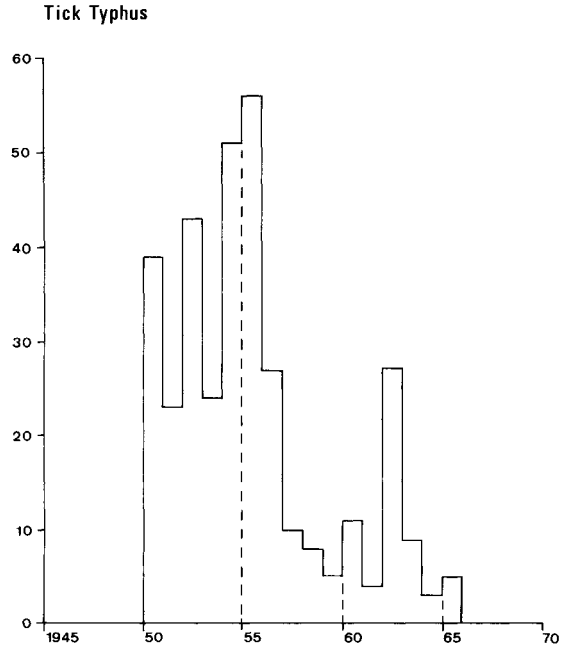

Figure 7. Tick typhus, number of annual notifications 1950 – 1972 (Source: Fendall and Grounds, 1965 c, Bulletin of Infectious Diseases, Ministry of Health, Nairobi, 1962 – 1972)

tick-borne typhus is caused by several variants of *Rickettsia rickettsii* (var. *piperi* and var. *conori*) ICD 0820.0, as well as *R. mooseri* (murine spotted fever) and *Coxiella burneti* (Q-fever) (Heisch et al., 1962). The rodent reservoir consists essentially of *Rattus rattus, Otomys angolensis, Arvicanthus abyssinicus, Lemniscomys striatus, Rhabdomys pumilio, Lophuromys aquilus* and *Aetomys kaiseri* (Heisch, 1961). The tick fauna is composed of *Haemophysalis leachi, Ripicephalus simus, R. sanguineus, R. pulchellus* and *Amblyomma variegatum* (Heisch, 1957, 1962). Details of regional variations in occurrence are not available.

Q-fever is caused by *Coxiella burneti,* another variety of rickettsia; since 1952 it has been occasionally diagnosed in Kenya. Evidently this anthropozoonosis is common among domestic animals, but without any major economic effect. The disease does not figure in veterinary medical reports. However, a special investigation did establish a more or less serious infection of dogs, camels, goats, and cattle. Human cases of Q-fever have been reported from Nairobi, Athi River, and the northern regions of Kenya (Craddock, 1974 b). More detailed studies by Vanek and Thimm (1976) have recently shown that Q-fever is present in man and cattle in all Kenya districts in which surveys have been made, in particular in the Eastern, Rift Valley, and Coast Province. However they assume that most of the adults, especially in the rural areas, are immune.

c) Relapsing fevers (Borrelioses transmitted by ticks and lice, ICD 087): Relapsing fevers are acute febrile infections caused by spirochaetes of the genus *Borrelia,* and are usually transmitted by ticks or body lice. They are characterised by febrile paroxysms lasting from one to seven days, and followed by a febrile period of from two to fifteen days.

Similar to the rickettsioses, the tick-borne borrelioses were of some significance in Kenya, whereas relapsing fever transmitted by lice and caused by *Borrelia duttoni* probably only once occasioned a more serious outbreak —in and around Mombasa in the years 1945 – 46, in which case the infection had probably been imported on an Arabian dhow (Garnham et al., 1947).

The occurrence of tick-borne relapsing fever is essentially dependent on the way of life of the transmitters, which at the same time constitute an important reservoir. The vectors are made up of different species and sub-species of *Ornithodorus,* a leather tick of partly wild and partly domestic occurrence. The wild species of *O. apertus* live in the burrows of porcupines and groundhogs, *O. porcinus domesticus* and *O. moubata* in various domestic surroundings. *O. p. porcinus* and *O. p. domesticus* are the most common vectors in Kenya (Walton, 1962 cit. after Fendall, 1965 c). These tick species are highly dependent on the temperature and humidity of their habitat. The rapidly changing conditions of life and domestic hygiene have since the mid-fifties, contributed to the virtual disappearance of tick-borne relapsing fever.

In the cool highlands of Meru, Kikuyu, Taita, Digo, and Kisii, with precipitation in excess of 1,000 mm, tick-

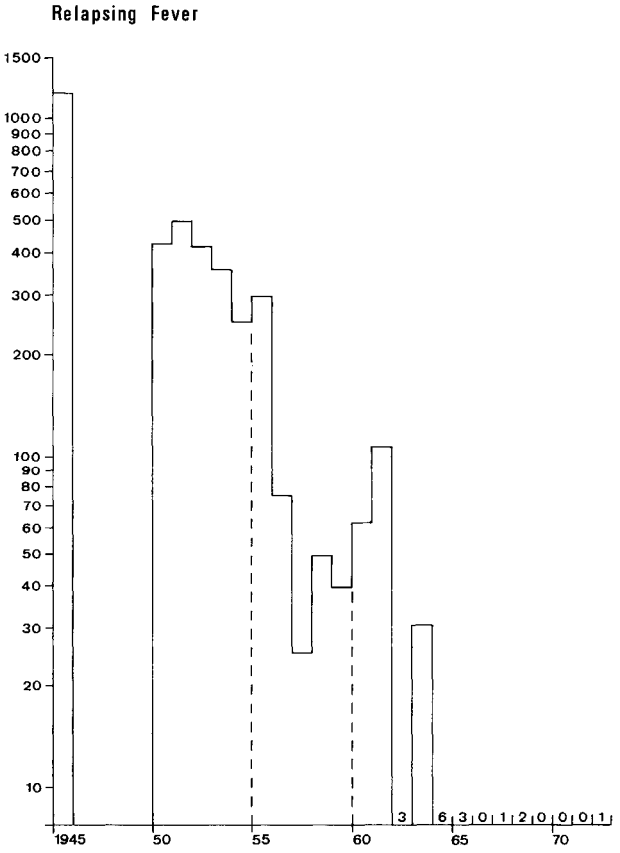

Figure 8. Relapsing fever, number of annual notifications 1945 – 1972 (Source: Fendall and Grounds, 1965 c, Bulletin of Infectious Diseases, Ministry of Health, Nairobi, 1962 – 1972)

borne relapsing fever posed a serious problem (Walton, 1955; Fendall, 1965 c). Thus children living in huts were particularly exposed, whereas adults, who spent more time outside, were less so. Cracks in the mud-built and urine-soaked sleeping quarters served as favourite hide-outs for the ticks. They also particularly favoured the infection of infants, who had not built up any immunity (Walton, 1950; Bell, 1953).

A recession in the frequency of infections began with the forced resettlement of the Kikuyu following the Mau-Mau Rebellion in 1952 – 1955, when people were obliged to leave their tick-infested huts and to move into square, better apportioned and ventilated ones. The introduction of bedsteads, the application of DDT for the disinfection of huts, and a general and clearly visible improvement of residential and living conditions, all contributed to the virtual disappearance of the disease (see Fig. 8).

6. Filarial Infections Transmitted by Arthropods

(Filariasis, ICD 125)

Nematodes of the Filarioideae family are described as filariae; their females carry living larvae, so-called microfilariae, which are periodically or non-periodically found in the peripheral blood or skin, from which sites they are taken up by insect vectors during the act of blood-sucking. The adult filariae (macrofilariae) live as parasites, partly in the connective tissue and partly in the lymphatic system. The following species are differentiated: *Wuchereria bancrofti* (ICD 125.0) and *Brugia malayi* (ICD 125.1) which are the causal agents of elephantiasis, as well as *Loa loa* (ICD 125.2), *Mansonella ozzardi* (ICD 125.5), *Acanthocheilonema perstans* (ICD 125.4) and *A. streptocerca* (ICD 125.6), *Onchocerca volvulus* (ICD 125.3). With the exception of *Wuchereria bancrofti* and *Onchocerca volvulus,* the remaining filiariae are of subordinate importance in East Africa; that is, nothing is known about their frequency or distribution.

a) Wuchereria bancrofti (bancroftian filariasis): Elephantiasis *("matende")* and Hydrocele *("Mapumbu")* have been known in the Kenyan coastal zone at all times, although these symptoms had not been linked with one and the same cause. From this, and on the basis of his own extensive parasitological investigations, Wijers (1974) concludes that filariasis has been for a long time endemic in this region, whereas it does not appear to occur in the remaining parts of Kenya. Further endemic East African foci appear to be located in the Mwanza-Musoma region of Tanzania and the Lango and Teso districts of Uganda (Burkitt, 1951; Spencer, 1962), albeit without confirmation of this suspicion by precise epidemiological investigations as in the case of the coastal region.

Epidemiology: In Kenya Wijers (op. cit, 1974) investigated two epidemiologically distinct forms of *W. bancrofti* filariasis. The urban form is transmitted by *Culex fatigans,* which breeds in latrines and stings chiefly around midnight, and the rural form, which is transmitted by the *Anopheles gambiae,* the vector of malaria, and by *A. funestus*, which breed in reservoirs, pools, and other open waters and sting from before sunrise until sunset. In the Kenyan coastal area infections do not extend beyond an altitude of 400 m., and even in such areas they appear to follow river courses, leaving the level interfluves free.

Epidemiological investigations of filariasis involve certain inherent methodological problems, which render it difficult to make statements on the distribution of an infection among a population based on some isolated instance that has been discovered, such as a proof of microfilaraemia or proof of clinical or serological symptoms.

In 1962, the first comprehensive survey in Kenya was carried out by Nelson, Heisch and Furlong (1962) in the coastal region. Without specified selection for the random sample, 2.6% (Mombasa) – 26.3% (Malindi) of all persons over 15 years of age were found to be infested with microfilariae, with the male sex showing an infection rate about 10 – 15% higher than the female—as in all filariasis surveys. A connection between these conditions and clinical indications such as hydrocele among men or elephantiasis of the extremities among both sexes was not established (Tables 24 and 25). If proof of microfilariae in a population is regarded as indicative of endemic occurrence of *W. bancrofti,* without attaching a quantitative statement to this, it must be noted that the entire Kenyan coastal region constitutes an area of endemic *W. bancrofti.*

Over recent years more detailed investigations have been carried out, mainly by Wijers and his colleagues, which show that the degree of infestation of the coastal strip is significantly higher than had so far been assumed (Division of Vector-borne Diseases, Kenya Filaria Research Unit, Annual Report, Mombasa 1973). Systematic control measures, or mass prophylaxis or therapy, have not so far been applied, save for unspecific mosquito controls in tourist centres and towns. In this context it should, however, be noted that in the urban area *Culex fatigans* is becoming increasingly resistant to insecticides.

A sample survey, covering over 5,000 male over 14 years of age in Kwale and Kilifi District, recently published by Wijers, 1977, Wijers and Kinyanjui, 1977, and Wijers and Kiilu, 1977 revealed that the degree of infestation of the coastal region is significantly higher than had so far been assumed. Hereby microfilaria rate, microfilaria density, sign rate (i.e. rate of hydrocele or elephantiasis formation), resulting in a "filariasis index" corrected for absence of male in the sample were taken into account. Four main foci could be identified: a focus in the south, bordering Tanzania, one west of Mombasa, one just inland from Kilifi town and one focus along the Sabaki River, all of which had a filiarisis index of over 50%. A lower prevalence (below 50% filariasis index) was found in the coastal strip and in the hills north of Mom-

Table 24. W. Bancrofti Microfilaria Rates as Found by Examining Night Blood Films of Persons Aged 15 Years and Over on the Kenya Coast

Locality	Males		Females		Total	
	Number examinde	Percentage positive	Number examined	Percentage positive	Number examined	Percentage positive
Lamu area	272	27.9	367	20.9	639	23.9
Kipini and Witu	182	19.2	111	11.7	293	16.4
Tana river	223	16.1	213	13.1	436	14.7
Malindi and Mambrui	150	20.0	91	14.3	241	17.8
Malindi and Kakoneni	368	27.7	225	24.0	593	26.3
Kilifi area	247	16.2	187	9.1	434	13.1
Mombasa	305	4.2	235	0.4	540	2.6
Mombasa and Mariakani	334	18.2	149	12.1	483	16.3
Mombasa-Gazi	379	10.0	149	9.1	538	9.6
Msambweni-Vanga	299	11.0	80	3.7	379	9.1

Table 25. W. Bancrofti Microfilaria Rates in Relation to Elephantiasis and Hydroceles in Persons Aged 15 Years and Over on the Kenya Coast

Locality	Sex	Number examined	Percentage W. bancrofti	Percentage elephantiasis leg	Percentage hydrocele
Faza	male	89	40.6	16.8	39.3
	female	132	31.1	6.7	—
Ganda and Kakoneni	male	136	28.3	5.8	34.5
	female	104	31.3	1.9	—
Jomvu and Mariakani	male	107	14.4	0.6	19.6
	female	156	14.5	1.3	—
Vanga	male	109	31.6	6.4	21.1
	female	102	33.3	1.9	—

(Tables 24 and 25 from Nelson, G. S., Heisch, R. B. and Furlong, M., *Trans. R. Soc. trop. Med. Hyg.*, 56, 202 – 217, 1962).

basa, i.e. in the areas with the highest rainfall and the highest population density. A still lower filariasis index was found in the sparsely populated area of Lamu District and along the Tana River, although the elephantiasis rate was found to be comparatively high.

b) Onchocerciasis (ICD 125.3): Onchocerciasis was formerly endemic in six well-defined areas of western Kenya. The familiar symptomology of itching skin and "river blindness" made it clear that this disease had occurred for a long period. Systematic investigation of the disease was begun in 1921 and followed by what has remained, until now, its successful eradication. Onchocerciasis in Kenya is one of the few examples of successful extermination of a medically-significant insect vector over a long period.

In Kenya the only vector was *Simulium (lewisellum) neavei.* This species of Simulium was discovered by S. A. Neave in 1911 on the Yala River in the Busia District. Its significance in the transmission of onchocerciasis became evident only twenty years later. Systematic investigations led to the discovery of foci of onchocerciasis in Kenya, situated in Kaimosi, Kakamega, Yala in Kakamega District, in Kodera in Kericho District, and in Riana and Ngoina in Kisii District. *S. l. naevei* was the vector in all these areas, totaling more than 8,000 square kilometres (see Table 26) (Buckley, 1949, 1950).

Extensive investigations along the southern and eastern slopes of Mt. Elgon, in the Cherangani Hills, and on the Mau Escarpment failed to provide evidence of the occurrence of *S. neavei* or the anthropophilic simulium flies. Nonetheless, other species of simulium do exist. Investigations in the area of Mt. Kenya, the Aberdares, and the Niambeni Range also failed to provide proof of *Simulium neavei.* The western slopes of Mt. Elgon near Mbale and one area on the southern slopes at Malakisi on the Kenyan border do, however, still have massive infestation by *S. neavei,* and onchocerciasis is equally common there.

There is a twin species of the *Simulium damnosum* complex in Kenya, which is still morphologically indistinguishable from the anthropophile one, although it evidently takes no part in the transmission of onchocerciasis (Highton, 1974 c). The occurrence of *S. (L.) neavei* in Kenya was restricted to shaded, fast-flowing perennial streams and rivers in hilly and mountainous terrain. The breeding places are dependent on the presence of the fresh-water crab *(Potamonautes niloticus)* (M. Edwards), which prefers sunny places in the water. The larvae of the simulium live in the shells of the crabs in well-aerated water. The adult simulium has a relatively short flying range, so that it always remains within the area offering conditions favourable to its life (Buckley).

The first control programmes were carried out by Garnham and McMahon in the Konder area in 1946 by

Table 26. Distribution of Onchocerciasis

District Locality	Area	vector	Population infection rate	Eye complication rate	Infection rate of mosquitoes	Control program carried out	No fresh infections result among children later born (1966)
Kenya:							
Kakamega							
Kaimosi	5,000 km²	S. neavei	72%	10%	10%	1947/48	"
Kakamega						1954/56	"
Yala							
Kericho							
Kodera	170 km²	S. neavei	49%	10%	10%	1946	"
Kisii							
Riana	42 km²	S. neavei	21%	1.6%	10%	1947	"
Ngoina	3,000 km²	S. neavei	29%	1.8%	10%	1952/53	"

Source: McMahon et al. (1958); Brown (1962).

the application of a DDT emulsion. Over the period 1952 – 54, McMahon arranged for this treatment to be applied to all streams and rivers within an area of 8,500 square kilometres, inhabited by more than a quarter of a million people (McMahon et al., 1958, Table 26). Investigations carried out by Nelson and Grounds in 1958 and by Robert et al. in 1967 showed that no new infections had occurred in the age groups born after the elimination of the vector. However, a reservoir of microfilariae for anthropophile simulium flies, which still possibly survive, remained among the older population. Even now this campaign may still be the most effective one ever to have been carried out for the long-term control of insect-borne diseases.

S. naevei still exists in some areas, especially in the focus near Malakisi, which is linked with the uncontrolled focus on the western slopes of Mt. Elgon. Highton, (op. cit., 1974) has given an urgent warning of the danger of the re-introduction of onchocerciasis as a consequence of systematic irrigation measures and hydroelectric power station projects.

II. Infectious Diseases Usually Transmitted Directly from Man to Man (Contact and Air-borne Diseases)

1. Tuberculosis
(ICD 010)

Definition: "A usually chronic infectious disease caused by *mycobacterium tuberculosis* or, more rarely, *M. bovis,* usually acquired by the inhalation of infectious material and sometimes by ingestion, especially in the case of *M. bovis.* It attacks most commonly the lungs and may spread by haematogenous dissemination to other parts of the body. The bovine form generally affects extrapulmonary organs (intestines, bones, lymph nodes, etc.)". *

* C.I.O.M.S. Communicable Diseases, Provisional International Nomenclature S. Btesh. (Ed.) Geneva, 1973

Tuberculosis is one of the greatest health problems in Kenya, as in all developing countries. The first epidemiological investigation in the field was conducted in the year 1948/49 (Haynes, 1951) during which almost 50,000 tuberculin tests were carried out in 18 localities in Kenya. The second investigation took place ten years in 1958/59 later and was organised by the Ministry of Health and supported by the W. H. O. (Roelsgaard and Nyboe, 1961); on this occasion, a representative sample of 8,500 persons (0.7% sample) was examined on the basis of the population distribution of the census in the year 1957. This recent investigation did not permit unambiguous estimates of regional differences in distribution to be made, since these were only given at the provincial level. It was pointed out that the volume and selection of the samples did not permit reliable regional differentiation (see Fig. 17, back of Map 9) — this had not been the aim of the investigation — but rather the verification of tuberculosis infection in the different age groups. It was found that 3% of the children in the age group below 5 years already had had some contact with tuberculosis and probably still had active tuberculosis at this age. About 13% of the children in the age group 5 – 9 years and more than 20% of the age group 10 – 14 years showed a positive Mantoux reaction. In the age group above 20 years, the male population was predominant, a fact attributable to the greater risk of infection at work outside the family. More than 3% of the age group over 10 years showed changes in their lungs following x-ray detection and were therefore suspected of having tuberculosis.

On the basis of the results it was estimated that in 1963 about 110,000 inhabitants of the 6 million were suffering from tuberculosis, 40% of whom in turn presented a source of infection; out of these about 38,000 were children below the age of 5 years. In the age group above 10 years 0,6% were found to be bacteriologically positive in terms of smears and cultures (Table 27).

Furthermore, the investigation showed the greater risk of infections among children below 5 years of age in those households with infected family members (26%

Table 27. Tuberculosis Survey in Kenya (after Roelsgaard and Nyboe, 1961)

Provinces	Percentage of infected children aged 0–14 years	Percentage of persons with tbc lung pathology	reported 1957/59 per 1,000
Rift Valley	11.5	6.77	0.74
Nyanza	7.5	9.00 *	0.45
Central	13.0	8.50	1.97
Southern	15.1	8.22	0.85
Coast Kwale Kilifi	9.9	3.85	1.42
Total	10.7	7.88	1.10

* Central Nyanza 14.96
Northern Nyanza 7.67
Southern Nyanza 7.93

Table 28. BCG-Vaccination in Kenya 1962, 1966, 1970, and 1973 *

	Total vacc. 0–16 per year	cumulative No. of vacc. 0–16	cumulative coverage 0–16 (%)
1962	20,961	20,961	0.5
1966	723,607	1,868,640	33.5
1970	729,970	4,447,826	61.4
1973	572,608	6,644,537	79.1

* For details see Fig. 9.

positive Mantoux reactions), as compared to 2.4% among children in households without infectious members of the family. In the age group 5 – 9 years the frequency of positive Mantoux reactions of both groups has already risen to 70%, and 12% respectively. The difference is less among older children because of the increasing additional exposure to tuberculosis outside the family.

Although the reported figures, whether furnished by the Annual Return of Diseases from the government hospitals or by the Notification of Infectious Diseases from the district medical officers, depend among other things on the availability of diagnostic facilities and above all the awareness of tuberculosis, it is possible to discern characteristic regional differences. Geomedical analysis of the hospital recording rate for tuberculosis in 50 hospitals within central and western Kenya has shown that the frequency of tuberculosis in hospitals located in cool and dry climatic regions was ten times higher than in hospitals in humid-warm or dry-warm climatic regions (Diesfeld, 1969 a).

The Tuberculosis Control Programme: On the basis of the Ministry of Health's tuberculosis survey carried out with assistance of W.H.O. in 1958 and 1959, an action programme was devised which primarily provided outpatient treatment using thiacetazone/isoniacid and inpatient treatment using streptomycin/PAS/pyracinamid for seriously infected patients. A follow-up of this programme showed a cure rate of 63%, although a primary isoniacid resistance of at least 5.2% must be assumed in Kenya (Kent, 1974).

In the early sixties, the first BCG vaccination programme was inaugurated by the Ministry of Health and supported by the W.H.O. and U.N.I.C.E.F., in the course of which 3.7 million children below the age of 16 were vaccinated during the 8 year period from 1962 – 69. Since 1970, this campaign has been combined with a National Smallpox Eradication Programme. By the end of 1973 a further 2.9 million doses of BCG vaccine had been administered (Fig. 9 and Table 28).

The programme is continuing on the basis of 22 regional sectors and under the direction of the Communicable Disease Control Division of the Ministry of Health. It is assumed that 500,000 – 600,000 annual revaccinations will effectually make up for the reduction in the collective protection of vaccination caused by the later-born population. Since an estimated 15% of newborn babies are seen in ante-natal clinics, 25% of children aged between 1 and 5 in child welfare clinics and nursery schools, and 70% either in school examinations or OPD-attendance at dispensary health centres and hospitals,

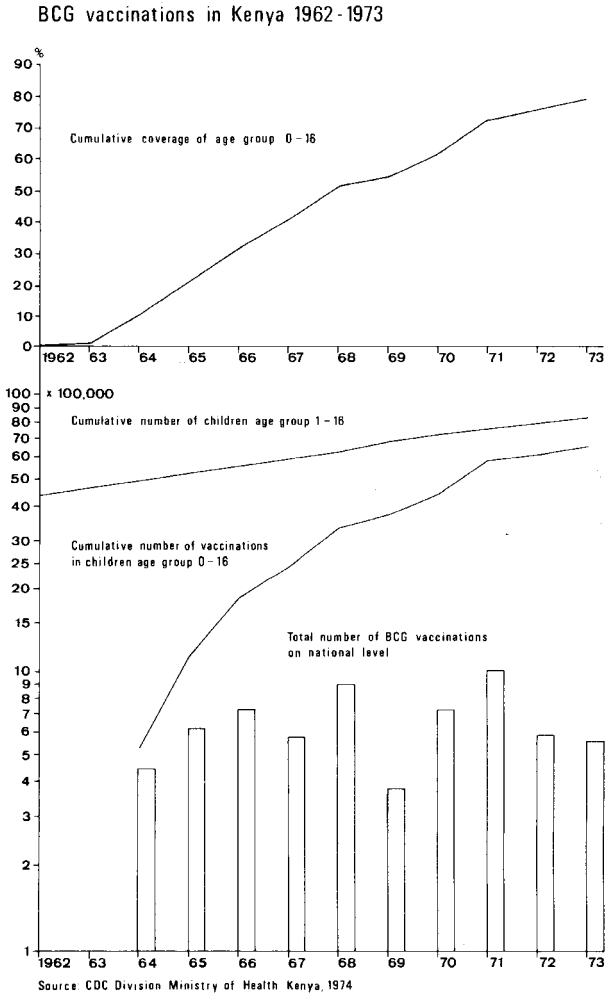

Figure 9. Tuberculosis vaccinations, 1962 – 1973.

where regular vaccination sessions are conducted, it may be assumed that an 80% coverage can be maintained.

It can be assumed that every child born in the coming years will receive BCG vaccination at one of these institutions in the course of time. Zonal supervision and permanent random assessments are carried out by independent teams. At the same time the treatment programmes continue in accordance with the accepted standards. If it is possible to maintain this national programme as planned, it can be assumed that within a generation the problem of tuberculosis will be under control in Kenya.

2. Leprosy
(ICD 030)

Definition: "chronic infective disease caused by *Mycobacterium leprae,* and characterized by a long incubation period and slow onset with vague pains and areas of paraesthesia followed by typical lesions on the skin, mucous membranes, and nerves. Two polar forms are described, namely lepromatous and tuberculoid leprosy; intermediate, borderline and indeterminate forms are also recognised."

Approximately 1% of the East African population suffers from leprosy (Schaller, 1970). At present there is no conclusive evidence which can explain the considerable regional differences of occurrence, which undoubtedly exist. The first large-scale investigations of the occurrence and distribution of leprosy in Kenya were carried out by Innes (1949); the methodological shortcomings contained in that work scarcely allow an interpretation to be made, although they do draw attention to crucial points in western Kenya and on the coast, which were confirmed and more accurately investigated in western Kenya through the more exact sampling methods of J. A. K. Brown (1959) between 1951 and 1959.

Table 29. Frequency of Leprosy — reporting from "Weekly Notifications of Infectious Diseases" (median annual notifications for 1963–1972 per 100,000 1969-Census-population) in descending order

District/Province	Leprosy Notifications per 100,000 population	
Area of high endemicity:		
1. Busia District	95.50	I
2. Kisumu/Siaya District	73.05	≧50/100,000
3. Kwale District	65.36	
4. Kilifi District	23.05	II
5. Bungoma District	17.68	10–49/100,000
Area of medium endemicity:		
6. Kakamega District	6.01	
7. Lamu District	4.54	
8. Kitui District	3.79	
9. Taita-Taveta District	2.70	III
10. Meru District	2.54	1–9/100,000
11. Embu District	2.23	
12. Central Province	1.55	
13. Mombasa	1.21	
Area of low endemicity:		
14. South Nyanza District	0.58	
15. Rift Valley Province	0.49	
16. Machakos District	0.42	
17. Kisii	0.20	IV
18. NE Province	0	<1/100,000
19. Tana River District	0	
20. Kisii District	0	

Fendall and Grounds (1965 b) estimate that three-quarters of all the 35,000 cases of leprosy assumed in Kenya—other estimates put the figure at 60,000–70,000 (Verhagen, 1974)—occur in this region.

However, systematic investigations carried out in other parts of Kenya between 1967 and 1968 show new, and so far unknown, foci in Kitui District, in the south of Embu District, and in eastern Meru District. A revision of the known leprosy areas in the Western Province using the same sampling methods resulted in a prevalence of 1.2 to 1.4% in two locations in the Kakamega District, of 3.2% in Busia, and of 1.4% in South Nyanza (Ziedses des Plantes et al., 1968).

Although the reporting is definitely incomplete, the regional distribution of the frequency of leprosy can, at least its tendency, be estimated from the Weekly Notifications of Infectious Diseases published by the Ministry of Health. From the median number of cases for the period 1962–1972 related to the census population of 1969, foci can be recognised which have been confirmed by various special leprosy investigations (see Table 29).

The median number of hospital leprosy cases reported in the "Annual Return of Diseases" over the period 1963–1972 and related to the estimated population in the catchment area (Census 1969) portrays essentially the same tendency, even if some hospitals in northern Kenya mention more leprosy than appears in the Weekly Notifications. On the other hand, no cases of leprosy are registered by the hospitals at Kwale and Msambweni, although it is well known that leprosy occurs particularly frequently in this part of the Coastal Province (Ministry of Health, Annual Report Coast Province, 1973).

Three clearly discernible grades of endemicity can be distinguished on the basis of information at present available, with a binding statement concerning prevalence only from individual random tests (see Table 29). So far as particular details are concerned the various leprosy foci can be characterised as follows:

a) Foci in western Kenya: Different investigations agree in locating the highest leprosy infection rates in the country (1–3%) in the Kisumu, Busia, and Bungoma Districts. One of the two large hospitals for leprosy patients in Kenya is to be found in Alupe near Busia; contrary to the "Weekly Notifications" and the "Annual Hospital Returns" this focus in the area of the Lake Victoria littoral extends further to south Nyanza (Leiker, 1966; Ziedses, 1968), thus wholly embracing the settlement area of the Luo, a Nilotic tribe which immigrated from the north (Kisimu 93%, Siaya 96%, South Nyanza 89%, according to the 1969 Census). In Busia District the proportion of Luo is smaller, but 31% belong to the

equally Nilotic Iteso tribe. In Bungoma District the Nilotic Iteso and Sabaot compose 15% of the population.

Towards the north, in Bungoma District, and towards the east, in Kakamega District, the frequency of leprosy appears to decrease. In Bungoma, Busia, and Kakamega the Luhya, members of the Bantu group, predominate. In his investigation of the Ugandan and Kenyan "leprosy belt", Brown (1959) suspects the existence of a genetically determined factor promoted by in-breeding which predisposes the geographically and ethnically isolated Nilotics as well as the displayed Luhya (affected by the inmigrating Nilotics) to leprosy, thus perhaps explaining this focal concentration of leprosy. The precise delimitation of leprosy towards the east, an area in which leprosy is evidently almost non-existent, is indeed remarkable.

b) Foci in the Coastal area: In the coastal area recent investigations (Hartmann, 1973) have demonstrated a rather variable frequency. Whereas leprosy does not appear to be a special problem in Lamu District and on the Tana River, in Kilifi and Kwale Districts an infection rate of about 1% of clinically diagnosable leprosy is usual. The differentiated investigations mentioned above show that the Waduruma and other Mijikenda groups as well as the Wakamba, who have moved into this region, and those groups in more isolated areas appear to be particularly affected. Among the non-Bantu groups in the northern coastal area leprosy seems to occur less frequently, indeed to be retreating.

c) Foci in the Taveta area: Among the *Taveta,* too, leprosy appears to be more frequent than is the case with the surrounding Taita (Innes, 1949), a fact clearly reflected in the hospital recording rates of Taveta (59/10,000) as compared to those at Wesu in the Taita Hills (2/10,000). There is no obvious explanation for the rather striking differences.

d) Focal areas in the Central Lowlands of Kenya: Leprosy foci that had long escaped notice were found in the Kitui District, in the south east of Embu District, and in the east of Meru District (Tharaka, Nyambene); these have a frequency rate in excess of 1% (Ziedses et al., 1968). This observation is also reflected in the Notifications and Annual Returns, which stand out clearly against the neighbouring regions.

In the Central Highlands (Central Province) leprosy appears to occur relatively rarely. The various districts of the Rift Valley Province, together with the northern and north eastern districts, report very low figures or nil returns. Nevertheless, more cases of leprosy are registered in the Annual Returns of Diseases from the Lodwar, Marsabit, and Isiolo hospitals than in the District Notifications of Infectious Diseases. It may at least be stated that leprosy evidently does not play as special a role in these areas, as it did in the four foci of leprosy described above.

e) Prevention and eradication: After the Second World War leprosy settlements gradually disappeared. In 1948 Innes introduced chemotherapy. Apart from Alupe, there is only one other hospital for leprosy patients remaining at the present time, located at Tumbe near Msambweni on the Indian Ocean coast. Alupe also houses a leprosy research station under the aegis of the East African Common Services Organization. After 1962 the treatment of leprosy in the form of open treatment was transferred to government hospitals, albeit with disappointing results. It remains to be seen whether the national campaign against tuberculosis will be able to control leprosy with BCG vaccine, which is thought to provide immunization against leprosy as well.

3. Smallpox
(ICD 050)

Smallpox is an acute infectious disease caused by the smallpox virus *variola major* which, owing to its highly contagious nature, gives rise to continuously recurring epidemics among unvaccinated populations.

At the initial stage, transmission results from droplet infection, at the pustular stage, by smear infection from person to person. The virus can survive for long periods in dust.

Until systematic immunization of the population was carried out in 1972, smallpox was endemic in Kenya, although the number of smallpox cases had declined over the years and despite the continuous improvement in the quality of smallpox registration and the thereby increased number of official registration of cases (Fig. 10).

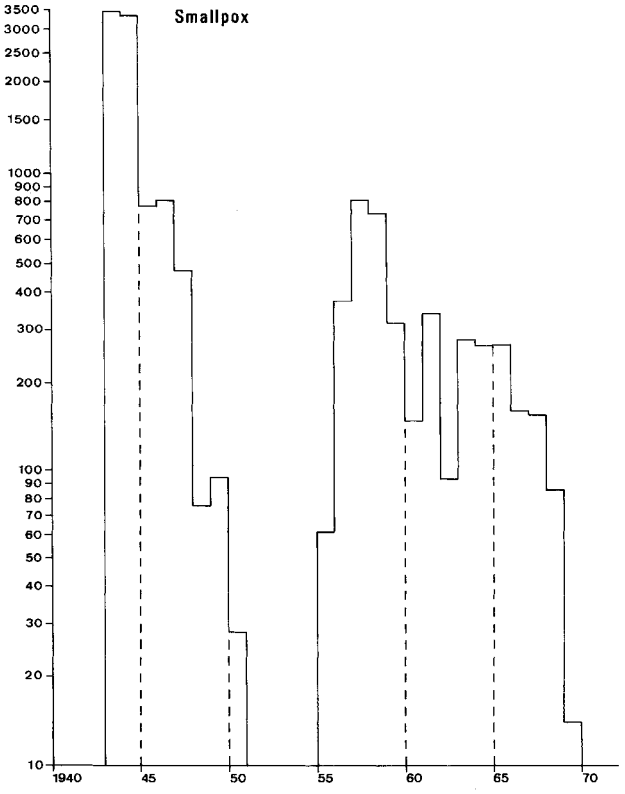

Figure 10. Smallpox, number of annual notifications 1942 – 1972 (Source: W. Koinange Karuga, 1974 and Fendall and Grounds, 1965 b)

Smallpox assumed epidemic proportions during and after both the world wars, during the recession of the thirties, and during the Mau Mau Revolution. In Kenya smallpox had a fatality rate of 1 – 2%, indicative of a relatively non-virulent virus in the main, infection by *variola minor.* Clinical cases occurred almost solely among non-vaccinated persons. Fairly extensive vaccination campaigns and voluntary vaccinations against smallpox were not enough to bring the smouldering endemic state to an end. Small, localized outbreaks effected an immediate improvement in people's readiness to be vaccinated, but this declined as the outbreak subsided.

The distribution of officially registered, and so far as hospitals are concerned, reported cases agrees in showing lower frequency in areas of comprehensive vaccination, covered in the reports of the district medical officers.

Seymour-Price et al. (1960) regarded the variable accessibility to the transport network as an important reason for the variation in the vaccination rate, and, correspondingly the variation in the smallpox frequency. Fendall and Grounds (1965 b) noted, on the other hand, that smallpox occurred more frequently in the settlements of plantation workers and other population groups living close together, although vaccinations could have been carried out quite easily in such places without undue communications difficulties. Nevertheless, smallpox has occurred less frequently in Kenya over recent years than was the case in neighbouring countries. The last cases of imported smallpox in Kenya mainly entered via the nothern borders, whereas over the past twenty years the coast and airport have ceased to provide points of entry.

In the case of reported, suspected or actual smallpox, a mobile unit of the Ministry of Health is now available to investigate the outbreak immediately and to take suitable isolation and vaccination measures. After appropriate planning, a national smallpox vaccination programme was carried out in 1972, in the course of which 13 million vaccinations and re-vaccinations were administered. Nine million of these were carried out by mobile vaccination teams, four million by static units. The programme was combined with a BCG vaccination programme. An independently conducted investigation of the outcome of vaccinations in five provinces showed that at the campaign's closing date an immunization rate in excess of 80% of the population had been achieved, which in the age group 5 – 14 years had, in some areas, been increased to more than 90%. In order to maintain the success of vaccinations it was followed up by a "maintenance phase" in June, 1972, during which one mobile unit for each of the 22 sectors of the country was to repeat the performance within a year (Koinange Karuga, 1974). It can therefore be assumed that under these conditions Kenya will remain free from smallpox infection, even if cases of the disease are imported from neighbouring countries.

4. Cerebrospinal Meningitis
(ICD 036)

Cerebrospinal meningitis (CSM) (meningococcal meningitis) is an acute purulent inflammation of the meninges due to *Neisseria meningitidis,* often occurring epidemically in certain tropical areas and sporadically with small outbreaks all over the world.

Extensive epidemics of meningitis, such as occur in the area of the "meningitis belt south of the Sahara" (Jusatz, 1961), have hitherto not been observed in Kenya. Smaller outbreaks, which affect few people, do occur every year (Fig. 17, back of Map 9), although they do not manifest the essential seasonal peaks, and those affected are mainly children (Fendall and Grounds, 1965 b). As yet epidemiological investigations in more detailed form have not been carried out. *Neisseria meningitidis* plays a minor role in bacteriological specimen investigated at the National Public Health Laboratory, Nairobi, which are sent in from Kenyatta National Hospital and from surrounding hospitals (Say, Itotia and Wamola, 1974).

The Weekly Notifications of Infectious Diseases" for 35 districts, covering the period 1963 – 72 and relating to 100,000 inhabitants of the median year 1967, show that

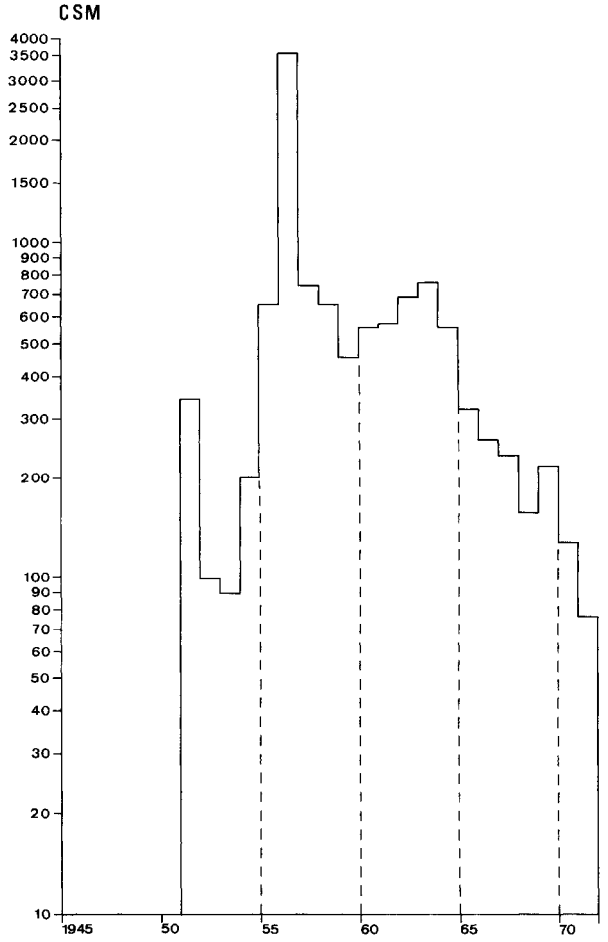

Figure 11. Meningococcal meningitis, number of annual notifications 1950 – 1972 (Source: Fendall and Grounds, 1965 b, Bulletin of Infectious Diseases, Ministry of Health, Nairobi, 1962 – 1972)

the Central Province has the highest annual rate of registrations with 6.69 cases per 100,000 inhabitants; it is followed by the Eastern Rift Valley and Western provinces, each with less than half the number of reported cases. The Coast Province, Nyanza Province, and Nairobi each report about one-tenth of that number of cases. Although these figures cannot be taken as absolute values, they do show the varying distribution, which possibly shows a connection with the higher altitudes of the settlements, in which relatively long sojourns within the dwellings, due to climate, and high settlement densities produce closer contact and with it increased risk of transmission (see Fig. 17, back of Map 9 and Fig. 11).

5. Yaws
(ICD 036)

Yaws is a chronic treponematosis, caused by *Treponema pertenue* which is transmitted by direct contact and, much as in the case of cosmopolitan syphilis *(T. pallidum),* leads to primary lesions of the skin as well as to secondary and tertiary destruction of skin and, above all, bones. In Kenya, as in the rest of Africa, it was once very common. The Annual Reports of the Medical Department reported on it from the twenties onward, with the numbers quoted there increasing at the same rate as treatment possibilities became available. In the period 1921 – 1932, tens of thousands of cases were treated every year, at first with Neosalvarsan and later with bismuth preparations; since the early fifties treatment has been with long-term penicillin (PAM). During the period when penicillin was being introduced, the numbers of those seeking treatment again showed a considerable rise, but since the midfifties their numbers have progressively declined. The reputation which Western medicine already enjoyed at an early stage can in essence be atributed to these successes (Fendall and Grounds, 1965 b; Vogel and Huma, 1974).

It was, however, very soon recognized (Annual Report, Medical Department, Nairobi, 1931) that even minimal improvements in personal and general conditions of hygiene—that is cleanliness—are no less important than chemotherapy in the control of this disease. Through these means, as well as through the specific and general distribution of penicillin through official and even unofficial channels, yaws is to all intents extinct in Kenya today. When checking the figures on yaws reported in the "Annual Return of Diseases" of the hospitals in the field, discussions with physicians showed that the registered cases appear to be relicts of tertiary yaws, and that no new cases had come to their notice for years. It is only among the nomadic herdsmen in northern Kenya that some isolated fresh cases may still be observed (Vanek, 1968, personal communication); as regards hygienic conditions in particular, the herdsmen are to be considered a marginal group.

The assumption that there is a cross-immunity between yaws and veneral treponematosis (syphilis) is contradicted by the fact that the disappearance of yaws has rendered the population more receptive to syphilis (Vogel and Huma, 1974).

6. Trachoma and Other Infections Causing Blindness
(ICD 076)

Scarcely any other disease is as favoured by lack of water and personal hygiene, by ignorance and poverty as trachoma, an infectious keratoconjuntivitis, caused by *Chlamydia trachomatis,* transmitted by contagious infection under poor hygiene conditions, especially in hot and arid areas of Africa and Asia.

Due to inadequate specialist diagnostic conditions, trachoma and its consequences cannot always be clearly distinguished from other chronic eye infections and their aftermaths. It is therefore wise to treat pronouncements on trachoma distribution with a certain scepticism. Medical statistics undoubtedly present a considerable number of eye infections under the heading of trachoma, which are in fact of bacterial or viral origin.

As soon as there is an improvement in conditions of personal hygiene and the possibility of washing the face each day with clean water becomes a reality, the frequency of chronic eye infections promptly decreases. Eye infections are thus closely connected with the availability and usage of water for washing, and are thus not only dependent upon hydro-geographical and climatic factors, but above all on socio-economic and socio-cultural factors. Investigations carried out in Kenya have provided a clear illustration of this situation (Bisley and Burkitt, 1974).

In Kenya, infections of the eye are most widely spread among children, and women are more frequently affected than men. Bisley and Burkitt (op. cit.) suspect that boys escape from the unhygienic domestic milieu at an earlier stage, and once outside it are more likely than girls to have the chance to take a bath. For example, in West Pokot the authors found eye infections among 98% of the population, whereas schoolchildren in the same area at a school near a river only manifested a 30% infection rate. Stones (1939) and Henderson (1968), both in Uganda, estimated that about 15% of the children are infected by trachoma, especially in semi-arid areas; Emiru and Dechet (1969), observed a frequency of 18% among the population in Uganda examined by them, and these same authors attributed 40% of the blindness to trachoma. According to Calcott (1954) 6.3% of 16,000 schoolchildren in Kenya were found to be suffering from trachoma when examined between 1953 and 1955. Trachoma is widespread in Kenya, but it appears to take a relatively mild course so long as it is not aggravated by additional infections. This may explain the fact that only 3.7% of blindness is unequivocally caused by trachoma (see Table 30).

The Opthalmological Survey of the British Empire Society for the Blind and of the Ministry of Health,

Table 30. Causes of Blindness among 1,093 Blind Persons in Kenya, 1953 – 1956 (after Calcott, 1954).

	Cases	%
Senile cataract	477	43.6
Panophthalmitis	183	16.7
Ulceration of the cornea	182	16.6
Optic atrophy	62	5.7
Glaucoma	50	4.6
Trachomatous scarring	40	3.7
Non-senile cataract	25	2.3
Irido-cyclitis	23	2.1
Injury	17	1.6
Onchocerciasis	15	1.4
Retinopathies	13	1.2
Degenerative conditions	6	0.5
	1,093	100

Kenya, conducted during the period 1953 – 1958 (Calcott, 1954 op. cit.), in which 7,747 persons were examined, produced 3,248 (=42%) normal and 4,499 (58%) pathological findings. Of these, 1,677 (i.e. 37% of the pathological total or 21% of the total number of persons examined) had to be attributed to trachoma. Calculations have shown the rate of loss of sight to be 12/1,000 of the population or, relating this to the then total population, the probability of 70,000 blind persons. The main causes were senile cataract (43.6%) and the aftermath of infections (45%) (see Table 30).

This report demonstrates that 80% of the causes of blindness can be prevented and consequently the decisive measures for its control are in the sphere of health education.

The regional distribution of the median annual illnesses classified as trachoma in the Annual Return of Diseases reported by 54 Kenya Government hospitals over the past ten years and calculated as a hospital recording rate for 1963 – 1972 provides (in accordance with the ideas advanced above) more of an indication of the conditions favouring trachoma and eye infections than a picture of trachoma distribution. In this semi-quantitative form, the presentation of this geographical frequency distribution takes on an impressive image. According to it, the greatest frequency occurs in the northerly areas with nomadic herding population. 10 of the 54 hospitals that were investigated (i.e., 18%) are situated north of a line linking Kapenguria—Tambach—Isiolo—Meru—Wajir. The hospitals next in order of precedence (30%) are located exclusively in central Kenya, together with the southern Rift Valley and Wesu (see Table 31).

The hospitals in the coastal area and western Kenya, Nyanza, the Western Province, and the central Rift Valley represent the lower half, with small to non-existent numbers of trachoma cases reported. This unambiguous distribution permits one to draw conclusions concerning conditions of hygiene and other factors favouring or restricting trachoma.

III. Infectious Diseases Usually Transmitted Indirectly from Man to Man by Water and Food (Water- and Food-borne Diseases) and Tetanus

In this chapter the most important water-borne and food-borne diseases are combined, although their actual distribution and significance are relatively little known. From an epidemiological viewpoint, this group includes —apart from the classical bacterial and protozoal enteritic infections such as cholera, typhoid fever, enteritic fever, and other salmonella, shigella and amoebic dysenteries—virus diseases such as poliomyelitis and virus hepatitis. Intestinal anthrax, although it could be included on etiological grounds, will be dealt with in the section on anthropozoonoses. Because of its close association with the conditions of soil, tetanus will be dealt with in this section.

1. Poliomyelitis
(ICD 045)

Generally not much is known about the distribution of poliomyelitis, since no epidemic outbreaks of the para-

Table 31. Hospital Recording Rate (HHR) distribution for trachoma

Number of reported cases of trachoma	Number of hospitals	HRR per 100,000	Hospitals
very frequent rank numbers 7, 8, 9	10 (=18,5%)	200 – 250	Isiolo, Marsabit, Maralal, Meru, Moyale, Kapenguria, Lodwar, Wajir, Mandera, Molo
frequent rank numbers 5, 6	17 (=31,5%)	30 – 170	Kiambu, Kerugoya, Thika, Tambach, Machakos, Embu, Narok, Wesu, Nanyuki, Kajiado, Lokitaung, Londiani, Murang'a, Kabarnet, Nyeri, Kitui, Kapsabet
infrequent rank numbers 1, 2, 3, 4	27 (=50%)	0 – 29	Mombasa, Nakuru, Thomsons Falls, Nairobi, Taveta, Voi, Garissa, Lamu, Kipini, Naivasha, Nandi, Kitale, Kapkatet, Eldoret, Bungoma, Kisii, Malindi, Kisumu, Kilifi, Galola, Kwale, Msambweni, Makindu, Tigoni, Kericho, Kakamega
	54 (=100%)		

lytic form occur. Mainly spread by faecal-oral transmission, this entero-virus infection attacks children in those places having generally low conditions of hygiene and where the majority of such infections escape detection and paralytic cases occur relatively seldom. From an epidemiological point of view this state of high endemicity leads to a good state of immunity in later life. In the tropics antibodies for all three types of polio virus are already found by the age of six in most cases. As the standard of living rises, the exposure to infection at an early age becomes less and paralytic forms of the infection will become more frequent, thereby leading to the classical form of "epidemic poliomyelitis".

Kenya has entered this epidemiological situation in the mid-1950's approximately. Whereas paralytic cases of poliomyelitis had scarcely ever occurred beforehand, an increasingly well-developed 3-year cycle of epidemic poliomyelitis set in from 1954 onwards (see Fig. 12). As a rule the epidemics occurred in September and October. In this manner epidemic peaks were observed in 1954, 1957, and 1960 (Fendall and Lake, 1958, Fendall, 1962; Fendall and Grounds, 1965 b; Kaur and Metselaar, 1967).

The 1960 epidemic clearly demonstrated regional differences (see Fig. 17, back of Map 9), in which Nairobi, the Central Province, and the Rift Valley Province produced the highest number of reported cases, whereas the Western, Northern, and Southern Provinces were less affected (see Fig. 17, back of Map 9). Even if this distribution is indicative of varying medical services and although the Western Province is comparatively better supplied than the Rift Valley Province, it is certainly —based on figures reported by hospitals—an expression of varying standards of development and hygiene within Kenya.

Virological investigations carried out in the period 1965 – 1969 have shown that of 559 poliomyelitis patients whose age was known and in whom polio virus could be isolated, 99% were under the age of six. No patient was older than 20 years.

Of 230 serum samples, which had been collected at different locations in Kenya over the period 1965 – 69 from children who had not had poliomyelitis, more than 80% of the 4 to 5 year olds and more than 90% of the 10 to 11 year olds had already developed antibodies against all three types of polio. On the other hand, among a total of 868 polio patients type 1 was isolated in 84% of all cases, type 2 in 10%, and type 3 in 6% of all cases (Nottay and Metselaar, 1973).

The epidemic predicted for 1963 on the basis of previous experience was prevented by the implementation of a massive campaign of oral vaccination against polio in the spring (Fendall and Grounds, 1965 b). On the basis of the virological and epidemiological investigations of previous years apparently successful methods were developed in campaigns of specific mass vaccinations under the prevailing communications facilities, public health services, finance, and staffing problems (Metselaar et al., 1973).

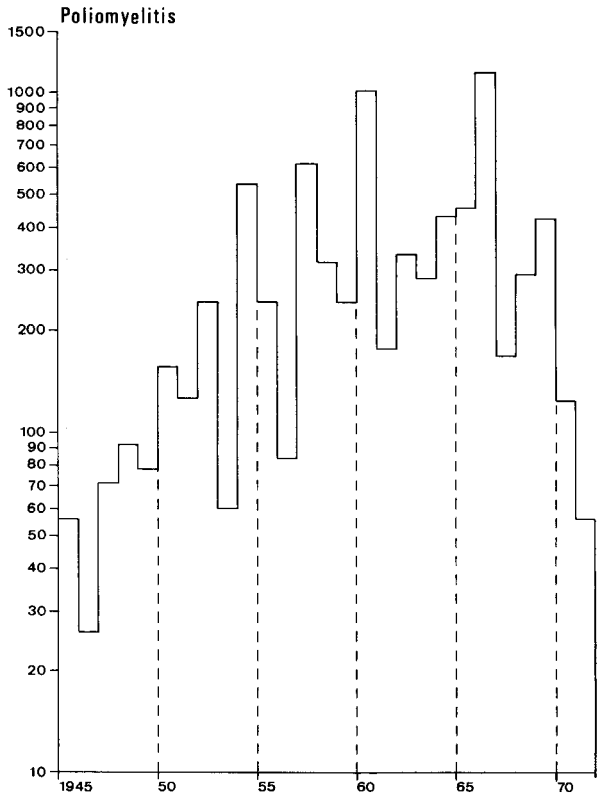

Figure 12. Poliomyelitis, number of annual notifications 1945 – 1972 (Source: Fendall and Grounds, 1965 b, Bulletin of Infectious Diseases, Ministry of Health, Nairobi, 1962 – 1972)

Since epidemiological surveillance suggested that a new epidemic was to be expected in spring, 1972, a pilot project for a national vaccination campaign was carried out on the basis of these earlier experiences. In this campaign it was demonstrated that a vaccination programme could be carried out in every province within two days with the support of lay assistants and all the public services and that such an interval method is to be preferred to long-term, continuous programmes. During the first vaccination campaign 1,736,995 doses of vaccine were administered, and these were followed by a further 1,374,480 doses in the second round, when attention was concentrated on children up to four years, the age group determined most receptive (Koinange, Rogowski and Metselaar, 1973). The authors cited above arrive at the conclusion that under the circumstances at present prevailing in Kenya, an intensive polio vaccination programme of this kind can keep poliomyelitis at an acceptable level in Kenya if carried out at three-year intervals, although rising standards of living and hygiene may well increase the danger of epidemic outbreaks.

2. Viral Hepatitis

(Virus Hepatitis Type A (ICD 070.1)
and Virus Hepatitis Type B (ICD 070.2)

Definition: a general illness caused by a virus, the indicative symptom of which is a hepatitis. Hepatitisvirus A (HVA) is regarded as an agent which is transmitted by

faeces (and presumably also by urine and possibly other secreta, orally as well as parenterally. There is world-wide endemic occurrence, with frequent lesser or greater epidemic outbreaks. Hepatitisvirus B (HVB) is considered the agent of viral hepatitis type B, the transmission of which is as a rule parenteral, although also at times oral. The evidence of the hepatitis antigen B (HB Ag) makes it possible to conduct a sero-epidemiological investigation of the distribution of the virus B hepatitis.

No conclusive investigations on the distribution of hepatitis in Kenya exist (see Fig. 17, back of Map 9). The distribution of the hepatitis B-antigen among healthy people in Kenya has been described by Bagshawe and Cameron (1974). In 3,763 examinations in Mombasa, Nairobi, Nakuru, Nyeri, Rumuruti, and at Kiteta (Machakos), 4.3 – 11.9% carriers of hepatitis B-antigen were found. Whereas the percentage in the lowlands below 1,500 m did not exceed 5%, it rose together with increasing altitude. No explanation of this feature was available, however. The Annual Return of Diseases of the hospitals and the Weekly Notifications of Infectious Diseases of the districts (see Fig. 17, back of Map 9), as far as they cover the central and western areas of Kenya and the south eastern parts of Uganda were first analyzed by Diesfeld in 1969 (Diesfeld, 1969 a) and compared with the figures for poliomyelitis. This demonstrated that both the infections experienced their widest distribution in the highlands and on the densely settled slopes of the Aberdares and of Mt. Kenya. There was also a striking correlation between the figures reported for poliomyelitis and hepatitis, which may in this respect be regarded as diseases indicative of inadequate sanitary conditions under circumstances of advanced socio-economic development and overpopulation.

An analysis of the Weekly Notifications of Infectious Diseases at district level in comparison with the Hospital Recording Rate of 54 government hospitals reveals that the distribution pattern of clinically-diagnosed hepatitis is expressed as the average over the last decade. By far the highest figures occurred in the Coast Province (40.75/100,000) with particularly high figures appearing for the hospitals at Msambweni, Garissa, and Kilifi. The returns for all the other provinces—namely, Central Province, Nairobi City, and North Eastern Province—follow in this rank order at intervals of the power of ten. The Rift Valley, Eastern, Western, and Nyanza provinces make up the lower end of the scale.

3. Typhoid Fever and Other Enteritic Infections

(ICD 002 – 003)

In the clinical and epidemiological sense typhoid fever and other salmonella infections are often grouped together as enteric fever, although a distinction is possible by means of precise analysis, and—above all—by bacteriological and serological control. Apart from this, attention should be given to the numerous shigelloses, entero-viral infections, amoebic dysentery, cholera, food intoxication, and intestinal anthrax, all of which can cause diarrhoeal diseases of varying degrees of seriousness, accompanied by more or less serious general health conditions.

In Kenya a bacteriological and serological identification can only be undertaken in exceptional cases, at the Kenyatta National Hospital for example, where cases were analysed in the 1961 – 1972 period. In this the most varied serotypes of shigellae, salmonellae, and pathogenic *Escherichia coli* were found (Wamola et al., 1974). Definitive statements on this occurrence in Kenya and on the natural history of the infection cannot be based on this, however.

Fendall and Grounds (1965 b) have provided a short review of the state of information concerning the situation in Kenya up to 1963. According to their review, considerations must start out from the assumption that typhoid fever in particular is endemic to a high degree, that the infection of the infant population takes place relatively early, and that massive epidemic outbreaks are thus relatively rare. These are in each case prompted by special local circumstances, resulting in a high number of cases, which in turn produce particularly high figures for

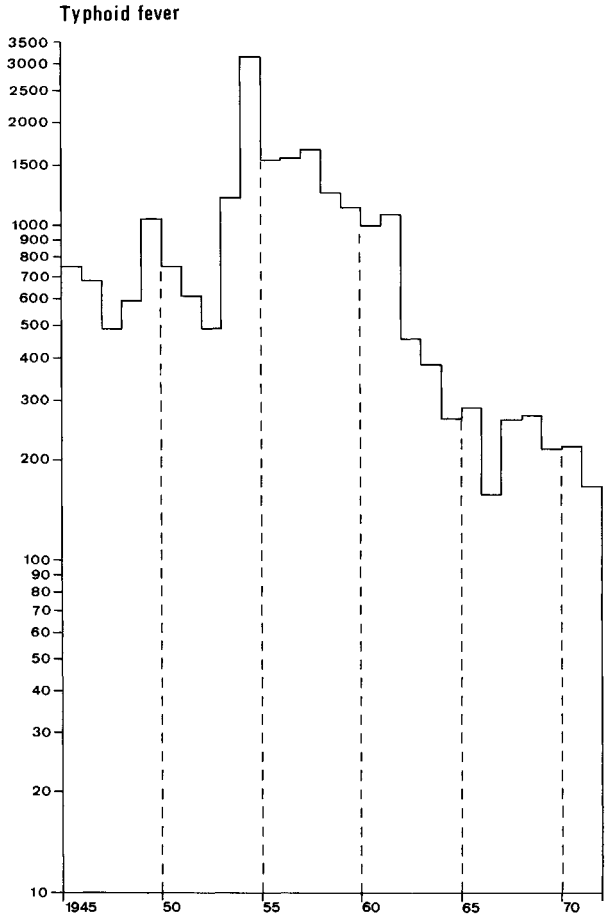

Figure 13. Typhoid fever, number of annual notifications 1945 – 1972 (Source: Fendall and Grounds, 1965 b, Bulletin of Infectious Diseases, Ministry of Health, Nairobi, 1962 – 1972)

the country as a whole in certain years. However, these do not permit the drawing of any conclusions as to the overall situation in the country.

Until about ten years ago the number of reported cases was especially high in the Central Province. It is not clear whether this is to be attributed to the high number of cases or to an efficient recording service. It appears to be established that an overall declining trend can be observed, which is probably justifiably attributed to the gradual improvement in the general conditions of hygiene and of water supplies (see Fig. 13). The consolidation of the population and the already densely settled urban and peri-urban areas with quite certainly inadequate hygienic conditions do not, on the other hand, exclude the possibility of the general latent infection remaining so high that epidemic outbreaks seldom erupt.

Numerous salmonella and shigella infections arise from unhygienic conditions in the food trades catering for the great majority of the population. Judged by experience so far, the tourist industry, by contrast, seems to maintain a considerably higher standard.

Infantile gastroenteritis, especially linked with malnutrition, still represents 60 – 70% of all deaths in these age groups, as well as more than 80% of all deaths caused by gastroenteritis. Together with respiratory illnesses, this group of diseases occupies first place in the mortality statistics. Every year tens of thousands of out-patients and thousands of hospital patients receive treatment in Kenya. Nothing but a general rise in the standard of living and in the standard of health education, personal hygiene, water supply, and sewage disposal, as well as in food hygiene, will alter this. These measures would do more to improve the nation's health than all medical measures taken together.

4. Cholera
(ICD 001)

In the years 1971 – 1973, cholera occurred in Kenya during the course of the VIIIth pandemic for the first time in this century. Presumably it was imported by nomadic herdsmen from the countries bordering in the north and north east and spread southward. Mngola (1974) has provided a detailed review. The main districts to be affected were Mandera, Wajir, and Garissa in January 1971; these were followed by Galole (Hola), Moyale, and Marsabit in summer of 1971. From Garissa, cholera penetrated as far as the northern part of Kitui District (March, 1971). In March, 1973, cholera once again advanced via Lokitaung up to Samburu in the Turkana District.

Typically the individual cases occurred as acute ones. The overall case fatality rate reached 17%. It has not really been established whether it was the protective measures, among others the aid of the Flying Doctor Service and the "Cordon sanitaire", which stopped the dissemination, or whether the disease collapsed on its own. It was mainly of the Cholera El Tor-Inaba strains (Radojcic, 1973).

5. Amoebic Dysentery
(ICD 006)

Although it is generally accepted that in contrast to the Highlands of Ethiopia (Diesfeld, 1965) amoebiasis does not play a special role in Kenya, stool examinations from Nairobi schoolchildren carried out by Wijers, Kinyanjui, and Rijpstra (1972) produced intestinal protozoa in 75.6% of the cases. Besides *E. coli* and *Endolimax nana,* 15.7% also contained *Entamoeba histolytica.* Indications as to pathogenicity were not detected during the course of these investigations.

Table 32. Hospital Recording Rate (HRR) for "amoebic dysentery" 1962 – 1973 (cases per 100,000 inhabitants in the catchment area as a yearly average)

Kerugoya	519	Embu	259	Thomson's Falls	180
Kiambu	362	Bungoma	231	Muranga	140
Kericho	349	Wesu	209	Meru	131
				Marsabit	129

The analysis of the Annual Return of Diseases for the period 1962 – 1973 showed that the 12 hospitals with the most frequent entries under the diagnosis "amoebic dysentery" had their catchment areas located in the uplands.

By contrast, all the others apart from Galole (127) remained markedly below these levels or reported no amoebic dysentery at all.

6. Tetanus
(ICD 037; 670)

Tetanus, a disease which takes an acute course is caused by the toxin of *Clostridium tetani,* the anaerobic spore-forming soil bacillus. Infection usually occurs by way of open wounds with germ- or spore-bearing material like dirt or filth.

In Kenya tetanus occupies the fourth place in the list of recorded causes of death; this is not so much because of its frequency as because of its high lethality (Ministry of Health, Bulletin of Infectious Diseases 1972). The lethality in Kenya fluctuates between 30 – 50% (Muller, 1974).

In Kenya, the enrichment of soil with tetanus spores is of great significance; they are taken up together with the grass and returned with the cattle dung. The contact with man appears to be particularly close in the case of mixed farming.

According to Rigby (1960), the increasing application of dung in agriculture is largely responsible for the 21-fold increase in the number of hospital admissions of tetanus-infected persons which occurred during the period 1933 – 1957. This increase is far in excess of the population increase or in the popularity of Western medicine and hospitals during the same period. Tetanus is particularly widely found in banana plantations and those of mixed banana and coffee cropping where the technique of mulching favours the clostridae (Diesfeld,

1969 a). Fendall and Grounds (1965 a) had already drawn attention to the correlation between agricultural activity in densely settled areas and the frequency of tetanus occurrence.

Baker (1963) noted the connection between the frequency among female patients in the 20 – 34 years age group with their agricultural activity, in the course of which numerous minor lesions provide entry points for the germs.

Tetanus is well known among the population; in the north of Kenya, where the authorities are probably far less often informed of its presence than in other remaining areas of Kenya, men have their lower incisors broken off as a "preventive measure" to ensure the intake of liquid by means of a straw in the event of lock-jaw (Vanek, 1968, personal comm.).

There are a multitude of cultural and ecological factors which determine the frequency and pattern of tetanus distribution.

A special problem occurs in tetanus in new-born babies (neonatal tetanus), when the end of the navel cord is infected as a result of unhygienic care of the navel at the time of delivery. Above all it is the traditional sealing of the navel with a mixture of earth, cow-dung and ash, widely practiced in the coastal region, which is responsible for countless deaths (Fendall and Grounds, 1965 a).

The number of cases recorded in the "Notifications of Infectious Diseases" 1962 – 1973 in relation to the average district population (Census 1969) (Table 33), as well as the Hospital Recording Rate per 100,000 inhabitants in the catchment area for the period 1958 – 1973 derived from the Annual Returns of Diseases from the 54 government hospitals (Table 34) show the distribution of recorded or hospitalised cases of tetanus. The focus is clearly situated in the Coast, the Central provinces, and in Kericho.

Table 33. Tetanus in Kenya (from: Notification of Infectious Diseases 1962 – 1973) *related to 100,000 inhabitants*

Provinces:			
Coast	23.0	Western	2.6
Central	12.0	Rift Valley	1.8
Nyanza	5.8	Nairobi	1.4
Eastern	3.6	N.E.	0

Table 34. Tetanus in Kenya: Mean annual Hospital Recording Rate (1958 – 1973) (per 100,000 population in catchment area)

upper 40 percentile					
Malindi	102	Msambweni	37	Lamu	21
Marsabit	54	Kerugoya	36	Meru	20
Nyeri	53	Kericho	35	Murang'a	20
Kilifi	52	Thika	35	Isiolo	19
Taveta	45	Embu	23	Tambach	17
		Kiambu	21	Galole	16
				Garissa	15
				Makindu	15
				Kisii	14
				Wesu	14

Within the framework of the recently improved ante-natal and child welfare clinics, an attempt is being made to induce a larger number of persons to avail themselves of tetanus prophylaxis by vaccination.

Since only very few women attend ante-natal clinics so far, the problem remains of how to provide pregnant women with an ante-natal tetanus prophylaxis during the third trimenon. The solution to the problem of neonatal tetanus is to be found in combined efforts towards comprehensive ante-natal care, together with health education, tetanus vaccination, and in the training of village midwives. The effectiveness of infant vaccination mostly fails on account of the world-wide and generally familiar high rate of defaulters on the booster immunisation required in tetanus prophylaxis.

IV. Helminthic Infections Associated with Soil, Water, and Food

1. Intestinal Helminths

(With particular regard to Roundworm (*Ascariasis,* ICD 127.0) Hookworm (*Ancylostomiasis,* ICD 126) and Beef Tapeworm (*Taeniasis,* ICD 123.3) infections)

Many intestinal worm infections are distributed throughout the world, some predominantly and others exclusively confined to tropical areas. Furthermore, some of these latter ones occur more or less frequently in all tropical climatic zones, whereas others are known to exist only in certain areas. Although it must be assumed that the majority of the population houses one or other intestinal parasite in the course of his life, its actual significance as regards mortality and morbidity is not quite clear. The intestinal helminths most frequently registered in Kenya are infection with roundworm (ascariasis), hookworm (ancylostomiasis) and beef tapeworm (taeniasis). On account of their differing development cycles, the three species of worms are in some way indicative of different ecological conditions favourable to their distribution (see Fig. 16, back of Map 7). Also fairly distributed are whipworm (*Trichuris trichiura,* ICD 127.3), threadworm (*Enterobius (Oxyuris) vermicularis,* ICD 127.4) and *Strongyloides stercoralis* (ICD 127.2). A not unimportant role is played by *Trichinella spiralis* as a human infection (trichinosis, ICD 124), as well as among carrion-eating carnivores and wild—though not domestic—pigs (Forrester, Nelson and Sander, 1961; Nelson, Guggisberg and Mukundi, 1963; Nelson, Blackie and Mukundi, 1966). With the exception of the analysis of laboratory reports from 43 hospitals compiled over the period 1958 – 1965 (Diesfeld, 1969 b, 1969 c, 1970 b), no thorough epidemiological investigations have taken place.

An analysis of these data in relation to environmental conditions in the hospital catchment areas resulted in increased findings of ascariasis, paralleled by the increase in the population density and the precipitation totals;

population density and precipitation were also closely correlated themselves, a fact that renders interpretation more difficult.

Since in the tropics ascariasis is essentially a dirt and household infection, spreading in the immediate surroundings of dwellings, increasing settlement density and subsequent decline in standards of hygiene are believed to promote the possibilities of distribution of the very resistant ova of *Ascaris lumbricoides.* Hookworm diagnoses were found to decrease with increasing altitude of the hospital catchment areas. A more detailed analysis including the annual climate, defined as "temperature-humidity-milieu" (Diesfeld, 1970 b), in the hospital catchment area demonstrated, by contrast with ascariasis, a regression in the infection frequency at both cool and dry (i.e., higher) and at warm and dry altitudes, whereas warm and humid altitudes did not produce any significant differences in the diagnostic frequency of ancylostomiasis and ascariasis. The parasitological identification shows *Necator americanus* as a rule, whereas *Ancylostoma duodenale* is only found in the coastal region (Foy and Kondi, 1960). Hookworm infection is highest in the coastal region and in the vicinity of Lake Victoria; in these zones hookworm plays a special role as the cause of serious anaemias.

The frequency of taeniasis diagnosis at the hospital laboratories confirmed the expected connection with the cattle population in the hospital catchment areas, together with the simultaneous decrease of the remaining helminths, especially of ancylostomiasis. *Cysticercus bovis,* the larval stage of the beef tapeworm, is found in about one-fifth of all lifestock. *Taenia solium,* the pork tapeworm, causes cysticercosis in pigs, but has so far not been found in man. *Hymenolepis nana,* the dwarf tapeworm, is often detected in children, but seems to be of little significance in Kenya from a clinical point of view (Rees, Mngola, O'Gary and Pamba, 1974). Cestodes, in which man acts as an intermediate host, and in particular the hydatid disease, will be discussed in another chapter.

The regional distribution of the diagnosed frequency of ascaris, ancylostoma, taenia, and schistosoma can be presented in a geomedical analysis based on the cumulative data of the "Annual Laboratory Returns" from the period 1958 – 1973; these refer to 54 hospitals in Kenya with a total of 321 laboratory/years, an average per hospital of 6.4 years, and over 1.1 million stool examinations and half a million urine tests (see Map 8). As already demonstrated in an earlier analysis of this kind (Diesfeld, 1969 a), this extended data "collective" permits collation of the individual places of investigation into groups having similar distribution patterns of helminths and similar geographical, ecological, and epidemiological conditions (Table 35).

Typical patterns of helminth distribution can be observed as indicative of these conditions, if due care is taken in the interpretation (Table XI, Annex).

The following 9 types can be distinguished:

1. *East Coast Type:* characterised by the dominance of hookworm and *Schistosoma haematobium* frequencies in diagnosis and the virtual absence of *S. mansoni* and taeniasis. Although it is located on the coast, the urban Lamu does not fit into the picture, partly because of the high proportion of *ascaris,* the almost complete lack of ancylostomiasis, and partly because the helminth pattern classes it together with Nairobi in an area of higher altitude and denser settlement. Galole, situated on the middle course of the Tana River and characterised by intensive irrigation schemes, must, on the other hand, be included in the coastal region.

2. *Kamba Type:* The Kamba type in Machakos and Kitui is characterised by the occurrence of both the agents of schistosomiasis: *Schistosoma mansoni* and *S. haematobium,* as well as a large proportion of taenia and a small proportion of *ancylostoma* by comparison with the frequency of *ascaris* diagnosis.

3. *Aberdare Type:* The Aberdare type represented by hospitals from the south eastern and eastern outliers of the Aberdares to the irrigation areas of the Mwea Plains, as well as the ecologically and agro-geographically similar areas around Voi, Taveta, and Makindu, is characterised by a roughly equally high proportion of *ascaris* and *ancylostoma* findings, together with a not unimportant proportion of *S. mansoni. S. haematobium,* however, occurs only on a small scale in Makindu and Voi, but to a considerable degree in Taveta.

Table 35. Relative frequency of Helminth-Laboratory findings from 54 "Annual Laboratory Returns" of Kenya Hospital Laboratories (1958 – 1973), grouped in 9 geo-ecological types

Geoecological Type	Ascaris	Ancylost.	Taenia	S. mansoni	S. haematob. *
East Coast Type	31.2	65.8	2.2	0.5	28.10
Kamba Type	35.5	11.3	10.5	42.5	10.83
Aberdare Type	44.1	32.6	14.5	7.9	5.97
Nyeri-Mt. Kenya Type	72.4	14.3	11.4	1.7	3.38
Narok-N.E. Type	37.9	12.7	42.5	6.9	0.51
Rift-Valley Type	37.7	14.2	47.6	0.5	0.36
Kisii Type	51.5	12.2	27.2	2.5	1.67
Nyanza Type	47.9	26.9	20.3	14.6	7.75
W. Province Type	27.1	51.1	18.4	0.3	0.32

* S. haematobium: =% pos of urine examination

4. *Nyeri-Mt. Kenya Type:* This type is characterised by a particularly large proportion of ascaris diagnosis and a very small proportion of *ancylostoma* diagnosis. Those hospitals situated at the middle and higher settlement altitudes around Mt. Kenya and those in the northern Aberdares present a striking homogeneity in their helminth pattern as they do in the ecological conditions of their catchment areas. The coastal town of Lamu and the hospital at Wesu, which is situated on a mountain-massif, can also be classified with this type. Both these hospitals, together with those of Embu and Naivasha, still manifest a high proportion of *S. haematobium* diagnoses; Naivasha's high proportion is striking, but it is not substantiated in any other way. The high number of findings in Wesu, Lamu, and Embu are the outcome of the adjacent endemic areas of *S. haematobium.*

5. *Narok-North-eastern Type* and

6. *Rift Valley Type:* As with those of northern Kenya, the hospitals of the northern, central, and southern Rift Valley show an absolute preponderance of findings of *taenia* and, by comparison with the much reduced findings of *ancylostoma,* a large proportion of *ascaris* diagnoses. The difference between both types consists of a small proportion of *S. mansoni* still to be observed in the southern Rift Valley and in north eastern Kenya, which occur only very sporadically in the remainder of the Rift Valley in northern Kenya, and never autochthonous. They result from importations by patients from outside. Both of these areas are characterised by extensive semi-nomadic and nomadic animal husbandry and large-scale farming, and by no more than marginal subsistence agriculture in favourable localities.

7. *Kisii Type:* Helminthologically this type is characterised by a 3 : 2 : 1 relationship of *ascaris : taenia : hookworm,* with findings of *S. mansoni* and *S. haematobium* occurring only sporadically. This rather homogeneous pattern of helminth distribution applies to the hospitals at the higher altitudes of Kenya's western highlands, where agriculture tends to be mixed. A similar pattern can be observed at Nakuru, which has a very large catchment area, and, interestingly, at Garissa.

8. *Nyanza Type:* Kisumu and Homa Bay, situated on the shores of Lake Victoria, are characterised by a proportion of *ascaris* findings among the intestinal helminths on the whole twice as large as those of hookworm and with only a small proportion of *taenia.* The relatively significant proportion of *S. mansoni* and *S. haematobium,* both of which are characteristic of the coast and the coastal hinterland of Lake Victoria, is conspicuous.

9. *Western Province Type:* In a manner similar to that of the East Coast, hookworm predominates absolutely over *ascaris,* with only relatively small numbers of *taenia* and an almost total absence of *schistosoma* findings.

2. Schistosomiasis
(ICD 120)

Schistosomiasis is a chronic endemic disease caused by a trematode of the genus *Schistosoma.* The intermediate host is a snail and the definitive host is men or animals. The free-swimming cercariae are discharged by the snail, pierce the skin of men, and reach the portal venous system where they develop into adult worms which migrate to the smaller veins of the bladder in the case of a *Schistosoma haematobium* infection or to the mesenteric veins in the cases of a *Schistosoma mansoni* or *Schistosoma japonicum* infection. In these sites the female deposits eggs that are excreted in the urine or stools. It is mainly the eggs which are responsible for the clinical features of the disease.

a) *The distribution of schistosomiasis in Kenya:* As everywhere in Africa, so in Kenya schistosomiasis occurs in the form of foci (see Map 9). Areas highly infested with one or both species of *Schistosoma mansoni* or *Schistosoma haematobium* alternate with others free of schistosomiasis. Besides the ecology of the transmitting snails, which essentially determines the distribution, it is the increase in irrigated agriculture of different kinds and the close contact between man and water which determines the pattern of distribution.

Apart from increased information on the distribution, which seems to indicate an increase in the problem, the increase in the number of small- and large-scale irrigation schemes does result in an authentic increase in the number of infected persons in these areas. At the present time control measures can be carried out only in large-scale irrigation schemes which are centrally managed and centrally supplied with water. The great majority of individual contact possibilities during the parasite's final development cycle remain largely unavoidable at the level of the smallholding, as long as the appropriate health education measures are not carried out and made effective. Schistosomiasis is one of those parasitic mass infections in the tropical developing countries the long-term control of which depends more on the improvement of the general conditions of living, hygiene, water, and sewage disposal and an appropriate health education and irrigation planning than on the application of available chemo-therapeutical means for the elimination of agents in man and the extermination of the molluscs acting as intermediate hosts.

Scarcely any agricultural irrigation scheme in an area of potential transmission has succeeded in avoiding the infection of the population by schistosomiasis through appropriate planning or by interruption of transmission in an already-infected population. However, one such model is to be found in Kenya in the Mwea Tebere Irrigation Scheme, when after 15 years' duration and progressive infection schistosomiasis was finally brought under control. Not only knowledge, but also the implementation of adequate measures resulting from the knowledge of the ecology of the final and intermediate

hosts are required if this chronic disease is to be exterminated.

b) *The ecology of the intermediate hosts in Kenya*

aa) *Intermediate hosts of S. haematobium* (after Highton, 1974 b):

The principal intermediate hosts of *S. haematobium* in Kenya are snails of the *Bulinus* genus and the subgenus *Physopsis,* which belong to the *Bulinus africanus* group. They occur extensively at altitudes below 1,800 m above sea-level. Above this height the water temperatures are too low for the optimal breeding conditions of *Bulinus.* Its habitat is generally to be found in perennial as well as seasonal streams, especially in dammed waters, and also in canals, furrows, and depressions containing water, reservoirs, drainage and irrigation ditches etc. The snails can survive 5 to 8 months of dehydration during the summer months ("Estivation" according to Webbe and Msangi, 1958).

B. (P.) africanus occurs from the coastal area up to altitudes of 1,800 m; at the higher altitudes it is chiefly found in Kitui, Machakos, Kiambu, and Murang'a districts. *B. (P.) globosus* is the chief vector in the coastal plains and in the valley of the Tana River. It rarely occurs above 1,250 m. *B. (P.) nasutus* occurs sporadically in isolated foci. *B. (P.) nasutus productus* is mainly responsible for the transmission of *Schistosoma haematobium* in the Nyanza and Western Provinces.

bb) *Intermediate hosts of S. mansoni* (after Highton, 1974 b): All species of the genus *Biomphalaria* are receptive to infections with miracidia of *S. mansoni,* and are thus also potential vectors. *Biomphalaria pfeifferi* and *B. sudanica* occur in a wide range of water accumulations —puddles, reservoirs, dams, ditches, canals, drainage ditches and even concrete-lined reservoirs—but seldom in swamps. *B. sudanica* occurs chiefly on the shores of lakes and swamps. It is of interest that species of *Biomphalaria* have never been found in the coastal region but only over 300 m above sea-level (Teesdale, 1954, 1962; Teesdale and Nelson, 1958). As a result of similar observations in other regions of East and South Africa, Sturrock (1965) tends to believe that water temperatures in the coastal hinterland of the Indian Ocean are too high for optimal living conditions of *Biomphalaria.* The absence of *Biomphalaria* is confirmed by the absence of autochthonous *S. mansoni* infections in these zones.

c) *Regional distribution of the frequency of schistosomiasis in Kenya:* Apart from investigations in the organised irrigation schemes, little information was available on the real distribution of schistosomiasis among the population until a few years ago. In the meantime it has been estimated that about 1 million inhabitants of Kenya are infected by these parasites. The hospital laboratory reports were consulted in order to gain a general view of the whole country. However, it proved impossible to draw any far-reaching conclusions from this concerning the actual state of infection. A summary and analysis of Annual Laboratory Returns from 54 hospital laboratories over the period 1958 – 1973 showed 1 million stool examinations and almost half a million urine tests for 321 laboratory/years (see Table XII, Annex). A regional distribution of the frequency of *S. mansoni*—positive stool tests—and *S. haematobium*—positive urine tests—clearly shows the previously known distribution pattern of the transmitting snails as well as the probability of autochthonous transmission and transmission-free areas (see Map 8). Together with the investigations recently carried out by the Parasitological Department of the Division of Vector-borne Diseases of the Ministry of Health and references in the literature it results in the following picture:

1. *East Coast and Tana River Valley:* On the coastal plain and in the lower and middle Tana River valley *S. haematobium* is the only form occurring. Stool examinations at Shimba Hill (Kwale District) produced egg excretion in 22.4% of all urine tests; in Kilifi this rose to 67.5% of all cases (DVBD Coast Province, Annual Report, 1972). In the Hola irrigation schemes (Galole on the Tana River), which comprise some 800 ha. of irrigated land, at present mechanical snail control is carried out, albeit so far without any recognisable reduction in the infection rate among the population. Even prior to its implementation it was possible to note a decrease in infection of the population proportional to the distance from the river (Teesdale, personal communication cited by Highton, 1974 b, op. cit.).

45 km north of Hola in Bura a further irrigation scheme is planned which will assuredly present an important agricultural reserve but will also embrace the problem of bilharzia from the very outset.

S. mansoni is practically non-existent in the coastal region. The same applies to those species of *Biomphalaria* acting as intermediate hosts.

This picture is reflected in the Annual Laboratory Returns of 1958 – 1973 (see Table 36 and 37). Between 14 – 45% of all urine tests are positive, whereas the stool tests are practically free from *S. mansoni.*

2. *Taveta:* There is a further important focus of *S. haematobium,* mentioned by Fendall and Grounds (1965 b), in the sisal plantations of Taveta on the border

Table 36. Stanine transformation of stool tests Annual Laboratory Returns 1958 – 1972 for S. mansoni

<1.0%	1 – 2%	2 – 3%	3 – 9%	>9%
	Mandera	Gatundo	Kimbimbi	Kangundo
	Kiambu	Thika	Kitui	
	Kisii Nandi	Taveta	Machakos	
	Kajiado	Makindu		
	Voi, Wajir	Londiani		
	Homabay			
	Kisumu	12%	7%	4%
60%	17%		23%	=100%

Table 37. Stanine transformation of urine tests Annual Laboratory Returns 1958 – 1972 for S. haematobium *

<0.5%	0.5 – 3%	3 – 10%	10 – 30%	30 – 45%
Maralal, Kapsabet	Busia, Nakuru, Kapsabet	Voi	Kilifi	Kinango
Moyale, Tambach	Th. Falls, Machakos	Kisumu	Mombasa	Kipini
Murang'a Kimbimbi	Kericho, Kisii, Thika	Mandera	Malindi	Galole
Kianyaga, Kakamega	Wajir, Embu, Makindu	Nandi	Msambweni	Taveta
Bungoma, Nanyuki	Londiani	Wesu	Kitui	
Kitale, Narok, Eldoret		Kangundo	Garissa	
Nyeri, Alupe, Kabarnet		Lamu	Homa Bay	
Kiambu, Kajiado			12%	7%
Kerugoya		17%		19
36%	20%		36%	

* The presence of *S. haematobium* in the urine sediment is determined by a concentration procedure. Moreover, *S. haematobium* has a significantly higher egg production, which results in the proof of *S. haematobium* being much more sensitive, comparatively speaking, than that of *S. mansoni* in a simple stool smear. In both cases, however, analysing the laboratory data the stanine transformation of frequencies emphasises the most important endemic areas.

with Tanzania to the east of Mt. Kilimanjaro. The Annual Laboratory Returns show a positive diagnosis in 45% of all urine tests. Although the proportion of *S. mansoni* amounts to only 8% of the helminth-positive stool tests, a much greater infection rate must be assumed, since the appropriate intermediate host occurs prolifically in the 4,000 ha. irrigation area (Chowdhury, 1975). The contiguous districts of Wesu and Voi show only a relatively small proportion of schistosome findings in the case of both *S. mansoni* and *S. haematobium.*

3. *Kamba-Kitui:* A large endemic area predominantly of *S. mansoni* is to be found in the hill country of Machakos District (Machakos, Kangundo, Makindu). By comparison with neighbouring Kitui, *S. haematobium* is relatively sparse. An examination of 7,000 schoolchildren in Machakos township over the period 1961 – 64 established an infection rate of 17% (Teesdale, 1966). Examinations of 2,900 schoolchildren undertaken in six divisions of the district by Mutinga and Ngoka (1971) demonstrated an average infection rate of 29.5% (see Table XIII annex) (Chowdhury, 1975, op. cit.).

3 a) *The adjacent highland around Kitui* constitutes an isolated climato-ecological focus in which examinations of more than 5,000 schoolchildren at 27 schools established the existence of a 17.5% infection rate. The frequency fluctuated between 2.5 – 26.5% (Annual Report DVBD 1972 – 1974). The Annual Laboratory Returns from the Kitui District Hospital reveal a mean frequency of 4.6% of *S. mansoni* diagnosis in stool tests, and a mean frequency around 32.5% of *S. haematobium* in urine tests.

4. *Aberdare and Mwea Tebere Irrigation Scheme:* The oldest and so far the largest irrigation scheme is located in the Mwea Plains in the upper Tana river basin at the foot of the Aberdares and Mt. Kenya. Since 1956 rice has been grown on irrigated land, which now covers an area of 5,500 ha. At present there are more than 3,000 formerly landless settlers living here, a total of about 30,000 persons spread over 34 settlements. Teesdale (1961) noted as recently as 1961 that schistosomiasis did not occur in this region. Nevertheless, as a result of increasing irrigation agriculture, schistosomiasis spread rapidly during the ensuing fifteen years. The settlers from the surrounding areas of Kiambu, Murang'a, Nyeri, Embu and Kirinyaga districts, where the problem of schistosomiasis was not known at this time, evidently brought in *S. mansoni,* which spread rapidly in the irrigation scheme.

The waters joining the irrigation network from the Nyamindi and Thiba rivers brought the snails. While the massive use of molluscicides since 1967 has succeeded in reducing the infestation by *S. mansoni* within the area of the Mwea Tebere Irrigation Scheme—at the Kimbimbi and Kombueni schools to 8.2 and 5% respectively—infection among children in the surrounding divisions remains between 30 – 60%. In Murang'a and Kiambu districts, too, and particularly at lower altitudes the infection rate in schools fluctuates between 6.4 – 38.2%; in this the Gatundu, Kiambaa, and Kijabe divisions stood out especially as foci. The Annual Laboratory Returns from the area's hospitals also reflect this situation.

5. Another endemic schistosomiasis area is the *Nyanza Region* on Lake Victoria. While the population living in Tanzania along the shores of Lake Victoria suffers considerably from schistosomiasis, especially in the area around Mwanza, infection by *S. haematobium,* as well as by *S. mansoni,* is much reduced in spite of the occurrence of transmitting snails. Only on the islands of Mfangano (Wijers and Munanga, 1971) and Rusinga (Pamba, 1974) was *S. mansoni* found—in 46% and 30 – 60%, respectively, of the schoolchildren examined. On Mfangano *Biomphalaria* occurs more in the interior, on Rusinga, more on the coast of the island. *S. haematobium* does not appear to be much in evidence. On the mainland *S. mansoni* prevails among the fishermen along the coast, whereas *S. haematobium* predominates among the farmers in the coastal hinterland. The Annual Labo-

ratory Returns for Kisumu and Homa Bay report *S. haematobium* in 3.85% and 1.7% resp. of all urine tests. In both laboratories *S. mansoni* is reported in 14.6% of stool samples.

The investigations of Kinoti (1971 a, b) conducted among 1,700 schoolchildren on the Kano Plains produced evidence of only 4% *S. haematobium:* this low grade of infection is correlated with the absence of *Bulinus africanus* and *B. nasutus,* the main intermediate hosts on the "black cotton soil" of this area.

The occurrence of *B. globosus* as isolated foci only in the Kano Plains is evidently responsible for the small degree of infection. The importation and establishment of transmitting snails in the train of large-scale, planned irrigation measures, and with it the massive occurrence of *S. haematobium,* can only be avoided by appropriate planning. Outside the Kano Plains *S. haematobium* is transmitted by *B. globosus, B. P. forskali,* and *B. P. africanus* (Brown, 1975).

An extension of the irrigation installations into the northern and eastern fringe areas of the Kano Plains, offering a potential of 14,000 ha., would open up contact to *Biomphalaria pfeifferi*—the highly potent intermediate hosts in that area, which multiply particularly successfully in irrigation installations (Brown, 1975). Periodic flooding and waterfowl present further possibilities for the spread of the snails.

Schistosomiasis is no problem in the *Ahero Pilot Scheme* which comprises an area of 880 ha. lying 22 km east of Kisumu and under control since 1968. It remains to be seen how far this will apply to more extensive areas.

The Yala Swamps, in the Busia and Siaya Districts of western Kenya, of which 6,000 ha. have been reclaimed with the assistance of the United Nations Development Programme (UNDP), is considered suitable for irrigation in the distant future. A 200 ha. pilot project was established at Bunyala. Schistosomiasis, both *S. mansoni* and *S. haematobium,* is already endemic in this area, where people have continuous contact with water. *B. sudanica* is confined to the lakeshore and edges of the swamps, whereas *B. pfeifferi* is common in the irrigation scheme (Chowdhury, 1975). Control measures since 1970/71 have reduced snail breeding.

The only irrigation scheme in which schistosomiasis has so far not been reported is at Perkerra, although the adjacent Lake Baringo harbours *B. sudanica.*

6. In the *Western Province* (Kimilili near Kakamega) the DVBD recently discovered another focus of *S. mansoni* and *S. haematobium,* in contradiction to earlier assumptions, a higher rate of infection is also to be expected here. At altitudes above 1,800 m schistosomiasis does not occur as an autochthonous infection. Autochthonous occurrences of schistosomiasis need not be anticipated either in the arid and semi-arid areas because of the absence of perennial streams, as long as no artificial irrigation is introduced which brings in more stagnant or running water than is the case in irrigation at intervals, for example, at Lake Baringo or in Galole.

A recent investigation by Brown (1975) was the first in Kenya to draw attention to the well-known association between haematobium infection and cancer of the bladder. In contrast to the white population in areas free from bilharziasis, the biopsy material from 76 cases presented peaks in two age groups: namely in the 4th and the 7th decades. Carcinomas of the bladder unambiguously, histologically associated with bilharziasis of the bladder were found in 39% of the cases. The mean age of these patients was about 45 years, that of the remaining patients 60.7 years. The regional distribution of the origin of these patients coincided with the areas in which *S. haematobium* occurs, whereas the occurrence of the remaining carcinomas of the bladder does not show such an indicative frequency.

This geomedical presentation of the problem of schistosomiasis clearly demonstrates the extraordinary problem in Kenya. On the one hand, marked scarcity of land inevitably demands large irrigation schemes, whereas on the other, the indisputably joint problem of the spread of schistosomiasis—that is, its control simultaneously with the development of the scheme—constitutes a considerable financial burden. Even if Highton and Chowdhury (1974) were able to show that snail control programmes so far conducted in the five existing irrigation schemes were relatively reasonable in expenditure in the context of overall economic development, they constitute a considerable, if not an intolerable, financial burden.

V. Anthropozoonoses

The term anthropozoonoses is applied to infectious diseases the agents of which are pathogenic to man and animal alike. Applying the term in a somewhat narrower sense, it refers particularly to all those infections of domestic animals which are of special importance to man as diseases—like anthrax, brucellosis, and echinococcosis (hydatid disease). In a broader sense, even leptospirosis and plague ought to be counted among the anthropozoonoses, but the latter will be discussed in the section of vector-borne diseases.

In Kenya relatively little information is available on the four most important anthropozoonoses mentioned above. It must be assumed that they are of greater significance than is presently accepted.

1. Anthrax
(ICD 022)

Anthrax is an infection of cattle and game by the spore-forming *Bacillus anthracis,* which may under certain circumstances also occur in man as a highly acute, febrile, septicaemic, and often lethal disease. A cutaneous form is transmitted by skin contact with infected animal material, chiefly hair, wool, skins, excreta, as well as with

soil and dirt contaminated by them. An intestinal form results from the consumption of infected meat. A pulmonary form in man is transmitted by inhalation of contaminated dust chiefly in those branches of industry concerned with the processing of animal products.

Anthrax seems to occur sporadically among Kenyan livestock, according to observations at abattoirs under the supervision of the Veterinary Department. Two-thirds of the cases originate from the Rift Valley Province, chiefly from the Kipsigis, Narok, Kajiado, and Sirikwa districts; most of the remaining cases come from the Central Province (Annual Report Veterinary Department, 1962 and 1963).

The forms observed among men are predominantly intestinal anthrax as acute diarrhoea. Mass infections following consumption of meat from collapsed or moribund cattle occur frequently and are particularly striking features in connection with local traditional celebrations (Fendall and Ground, 1965 a). As in most cases the diagnosis is only a clinical one and lacks bacteriological confirmation, it is not always possible to distinguish, whether as such it was an anthrax infection or not. Figures taken from the hospital reports agree with the Notifications of Infectious Diseases (see Fig. 14 and Fig. 17, back of Map 9) on the regional distribution of anthrax among domestic animals as indicated by the Veterinary Department. The long-term averages of the "Weekly Notifications of Diseases" present the highest figures reported per 100,000 inhabitant for the Rift Valley Province, which is followed by the Central, Eastern, Coast, and Nyanza provinces. The remaining provinces either report no cases or only isolated anthrax occurrences in their long-term averages. The same applies to 77% of the 54 government hospitals: only 14 of them register more than 10 cases per 100,000 inhabitants from within their catchment areas as a long-term average and these are almost exclusively located in the provinces mentioned above.

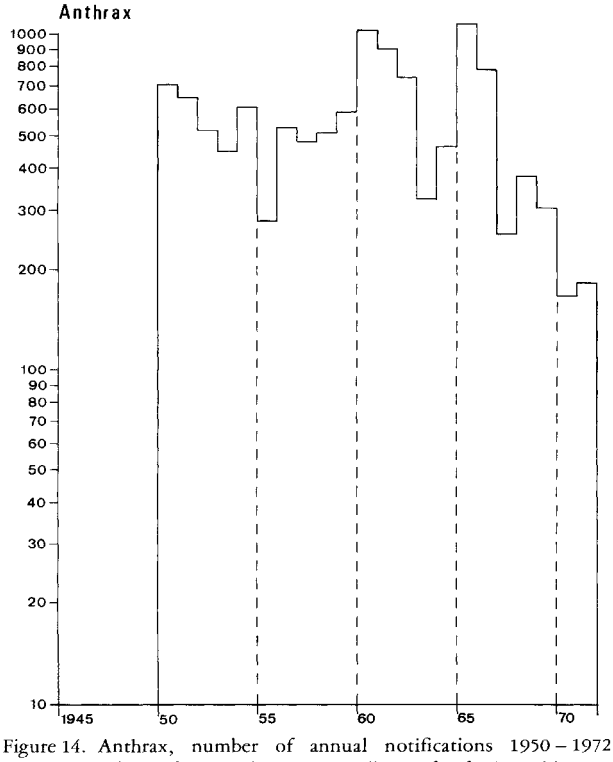

Figure 14. Anthrax, number of annual notifications 1950 – 1972 (Source: Fendall and Grounds, 1965 a, Bulletin of Infectious Diseases, Ministry of Health, Nairobi, 1962 – 1972)

2. Brucellosis

(Undulant fever, ICD 023)

In East Africa brucellosis is an infection of cattle involving *Brucella abortus* and of goats, and sheep involving *Brucella melitensis.* The germs are transmitted through milk and inadequately sterilised milk products, as well as meat, blood, placenta, still-born foetuses and the excreta of infected animals. Its distribution among livestock is not even approximately known. As an infection it constitutes a considerable economic loss, since it leads to miscarriage on an epidemic-like scale (see Fig. 17, back of Map 9). The infection rate of man was found to be particularly high where *B. melitensis* infection of goats and sheep was predominant. The hygienic standard of cattle, goats, and sheep under semi-nomadic or smallholder conditions seems to enhance the spread of brucellosis among animals and between animals and man more than under nomadic conditions (Vanek, 1976). Serological examinations of cattle, goats, and sheep over the period 1969 – 1972 showed a positive agglutination test among 1.5 to 7.5% of the animals tested, with no discernible connection between the type of animal husbandry and the degree of infection.

A remarkably high percentage of 10.6 – 11.1% was found among cattle of the Coastal Province and in Narok. No positive titer was found in the Nyanza Province (Oomen and Wegener, Oomen, 1976 and Table 38).

Although the diagnostic possibilities are limited, the Weekly Notifications of Infectious Diseases as well as the Hospital Reporting Rate of the Annual Return of

Table 38. Serological Survey among cattle, goats and sheep in Brucellosis (1969 – 1972)

Area	Cattle		Goats and Sheep	
	No.	% positive	No.	% positive
Turkana	201	1.5		
Marsabit			187	1.6
Samburu, Isiolo	557	5.2		
North Eastern Province	1,472	7.5		
Baringo			326	3.4
Laikipia	487	2.1		
Narok	667	11.1	807	1.4
Kajiado	557	3.9		
Machakos, Kitui	46	4.3		
Nyanza	185	0		
Kiambu	208	2.4		
Coast Province	724	10.6		

Source: Oomen and Wegener, 1974

Diseases allow a largely coincidental tendency to be observed in an average of ten years (see Fig. 17, back of Map 9). The highest figure is reported from the North Eastern Province with 3.66 per 100,000 inhabitants, followed by the Coast Province with 1.8 per 100,000 inhabitants, the Rift Valley Province with 1.49, the Eastern Province with 0.63, and the Central Province with 0.36. No cases of brucellosis appear in the long-term averages of the remaining provinces. The same thing applies to over 60% of the 54 government hospitals. Of the 21 hospitals which report brucellosis, only six record more than 10 per 100,000 inhabitants in their catchment area. These are the hospitals of Marsabit, Taveta, Voi, Wesu, Narok, and Kajiado (see Fig. 17, back of Map 9), this matches with the findings of Vanek (1976).

3. Echinococcosis
(Hydatid disease, ICD 122)

Echinococcosis is the term applied to the infection of man by the larvae of the canine tapeworm *Echinococcus alveolaris* and *Echinococcus cysticus,* the adult form of which lives as a parasite in the intestines of canines such as dogs and foxes. Generally cattle, sheep, and other mammals act as intermediate hosts, although man does so accidentally. The development cycle is closed when canines eat material containing cysts; adult worms develop in the intestines of the canines, and eggs are passed with the faeces, which in turn are taken up orally by the intermediate host on the pasture. Man is infected directly or indirectly by close and unhygienic contact with infected dogs and with dog faeces. Cystic echinococcosis form inside man when he is infected by *Echinococcus granulosus* and the alveolary echinococcosis when infected by *Echinococcus multilocularis.* The analogous infection of cattle, sheep, and goats occurs—so far as this is known through veterinary investigations—in up to 30% of all livestock in East Africa (Fendall and Grounds, 1965 b). Echinococcosis often occurs in association with brucellosis and anthrax in nomadic cattle economies where many dogs are kept which live in close contact with the people (Bell, 1956; Nelson and Rausch, 1963). The highest level of distribution of echinococcosis is found among the Turkana of northern Kenya, a fact which is also reflected in the hospital recording rates. The only hospitals recording a long-term average of echinococcosis are: Lodwar (116 cases per 10,000 inhabitants in the catchment area), Lokitauung (48), Marsabit (10), Narok (10), and Nakuru (2). In the remote hospitals of the north it is especially the surgeons of the Flying Doctor Service, who have specialized in operations on what are often gigantic hydatid cysts.

Recently O'Leary (1976) pointed again to the fact that hydatid disease is mainly concentrated in the northern parts of Turkana district. The mode of transmission is through close contact between infected dogs and infants within the homesteads. This is also reflected in the age and sex distribution: 42% of all hydatid cysts seen were in children under the age of 15, 55% of the children were females, while in adults 64% were females. Since the dead bodies, except those of the grandparents, are not buried and are therefore consumed by wild animals, man is not a dead-end host.

4. Leptospirosis
(ICD 100)

Another anthropozoonosis, leptospirosis, affects mainly rodents and domestic animals. Human infection occurs accidentally by direct contact with infected animals or through water which has been contaminated by sick animals. The causative agents are different, serologically discernible types of *Leptospira interrogans.*

In Kenya leptospirosis was first discovered among dogs by Piercy (1951). It was first described by Burdin et al. in 1958 in the course of observing an outbreak among cattle, sheep, and goats in Nanyuki District that had been caused by *Leptospira grippotyphosa.* As far as is known to date, leptospirosis in man in Kenya occurs in foci; labourers on the sugar plantations appear to be particularly prone to infection, to such a degree that it might be called an occupational disease. In the vicinity of Kisumu (Nyanza Province) on the sugar plantations of the Kano Plains 24% of the healthy persons medically examined produced positive serum titer; in the vicinity of the Shimba Hills and in the sugar cane plantations at Ramisi, south of Mombasa, the level rose to 26%. In accordance with the degree of exposure at work on the sugar plantations, it is young men who are particularly prone to infection. The causative agents belong mainly to the serum groups *L. australis, L. ictero haemorrhagiae,* and *L. hebdomadis.*

In the sugar plantations it is the bush rat *(Aethomys kaiseri)* which may be regarded as the main reservoir of *L. hebdomadis,* since it displays a 5.7% infection rate. Cattle seem to be by far the most important reservoir (Ball, 1966). The desert gerbils (*Tatera robusta* and *Acomys wilsoni*) also manifest high infection rates, fluctuating between 9.7 – 6.2%; they are the most important reservoir of leptospiroses of the *Icterohaemorrhagiae* serogroup. No reservoir of *L. australis* has so far been found (Forester et al., 1969; Kranendonk et al., 1967, 1968; De Geus et al., 1969; De Geus, 1974).

VI. Nutritional Problems and Anaemias

1. Nutritional Problems

In Kenya, as in many other African countries, the nutrition of the population, especially that of children, and all the problems connected with it must be considered under two aspects: the ecological "productive" and the socio-economic "consumptive". Although these two

aspects are frequently inter-related, they are in themselves affected by other factors. The problems relating to ecology and production have been described in Chapter A. IV, while those concerning decisions on production and distribution have been referred to on more than one occasion.

To sum up, it may be stated that ever since the colonial period it has been the declared aim of agricultural policy to give priority to food supplies for the population, particularly that affecting the self-sufficiency in home-grown products of the farming community. It was not until the basis of guaranteed supplies for home consumption among the farming population was secured that the commercialisation of small-scale African agriculture, i.e., cash cropping, was propagated. During the colonial period the development of the large farms remained, almost to the end, in the foreground of agricultural policy; small-scale agriculture began to secure increasing support and encouragement in the move from a purely subsistence economy to one partially based on market orientation after the implementation of the Swynnerton Plan.

In the pre-colonial and early colonial times, crop failures resulting from droughts and vermin attack, possibly combined with epidemics, led to famine and population losses. Events of this sort are to be found in reports for 1890, 1907, 1919, 1943, 1961, 1963, 1966, and 1971/72. Apart from these, the unpublished agricultural gazetteers compiled at district level by administrative officers in the late fifties tell of numerous regional crop failures and crises in food supply. As a rule, the stocks of foodstuffs produced on the farm only last from one harvest to the next. Thanks to the construction of facilities for transport, marketing, and storage, Kenya is now able by its own resources to meet bottlenecks in supply occasioned by regional crop failures and to even-out the considerable differences in yields that occur from one year to the next.

However, in the densely settled highlands the nutritional status of farmers with little or no land is deteriorating as the increase in population reduces the proportional availability of land. The reduction in soil fertility due to over-cropping, which has been described above, is further aggravating the position of this group of the population. If a crisis in the food supply for the rapidly increasing population is to be avoided, the stepping up of food production within the country will continue to be the most urgent task in the formulation of Kenya's agricultural policy. Improvement of cultivation methods, selection of seeds, use of fertilizers and the extension of irrigated cultivation can be employed in further increasing the productivity of large and small-scale agriculture. But the actual problem of nutrition is not only a question of improvements in agricultural techniques. It arises in the socio-economic and socio-cultural sectors as well—as has been pointed out time and time again in the annual reports of the provincial medical officers and of the Ministry of Health. The Annual Report of the Ministry of Health for 1962 (Annual Report, Min. of Health 1962) refers to the increasing problem of protein and calorie malnutrition (PCM). Besides drawing attention to the tradition-conditioned deficiencies and nutritional insufficiencies arising from too short births-intervals and ignorance of a more suited weanling diet, there is also special emphasis on nutritional disorders resulting from intensified food production for the market at the expense of that for domestic requirements.

A first Nutrition Survey, covering the period 1964 – 68, was carried out by the Ministry of Health with the support of the WHO, FAO, and UNICEF. On a sample basis this provided important information on the nutritional condition of the population (M. Bohdal, N. E. Gibbs, W. K. Simmons, Nairobi 1969: mimeographed report to the Government of Kenya). Three sub-locations of the Kikuyu tribe, two of the Luo, and two of the Kamba were selected for the investigation of the nutritional conditions as well as the dietary habits of the population. Though it had become more secure with regard to the regularity and quantity of yields since the introduction of maize as the staple crop, the traditional and formerly well-balanced diet has become unbalanced in respect of both amino acids and mineral content.

The Nutrition Survey showed that in the three areas under investigation, 75.8% of the calorie intake was contributed by carbohydrates, 14% by protein, and 10% by fats. Samples taken in the Central, Nyanza, and Kamba regions during the 1965 period of the investigation revealed that towards the end of the agricultural year more than 50% of the families consumed less than 80% of the appropriate quantity of foodstuffs. The individual supply situation varies according to the season; it is precarious before the harvest, and on the whole is always threatened by recurring setbacks due to drought, soil impoverishment, or flooding, and increases in family size. Even published production figures such as those quoted in the Economic Survey for the year 1973 should not blind one to the facts.

Various kinds of millet, especially finger millet (Wimbi) and sorghum (Mtama), were once the staple food of the African farming population in Kenya, and this food was noted for its high nutritional value. Some kinds of millet continue to be used for brewing beer with a low alcoholic content, and this can be regarded as a valuable foodstuff. The discovery of the New World allowed three important crops to be introduced to East Africa namely maize, cassava, and sweet potato. Mixed with beans or at times with meat, maize has now become the staple diet almost everywhere in Kenya. Only in a few areas—western Kenya and the coastal region—has sorghum retained its equal or greater importance alongside maize in the feeding of the population. So far as home consumption is concerned, rice is cultivated by only a few farmers along rivers in western Kenya, and 90% of the country's needs are supplied from the Mwea Tebere Rice Irrigation Scheme. This rice is consumed mainly by the Asian group of the population, the majority of whom live in towns. Beans play an important role

as sources of protein in almost all of Kenya's population groups. In some areas beans are capable of providing two or three crops a year. Cassava and sweet potato developed their important roles as reserve crops during periods of drought-induced crop failure as far back as the colonial era. Farmers in drought-prone areas are obliged by government decree to cultivate sweet potatoes and cassava. As the final crop in a rotation, cassava can remain in the ground for several years without perishing. Because of its modest demands on soil and attention, as well as its relatively high yields per acre, cassava a "lazy crop" is more popular among the farming population of the coast and western Kenya than is desirable for the community's health. Carbohydrates apart, cassava does not contain any important nutrients. Desirable quantities of animal protein, milk, and fat are only available to the better-off sections of the population, whereas the poorer population groups enjoy opportunities to consume these high-value and expensive foodstuffs only at irregular intervals. Limited supplies of fish are available on Lake Victoria and the coast, as noted in Chapter A. IV 6. The setting up of fishponds is only feasible in a few areas of Kenya and requires considerable "know-how". The development of a fishpond economy is for this reason still in its beginnings.

The food of infants after weaning is in particular very unbalanced and contains little protein; moreover, such feeding takes place too soon in many cases because of the onset of another pregnancy. It is known from certain studies that mothers usually breast-feed their children until the end of the first year and introduce supplementary food from the sixth month. This consists chiefly of maize gruel, and no vegetable or significant amount of protein is given. In rural areas about half of the mothers continue breast-feeding until the end of the 18th month; in urban areas it is rather less. Only 20% of the mothers continue with breast-feeding beyond the 18th month.

There are hardly any data on the clinical or subclinical malnutrition of children under fives, Blankhart (1974) being the only exception. Where 40% of the children under five are under-weight, the area is described as being of high risk, with shortage of food mainly contributing to this high prevalence of protein-calorie malnutrition. This was found, for example, in the Central Province at lower Mohito and Kituki, in the Eastern Province at Mbooni and Iveti-Kombu, in the inland coastal area near Kwale, and in the Eastern Province and northern Rift Valley.

According to the Annual Returns of Diseases of the Ministry of Health (Nairobi, 1969), nutritional disorders accounted for 6% of all deaths, being fourth in overall importance; of all cases admitted to government hospitals, this group accounted for 14.7% of the total and was third in importance.

The median treatment figures in the period 1965 to 1972 amounted to more than 25,000 annually in the case of kwashiorkor (code no. 286-6), to more than 6,000 for avitaminosis and other deficiency states (code no. 280-289), and to more than 14,000 cases for anaemias (code no. 290-293).

These statistics do not distinguish between kwashiorkor, marasmus and mixed forms of protein-calorie malnutrition.

In this considerable under-reporting must be noted, together with the fact that conditions of malnutrition are concealed behind the reported infectious diseases, particularly the lethal ones, and are responsible for the fatal results. The reporting of measles at a national level may serve here as an example, since it was the first statistical investigation of a relationship with kwashiorkor. The clinical progress of measles is influenced unfavourably by latent protein deficiency, and in the same manner a latent protein deficiency may become manifest through infection by measles (Morley, 1973).

From the aspect of geomedicine and epidemiology, this relationship can be very clearly demonstrated in Kenya (see Map 9 a).

The hospital recording rate of the 54 government hospitals confirm, after rank transformation, a relationship between measles and kwashiorkor for the years 1963 to 1972 (see Table 39). Making all due allowances for the weight and reliability of the data, this result can be fundamentally interpreted to the effect that, due to better

Table 39. Hospital Recording Rate (HRR) for kwashiorkor and measles of 54 Government Hospitals (1963 – 1972)

Measles, HRR (1963 – 1972) \ Kwashiorkor HRR (1963 – 1972) stanine rank (%)	lower 23% of hospitals with HRR <93	middle 54% of hospitals with HRR 93–465	upper 23% of hospitals with HRR >465	Σ
upper 23% of hospitals with HRR >1133	0	4	8	12
middle 54% of hospitals with HRR 221–1133	5	22	3	30
lower 23% of hospitals with HRR ≦220	7	4	1	12
Σ	12	30	12	54
	$\hat{\chi}^2 \gg \chi^2_{4,001}$			$\hat{\chi}^2 = 24{,}32$

protein nutrition, the interaction effect of nutrition and infection is less evident in areas with a low kwashiorkor and measles recording rate than in those areas with a high kwashiorkor and measles recording rate. In spite of the inadequacy of the data, this analysis permits conclusions about a regional distribution of the nutrition problem in Kenya.

It is possible to recognise three different areal groupings:

(1) Areas with hospitals having a *low kwashiorkor frequency,* with a *low or medium frequency of measles* (12 hospitals):

(a) Malindi, Mombasa, Galole.

(b) Narok, Kajiado, Moyale, Makindu.

(c) Nanyuki, Kapenguria, Kapkabet, Kisii.

This group includes coastal places with fishing (a), and areas where the population exercises a pastoral economy (b) and regions in which cattle keeping occupies a dominant position alongside arable farming (c). Although unable to keep many cattle because of the scarcity of farming land and the smallness of the farm units, the farming population of the Kisii Highlands carries on intensive agriculture which assures their food supplies.

(2) Areas with hospitals having a *medium frequency of measles and kwashiorkor* (26 hospitals):

Bungoma, Kakamega, Kisumu
Kitale, Tambach, Kabarnet, Maralal
Kapsabet, Nandi
Thomson's Falls, Nakuru
Nyeri, Muranga, Kiambu, Tigoni, Nairobi
Machakos, Kangundo, Kitui
Voi, Taveta
Kwale, Msambweni, Kilifi, Kipini and Lamu.

Considering the great variety of the areas listed above, it is not possible to give an explanation which would have general validity for this group.

(3) Areas with hospitals having a *high frequency of kwashiorkor, together with a medium or high measles frequency* (14 hospitals)

(a) Thika, Kerugoya, Embu, Meru, Wesu

(b) Kericho, Londiani, Molo, Naivasha

(c) Isiolo, Marsabit, Lodwar, Garissa, Wajir

This category characteristically includes areas of high population density and land scarcity (a), as well as large-farm and plantation areas with a large proportion of agricultural labourers and their families (b). It is difficult to explain the high frequency of kwashiorkor in area (c), in which the population carries on a pastoral economy. Malnutrition is to be expected, insofar as population has become too numerous for the numbers of cattle, and the areas affected are often afflicted by drought; but bearing in mind that the diet always consists in part of milk or meat, the incidence of kwashiorkor ought not to be too high. Although this analysis of hospital statistics allows no direct conclusions concerning the actual conditions of nutrition, they must nevertheless be regarded as geomedical-epidemiological indicators despite the limited evidence they offer.

Apart from the Nutrition Survey of 1964 – 1968, it is in Blankhart's work (1974) that attention is drawn to the serious problem. Above all this author has called for further investigations of the relationship between nutrition and infectious diseases in children under the age of three; for investigations into simple, cheap infant feeding formulas based on locally and seasonally available and acceptable food to be prepared at home; for speedy and frequently repeated examinations of the frequency of slight protein-calorie deficiency symptoms in relation to weaning patterns, particularly in areas which have fo far remained uninvestigated, as well as over local habits of child feeding with a view to encouraging good habits and changing harmful habits, and an analysis of the existing practices of nutrition education. Despite all these proposals nutrition does not yet appear to play the role in the country's development policy which this problem now deserves and will deserve even more in the future.

Although the development plans for 1970 – 1974 and 1974 – 1978 contained references to a planned National Nutrition Council, WHO and UNICEF have carried out investigations into nutrition and the annual reports of the Ministry of Health occasionally mention local nutrition problems, no greater importance has to date been attached to this decisive factor in health and development.

A nutrition unit at the Ministry of Health supervises 100 nutrition field workers at the provincial and district level. In district hospitals, in mother and child clinics, and in mobile health teams, explanatory talks on nutrition, cooking instructions and demonstrations are carried out on the basis of foodstuffs locally available. Considering the size and distribution of the groups addressed and aimed at, this number is totally inadequate given the nature of the problem.

2. Anaemias

Anaemia is a major cause of ill-health among large groups of population in many countries, particularly those of the tropics. The most vulnerable groups are females in the reproductive age and children after weaning. A major contributory factor is the infestation with certain parasites, resulting in blood-loss through the intestines or bladder, as in the case of hookworm or schistosomiasis. Inadequate iron intake due to iron deficiency of nutrients or impeded iron resorption due to gastro-intestinal disorders of various kinds or other metabolic disturbances result in a negative iron balance, enhanced by blood-loss during menstruation or delivery in the case of the female, or high iron demand or protein-calorie malnutrition during infancy and childhood.

Although hospital statistics barely distinguish between the different forms of anaemia, iron deficiency plays the dominant role. It is followed by megalocytic

anaemia as a result of folic acid and/or vitamin B 12 deficiency, anaemia due to malaria or sickle cell anaemia, and kwashiorkor (Young, Kondi and Foy, 1974).

The lack of capacity to work in the individual or population as a whole is difficult to measure and remains unknown in a country like Kenya, although it must be assumed to be considerable. Serious anaemias are a daily feature in medical practice, although there are distinctive regional variations. The actual distribution throughout the country remains unknown. The figures laid down in the hospital statistics provide, if anything, only a rough guide to the regional distribution of the problem.

Without entering into the particulars of the quantitative and qualitative problems of the relationship between hookworm infestation and anaemia, which has been investigated in Kenya by Foy and Kondi (1960), it should be noted that anaemias are quite frequently observed in the hospitals of the coastal towns where hookworm infections are also very commonly reported (see Map 8 and Chapter IV).

In the remaining parts of Kenya as well, a certain relationship can be recognised between the frequencies of anaemia and hookworm, and also between anaemia and kwashiorkor.

Another group, particularly typical for Africa, is that of the haemoglobinopathies. Foy and Kendall (1974) presented a survey which showed considerable variation in the geographical distribution of sickle-cell anaemia and the sickle-cell trait.

The highest frequencies were observed among the Luo in western Kenya, on the coast south of Mombasa, and in Taveta. Although the cause has not been completely established it is assumed that carriers of the sickle-cell trait experience a lower malaria morbidity and mortality than the carriers of normal haemoglobin, the former being subject to a positive selection as far as malaria is concerned. Other, rarer, haemoglobinopathies were also found sporadically, but no details of their frequency and geographical distribution are available.

VII. Diseases of Special Geomedical Relevance

Apart from the diseases described so far, the geomedical relevance of which is more or less evident from the ecological conditions of the disease vectors or the animal reservoir of the causal agents, or from special conditions of life or hygiene (i.e., the ecology of man), there are some other pathological conditions which are geomedically relevant in the narrower sense of the word. Endemic goitre or fluorosis are cases in point.

For years the observation of distinct regional variations in the occurrence of certain types of cancer in Kenya has provided particular stimulation for the undertaking of further investigations into cancer causation. Possible genetic or environmental factors which may contribute to this situation can be readily studied in the comparatively stable—although in life styles differing—population of Kenya.

1. The Geographical Distribution of Endemic Goitre

Hanegraaf and McGill (1970), the first to provide an extensive review of the relevant literature, arrived at the conclusion that the distribution pattern corresponds to that of other continents insofar as the inhabitants of mountains suffer more often from goitre than the remainder of the population. Endemic goitre appears to occur with marked frequency in the highlands of the central Rift Valley, the Aberdare Ranges, and in the Western Highlands. A detailed investigation was carried out by J. A. Munoz of the WHO Nutrition Unit during the period 1960 – 64. Goitre rates varying from 15 – 72% were discovered, the highest of these occurring in the highlands of the Rift Valley, in the Central Province, and in the Western Province (Bohdale et al., 1968). Carried out among school children, these investigations are not fully representative insofar as visible indications of a malfunctioning of the thyroid gland generally become apparent only in later years. Hanegraaf et al. (1970, 1971, 1974) therefore checked three rural areas near Naivasha, Kericho, and Machakos by conducting sample surveys among all age groups and examining iodine excretion, the protein-bound iodine in the serum, and the capacity for iodine uptake. There was a good consistency so far as epidemiologically established goitre, reduced iodine excretion, and increased iodine uptake were concerned. The authors concluded that in goitre areas where goitre occurs in younger age groups, the female sex, with the exception of the age group up to 6 years, is more prone to the disease, that cretinism does not appear, and that toxic disturbances of the thyroid gland are rare.

In the hospital recording rates the following hospitals stand out above all the others: Kericho (75 cases per 100,000 inhabitants in the catchment area as a long-term average), Nanyuki (46), Nyeri (33), Molo (17), Voi (16), Embu (12), Thomson's Falls (11), Nakuru (11), and Marsabit (10).

In 1970 preventive measures were started on an experimental basis by issuing iodized salt. Results have not been clearly established yet.

2. Fluorosis
(Mottled enamel)

While a reduced fluorine content in drinking water (< 1 p.p.m.) leads to susceptibility to dental caries, fluorine content of more than 4 p.p.m. among young people results in hypo-calcification of the dental enamel and thus to mottled enamel. Changes in teeth in accordance with this were observed mainly among school-children in the Rift Valley (Fendall and Grounds, 1965a). As a result of volcanism, the surface waters contain excessive fluorine content: Lake Rudolf (13.1 p.p.m.),

Lake Naivasha (30 p.p.m.), Lake Nakuru (2.8 p.p.m.). Well water also contains an increased fluorine content (Bakshi, 1974).

3. Cancer and Other Malignant Tumours

Whereas the total incidence of cancer in Africa, especially, in East Africa and Kenya, may be lower than in Europe and America, there are certainly some tumours which are far more common in Kenyans than in Europeans or Asians or other non-African communities. On the other hand, those cancers are few which are characteristic of industrialized western countries, or of populations with a high life expectancy or a child and infant population no more at risk to early infectious diseases and death. The quite distinct differences of cancer occurrences within African communities or localities lead to the view that environmental, dietary, or traditional factors influence the causation of particular tumours. The first person to draw attention to this fact was Denis Burkitt, who described what later came to be known as *Burkitt's lymphoma.* In 1964 – 1967, he carried out a large cancer survey for the most frequent and conspicuous tumours—such as oesophagus, stomach, liver, and penis cancer, scar epithelioma, Kaposi sarcoma, and Burkitt's lymphoma (Burkitt et al., 1969, Cook and Burkitt, 1970). Since 1967, a national cancer register has existed in collaboration with the International Agency on Cancer and the United Kingdom's Medical Research Council (Linsell, 1974). Data from these surveys and this register are quoted here.

Cancer of the oesophagus shows a great variation among Africans; with few exceptions it is almost unknown in Europe. Cancer of the oesophagus is the most frequent cancer in the Kisumu area and throughout the highlands of the eastern Rift Valley, while in other areas it is almost unknown. It seems to be predominantly confined to the Luo and Luhya. No plausible ecological explanation has so far been found. Stomach cancer seems, on the other hand, to be rare.

Cancer of the penis occurs almost exclusively among those tribes, the Luo, Samua, and Turkana, who do not practice circumcision, regardless of the type of circumcision or the age at which it is performed. As *cancer of the cervix uteri* is the most frequent female cancer in Kenya, it shows an association with the pattern of distribution of penis cancer.

Cancer of the liver: Heptocellular cancer is known to be common throughout tropical Africa. In Mozambique it accounts for over 50% of all cancer, in Ethiopia for more than 30%. In Kenya it appears to be common everywhere. It may possible be associated with chronic liver damage, with malnutrition, alcohol, virus infections, or the hepatotoxic aflatoxin produced by yeasts on stored cereals.

The *kaposi sarcoma,* a tumour of the vessel-forming mesenchym, which leads to knot formations on the skin, is much more widely spread in Africa than in Europe; it occurs in Kenya, but even more so in Uganda.

Nasopharyngeal cancer is especially frequent among the Kipsigis and Nandi, as well as among other highland dwellers, whereas it remains practically unknown in the lowlands. It has been suggested that this is to be attributed to the climatically-conditioned protracted sojourns in the smokey and badly ventilated huts; these with their continuously smouldering acacia wood fires lead to chronic conditions of irritation and carcinogenic effects (Fendall and Grounds, 1965 a).

Among the *tumours of the infant age group,* it is particularly *Burkitt's lymphoma* which shows a distinct correlation with climatic zones in which malaria is transmitted throughout the year. There seems to be a relationship with the *Epstein Barr virus,* possibly in connection with malaria infections.

These few examples show that the study of the geographical distribution of tumours and the conditions of life of their carriers can make an essential contribution to investigations of cancer causation.

4. Diabetes

Among Africans diabetes is by no means as rare as had previously been assumed. The problem of diabetes in Africa is mainly characterized by the lack of medical facilities and at present the virtually non-existent possibility to organise proper management of diabetes by the patient and the medical personnel. Diabetes clinics exist only at the Kenyatta National Hospital in Nairobi and at the Coast Provinces General Hospital at Mombasa. Chronic diabetics will soon die from the consequences of the disease, although disturbances of the metabolism, so frequent in Europe, are rarer as a result of a diet relatively devoid of fats. The earliest clinical-epidemiological analyses of cases of diabetes in Kenya were undertaken by Darragh, Hutchinson, and Mngola (1971) from Nairobi, and Mngola (1974) from Mombasa. The analysis of the past ten years of the hospital recording rate for diabetes shows a remarkable frequency in the hospitals on the coast, in the Central Province, and at Kericho. A detailed investigation of the frequency of this distribution would be informative. The 12 hospitals with the greatest occurrence of diabetes are situated on the coast of the Indian Ocean: Lamu records an annual mean of 51 cases per 100,000 inhabitants in its catchment area, followed by Kipini (35), Malindi (27), Kilifi (13), Mombasa (21), Msambweni (25), and Voi (20). This observation confirms the often discussed frequency of diabetes among Semitic and Hamitic population groups. In the Central Province, Meru reports 45, Embu 35, Nyeri 30 and Nairobi 14; Kericho with 49 cases is the only hospital outside both these regions which has a higher number of cases. The remaining 77% of the hospitals for which registration figures are available report insignificant occurrence or non-occurrence of diabetes. These regional differences certainly have reasons which would be interesting to investigate.