

Best Wishes, David Rogers

Rev. sci. tech. Off. int. Epiz., 1994, 13 (4), 1075-1124

Tsetse flies and their control

D.J. ROGERS *, G. HENDRICKX ** and J.H.W. SLINGENBERGH ***

*Summary: The authors use a quantitative modelling framework to describe and explore the features of the biology of tsetse flies (*Glossina* spp.) which are important in determining the rate of transmission of the African trypanosomiasis between hosts. Examples are presented of the contribution of previous research on tsetse to quantified epidemiological and epizootiological understanding, and areas of current ignorance are identified for future study.*

Spatial and temporal variations in risk are important (but rarely-studied) determinants of the impact of trypanosomiasis on humans, domestic animals and agricultural activities. Recent grid-based sampling surveys of Togo provide valuable data sets on tsetse, cattle and trypanosomiasis throughout the country.

*A combination of ground-based meteorological and remotely-sensed satellite data, within linear discriminant analytical models, enables description of the observed distributions of the five species of tsetse occurring in Togo, with accuracies of between 72% (*Glossina palpalis* and *G. tachinoides*) and 98% (*G. fuscica*). Abundance classes of the two most widespread species, *G. palpalis* and *G. tachinoides*, are described with accuracies of between 47% and 83%. This is especially remarkable given the relatively small differences between the average values of the predictor variables in areas of differing fly abundance.*

Similar analyses could be used to predict the occurrence and abundance of flies in other areas, which have not been surveyed to date, in order to plan tsetse control campaigns or explore development options.

Finally, some recent tsetse control campaigns are briefly reviewed. The shift of emphasis from fly eradication to fly control is associated with a devolution of responsibility for control activities from central government to local areas, communities or even individuals. The future role of central governments will remain crucial, however, in determining the areas in which different control options are practised, in facilitating control by local communities and in protecting controlled areas from re-invasion by flies from other areas.

KEYWORDS: Discriminant analysis – Disease transmission models – Epidemiology – *Glossina* – Population models – Remote (satellite) sensing – Togo – Trypanosomiasis – Tsetse – Vector abundance – Vector control – Vector distribution.

* Department of Zoology, University of Oxford, South Parks Road, Oxford OX1 3PS, United Kingdom.

** Projet Trypanosomose, B.P. 114, Sokodé, Togo, West Africa.

*** Food and Agriculture Organisation of the United Nations, AGAH (Animal Production and Health Division/Animal Health Service), Viale delle terme di Caracalla, Rome, Italy.

INTRODUCTION

In this article, tsetse flies (*Glossina* spp.) are examined as vectors of the human and animal trypanosomiasis in Africa. The discussion is based on a standard equation for the transmission of vector-borne diseases and explores the biological significance of each of the variables and parameters involved for the particular case of the African trypanosomiasis. This limitation of the discussion purposely excludes many fascinating details of tsetse biology which, although intriguing to entomologists, are irrelevant to the problems of the diseases transmitted by tsetse. Equations such as that which is employed here, however, do not take into account either spatial or temporal variation in disease transmission, and these topics are dealt with separately. The importance of spatial variation is highlighted by reference to standardised data sets, collected through grid-based sampling methods which have only recently been applied to the problems of tsetse and trypanosomiasis in Africa. These ground-collected data sets can now be complemented with information derived from remote-sensing satellites, which provide extensive yet uniform views of terrestrial vegetation cover and other surface characteristics. In combination, the standardised ground and satellite data sets, analysed using a variety of multivariate techniques, provide challenging insights into the relationships between vectors, hosts, habitats and disease agents, and allow both the identification of current problem areas and a prediction of the importance of tsetse-transmitted diseases in proposed development areas. However, care must be exercised in extending, in both time and space, the understanding gained by analysis of contemporary and coincident data sets.

Finally, modern methods for the control of tsetse are reviewed, leading to the conclusion that predictive modelling of disease risk areas can make a valuable contribution to cost-efficient and effective fly control.

AN OVERVIEW OF THE PROBLEM OF TSETSE AND TRYPANOSOMIASIS IN AFRICA

Tsetse are a small group of specialised flies (Diptera: Cyclorrhapha: Glossinidae) which are now restricted to tropical Africa. Adults of the 23 extant species and eight subspecies range in length from approximately 6 mm to 15 mm, and are generally dull yellow, pale brown or dark brown in colour, with occasional black markings. Tsetse are remarkable for their viviparity, females producing one, fully-grown larva approximately every 8-10 days. Larvae are deposited in shady places and usually burrow a short distance into the soil where they pupariate within a few minutes and complete development to the adult stage in approximately three weeks. Adults newly emerged from the puparia are soft to the touch and referred to as 'teneral', a stage which ends with the first blood meal. For nutrition, both male and female tsetse rely solely on vertebrate blood, which is taken in via the stylet-like proboscis. The nutritional imbalance of vertebrate blood means that a considerable proportion of the energy of each blood meal is devoted to transforming the meal into lipids ('fat'), which form the metabolic reserves of the fly (14, 15). These reserves are used for general metabolism and other activities, with an emphasis on flight in males and reproduction in females (121, 139). Flies live independently of their vertebrate hosts, except during the short periods of contact required for blood feeding, and are thus likely to encounter a variety of host individuals and/or species during their life-long quest for food. This factor,

together with the fact that female flies must live for a long period of time if they are to maximise the low reproductive output rate, makes tsetse flies ideal vectors of the African trypanosomiasis.

Dependence on shade restricts the distribution of tsetse to areas with at least some tree cover, and different *Glossina* spp. have different habitat requirements for survival. The (probably) ancestral *fuscus* group species are mainly forest-dwelling and consequently have little epidemiological significance (79). *Palpalis* group species generally inhabit forests and riverine-forest habitats, but some species, such as *Glossina tachinoides* Westwood, survive in otherwise very dry areas. Finally, *morsitans* group species are found in woodland savannah habitats where, traditionally, both humans and domestic animals are also most abundant. At certain human population levels, the abundance of these flies and their catholic feeding habits make the *morsitans* group species of particular importance in the transmission of both human and animal trypanosomiasis. However, as human population pressure increases, *morsitans* group flies tend to disappear; *palpalis* group species may continue to play a vector role in such areas, although they are less efficient transmitters of the cattle trypanosomiasis. Tsetse distribution in Africa was mapped by Ford and Katondo (38). Recent information on the present distribution of tsetse and on host ranges as determined by blood meal identification for the period 1953-1991 is given by Moloo (92).

The African trypanosomiasis of economic importance also fall into three major groups (59, 93). *Trypanosoma (Duttonella) vivax* Ziemann and *Trypanosoma (Nannomonas) congolense* Broden occur in many wild animal species and can cause anaemia, production losses, abortion and mortality in domestic herds. The impact of these trypanosomes depends on the type of stock involved. The physically smaller races of cattle centred on West Africa (*Bos taurus* L. type, e.g. N'Dama, Somba, Muturu, Lagune, etc.) show the least pronounced effects of trypanosome infection and are described as 'trypanotolerant'. Undoubtedly, however, they are affected by trypanosomiasis in the same way as other cattle races (113, 158), but at higher levels of trypanosome challenge. The more abundant zebu races (*Bos indicus* L.) are generally described as 'trypano-susceptible', but nevertheless show a greater degree of resistance to infection than introduced or exotic animals (102). A range of trypanotolerance therefore seems to exist in the various cattle stocks in Africa, each stock having a different history of introduction to, and distribution within the continent (99). Coupled with this is a complex of associated features of the different cattle races in terms of drought resistance and susceptibility to a whole range of other diseases, a reflection of the great variety of habitats and conditions throughout the continent (157).

The wide distribution of the animal trypanosomiasis in Africa, and their severe impact on animal production and agricultural production systems, make these diseases among the most important constraints on many economic activities of the agricultural sector in much of sub-Saharan Africa, an area of approximately 10 million km² (33, 97). Losses are generally so severe that they preclude the keeping of zebu stock in most tsetse areas.

The third important trypanosome, *Trypanosoma (Trypanozoon) brucei* Plimmer and Bradford, is widespread in the same hosts as the other two trypanosome species, but is usually less pathogenic to cattle. Only two strains of *T. brucei* infect humans, namely *T. b. gambiense* Dutton in West Africa and *T. b. rhodesiense* in East Africa; these species cause Gambian and Rhodesian sleeping sickness respectively, both of which are generally fatal unless treated. Rhodesian sleeping sickness (occurring in East and Southern Africa) is often epidemic, while Gambian sleeping sickness (occurring

throughout the moister parts of West and Central Africa) is usually endemic, with occasional epidemic outbreaks in relatively drier areas where disease symptoms can be much more like those of the East African disease (31). Sleeping sickness affects approximately 25,000 to 50,000 people per year in Africa, and cases tend to be restricted to relatively small geographical foci within the total range of tsetse flies. Although sleeping sickness is relatively uncommon compared with other human diseases in Africa, recent studies suggest that it ranks third in economic importance (after malaria and schistosomiasis) of all vector-borne human diseases in the countries concerned (180). Continual surveillance is necessary due to the ability of sleeping sickness to recrudescence in areas from which the infection was thought to have been cleared (26). Part of the reason for this is the zoonotic nature of the disease, even in West Africa where the existence of non-human reservoirs was at first doubted but is now generally accepted (53, 64).

FLY SAMPLING

As tsetse are attracted to a wide range of vertebrate species (including humans) in their quest for food, it is relatively easy to capture flies with modified entomological hand-nets, and this became one of the first methods of sampling flies. In order to standardise catches further, fly sampling was regularly performed over set paths through the bush, with a constant number of fly catchers and recorders stopping at fixed intervals to catch approaching flies ('fly-rounds'). It quickly became obvious, however, that fly-round sampling using human baits yielded catch rates which were unrepresentative of the species, absolute abundance, sex ratio and nutritional condition of the population of flies in any area. Nevertheless, the method did appear to give reasonable relative estimates of fly population size [confirmed in the case of *G. morsitans* Westwood by Jackson (61)], and could therefore be used to monitor seasonal and longer-term changes in relative fly abundance. Despite the obvious limitations, fly-round studies were initiated in both West and East Africa and provided most of the current insight into both the *morsitans* and *palpalis* groups during the early decades of this century. Modifications to this basic approach included taking an ox on the fly-round or carrying a black cloth screen between the fly-catchers; both modifications increased the catches of species not readily attracted by man alone.

These hand-net studies of tsetse developed in parallel with stationary traps. Many early designs aimed at mimicking some visual feature of tsetse hosts, such as size or shape [e.g. (96)], but this approach produced traps which were too bulky for routine field sampling. Nevertheless, much early information on species not usually attracted by humans (such as *G. pallidipes* Austen and *G. longipennis* Corti) came from trapping studies. Trapping was revolutionised in the 1970s by the invention of a lightweight, collapsible bi-conical trap (18) which, after much initial success with *palpalis* group species, was also applied to other species including *morsitans* group flies. The great success of this trap in catching flies, and the modest cost, increased the possibility that traps alone might be used to suppress fly populations; this approach was tested successfully in Central Africa (24, 48, 70). As with fly-round sampling, the relationship between real fly population abundance and trap catches has only rarely been investigated, although results suggest a simple linear relationship between these two variables (8).

A number of early studies had suggested that tsetse are attracted by host odours, dung or urine (19, 20) and by the smell of the hessian cloth sometimes used in trap construction (45). Little attention was paid to these results until Vale began a series of

elegant experiments which showed quite clearly that host odours are important in host location by *G. morsitans* and *G. pallidipes*, and that the use of odours can increase the catches of flies around visual targets (160, 164, 165, 166). These studies were strengthened by the use of electricified netting which allowed the manipulation of natural host odours and visual cues in the absence of human observers, thus also revealing that certain species of flies are repelled by humans (163, 167). Field studies on host odours and electric traps (10), combined with studies on tsetse antennal responses to odour components (7, 114, 115, 116, 148, 152), and on the activity and orientation of flies to these odours in wind tunnels (13, 117, 172), led to the production of both efficient odour baits for flies and modified designs of fly traps (74). Unfortunately, very few of the identified odours, except carbon dioxide, have as pronounced an effect on trap catches of *palpalis* group tsetse as on the *morsitans* group (68, 88, 101, 150).

Odour baits increase trap catches of all segments of the fly population, but often differentially. Flies with relatively high fat contents are scarce in unbaited catches but proportionally more common in baited-trap catches (123, 124, 125). This occurs either because the odour baits stimulate flight activity in a higher proportion of high-fat content flies, or because the baits increase the tendency of such flies to enter the traps.

At present, therefore, a series of trap designs exists which – in combination with odour baits for *morsitans* group flies – can increase fly catches many-fold in comparison with earlier trap designs, thus increasing the prospects of fly control (22). The problems of sampling biases remain, however, and these problems must be tackled soon if the considerable advances in the ability to catch flies and the understanding of fly behaviour are to be used not only for the suppression of fly populations but also for the study of tsetse population dynamics.

One final comment may be made on this subject. During the evolution of tsetse trapping methods, the traps have been made to **look** less and less like the wild animal hosts of the flies, but to **smell** more and more like them (98). Some advantage may be gained by exploring anew the visual attractiveness of traps to flies, by introducing modifications to make the traps, once again, rather more host-like in appearance.

TRYPANOSOME TRANSMISSION DYNAMICS

A number of mathematical models, both simulation (54, 55) and analytical (91, 134), have been proposed to describe the African trypanosomiases. The analytical models are derived ultimately from a formulation familiar to malariologists, the Macdonald model (81), a modified version of which is the ‘vectorial capacity’ equation first put forward by Garrett-Jones (40). The equation used by Macdonald describes the number of new cases of a vector-borne disease which will arise at some time in the future from one case at the present time. This quantity – sometimes called the ‘basic reproductive rate’ of a disease (more correctly, the ‘basic reproductive number’), R_0 – is defined as follows:

$$R_0 = \frac{a^2 m b c e^{-uT}}{ur} \quad (\text{equation 1})$$

where a is the vector biting rate (i.e. the reciprocal of the feeding interval), m is the ratio of vectors to hosts, b and c are the coefficients of transmission from the vector to the vertebrate and vertebrate to vector respectively, u is the vector mortality rate, T is the

incubation period within the vector and r is the rate of recovery of the infected vertebrate from infection. The following are well-established tenets of disease epidemiology:

- a) for the disease to persist, R_0 must be greater than 1.0;
- b) an increase in R_0 will increase the rate of disease spread after initial introduction in any area;
- c) to control the disease, R_0 must be reduced to less than 1.0;
- d) to eradicate the disease, R_0 must be kept below 1.0 for a sufficient period of time for the disease to drive itself to extinction.

It is therefore necessary to examine each of the parameters and variables in equation 1 in some detail, in order to understand the maintenance and spread of trypanosomiasis.

Tsetse fly biting rates

Tsetse flies take blood meals at intervals of approximately 2-3 days in the hot season and possibly up to 6-7 days in the cool season. Flies begin to die of starvation when their fat reserves fall to about 6% of their total dry body mass. With inadequate food, female flies are more likely to abort and thus reduce their lifetime reproductive output.

The need to find food within a relatively short period of time has led to the evolution of efficient host-finding mechanisms in tsetse. While the vision of flies, like that of most arthropods, is rather limited (hosts can be detected at distances of up to 50 m), the studies referred to above (in the section entitled 'Fly sampling') clearly demonstrate the importance of odour in host location, at least for the *morsitans* group. The results are consistent with the hypothesis that the odour of a single ox operates over approximately twice the distance over which visual cues are effective (162). Host detection occurs through a combination of upwind anemotaxis, a slowing of the rate of movement within odour plumes and a final, shorter-range visual orientation towards the host (50, 156, 165). The attraction of hosts is size dependent, perhaps a function of the amount of certain odours, such as carbon dioxide, that they emit (160). Despite this, many abundant host species – especially certain antelopes, such as the impala (*Aepyceros melampus* Lichtenstein) – are only rarely represented in the blood meals of captured flies. These hosts may be described as 'nervous' and can be seen to ripple their skin and flick their tails when attacked by flies, undoubtedly making feeding more hazardous for the flies. It has been suggested that such host reactions impose additional, feeding-related mortalities on tsetse flies (and other blood suckers), and that for this reason tsetse take blood meals as infrequently as possible, commensurate with fly survival. Simple mathematical models can be used to demonstrate that, over a rather wide range of values for feeding-related mortality (including that due to predators which gather around hosts and attack recently-fed flies), the optimum feeding interval for tsetse (i.e. the interval leading to maximum reproductive fitness of the flies) is approximately three days (126). This figure coincides with the mean interval between blood meals which has been deduced from mark/release/recapture studies (43, 129), from interpretation of the fat and/or residual blood meal contents of captured flies (121, 132) and from the frequency distributions of the calculated feeding times of females assigned to different days of the pregnancy cycle (125). Of course, these are all indirect methods of estimating the mean interval between blood meals, and are still the subject of much discussion among entomologists (57).

As flies feed on a wide range of hosts, and on some hosts more readily than on others, overall disease transmission rates depend on the precise composition of the choice of

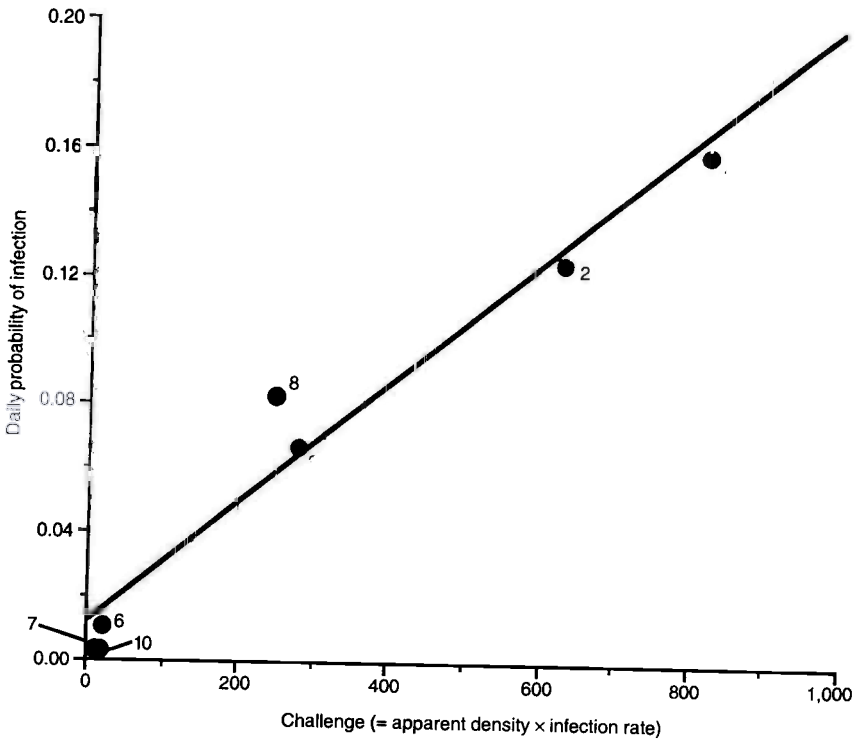
host species among local tsetse, the abundance of each host species, and its susceptibility to infection. Equation 1, in effect, must be written for each host species in turn, and the overall basic reproductive number of the tsetse-transmitted diseases is the sum of the reproductive numbers calculated separately for each host species. The biting rate on any particular host species may be calculated by multiplying the overall biting rate (i.e. the reciprocal of the average interval between blood meals on all hosts) by the proportion of blood meals which the flies take from that host species. Accurate estimation of this rate is important, as equation 1 shows that a doubling of the rate leads to a four-fold increase in the resultant R_0 .

Vector and host abundance

Equation 1 shows that disease transmission rates are determined by neither the abundance of tsetse nor the abundance of hosts *per se*, but by the ratio of these two values. Large populations of relatively mobile animals may be less satisfactory hosts for the flies (given the need for shade of both adult flies and puparia) than small populations of more sedentary species living in the same habitats as the flies. Classical studies of disease transmission foci confirm this impression. For example, a few sedentary and secretive bushbuck (*Tragelaphus scriptus* Pallas) present near human habitations may maintain trypanosomiasis long after most other wildlife species have disappeared or been eradicated (177). Similarly, a very small population of tsetse flies in close and frequent association with humans and domestic animals (e.g. near watering points) may cause persistent disease problems out of all proportion to fly numbers (25, 39). These observations, however, apply only to certain areas, and must not be allowed to obscure the general pattern of the normal relationship between fly abundance and disease risk shown in Figure 1. In the large majority of areas in which both animal and human trypanosomiasis occur, a direct and linear relationship exists between disease risk and either tsetse numbers or 'tsetse challenge', which is a product of relative fly abundance (as measured by fly-rounds or standard trap catches) and fly infection rate (83, 133). As fly infection rate tends to be a less variable component of fly challenge than fly abundance, it is the latter variable which requires more careful study. Reducing fly numbers by any method tends to lead to a proportional reduction in both human and animal trypanosomiasis (47, 49, 69, 95).

Figure 1, taken together with equation 1, implies that the ratio of tsetse vectors to hosts increases as fly density increases (i.e. fly numbers are more variable than host numbers). The dynamics of tsetse fly populations result from the impact of abiotic (non-living) and biotic (living) factors on the demographic rates of birth, death, immigration and emigration of the flies. In order to simplify studies, without losing too much detail, tsetse population models tend to ignore immigration and emigration and further assume that changes in birth rate, other than those associated with changes in mean temperature (which is known to affect birth rate directly by a change in the inter-larval period), can be expressed as an equivalent change in mortality. In studying fly population dynamics, it is important not only to quantify mortality rates but also to establish which of these rates depend on the density of the fly population. Only density-dependent rates can operate as 'negative feed-back' to regulate fly population size. The subtle but important difference between population regulation and population control is beyond the scope of this article, but this difference underpins much of the following discussion (131).

The abiotic factors which affect tsetse mortality rates include temperature and atmospheric moisture acting on the adult fly (16, 142), extremes of temperature (both high and low) causing puparia to metabolise all their fat reserves and die before



$$y = 0.012 + 0.000186x \quad r = 0.978 \quad P < 0.001$$

Relationship between the daily probability of trypanosome infection (all species) of zebu cattle in East Africa and the tsetse challenge (the product of tsetse apparent density and infection rate)

Sites were in Kenya (5, 6 and 10), Uganda (2, 3, 4 and 8) and Tanzania (1 and 7).

Site 1 is not included in the calculated regression for reasons given by the author in another paper, which contains further details (133)

development is completed (11), and seasonal heavy rainfall which floods puparial sites. During seasonal periods of stress, females also produce smaller puparia, giving rise to under-sized adults with a higher mortality rate than normal-sized adults (28, 118).

The biotic mortalities of tsetse arise from interactions with other tsetse of the same or different species, with hosts, and with parasites and predators. The first two factors tend to be related, as important interactions occur at or around hosts, resulting in density-dependent feeding success of the flies. For any given individual host, the chance of a fly obtaining a blood meal appears to be affected by the number of other flies present near the host and by the responses of the host to being fed upon. Hosts attempt to disturb or dislodge feeding flies more frequently as fly numbers increase (160); anaesthetised hosts, which show none of these responses, are fed upon more successfully than non-anaesthetised hosts (161). Thus, as the ratio of flies to hosts increases, the chances of flies feeding decrease, possibly leading to an increase in fly mortality, although this has never been directly quantified.

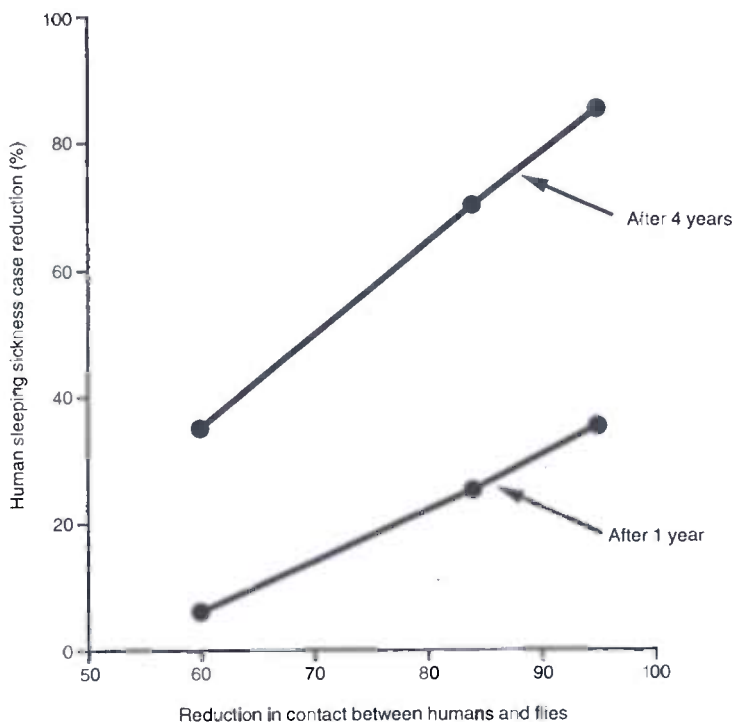


FIG. 1b

Relationship between the percentage reduction in human sleeping sickness cases and the reduction in contact between humans and flies brought about by the clearance of tsetse habitats along river courses in northern Ghana

(95)

Lines are shown for the periods one and four years after bush clearance (133)

Predation of both adult and puparial tsetse may be either density-dependent (128) or density-independent (143), apparently depending on the local concentrations of tsetse and the species of predators involved. Ants, especially those in the large genus *Pheidole*, removed up to 70% of buried *G. fuscipes fuscipes* Newstead puparia in Uganda over a period of 14 days (138), while unidentified bird predators removed up to the same percentage of adult flies on the vegetation within a period of 24 h. Less puparial predation occurred on *G. pallidipes* at Nguruman, in south-west Kenya, and there was no obvious sign of either adult or puparial predation being density-dependent over the (lower) ranges of densities used. Nevertheless, the average density-independent puparial mortality recorded at Nguruman – when combined with the results of previous studies in this area on the natural mortality rates of adult flies (28) – indicated demographic balance in the population, suggesting that no important additional mortality was missing from the calculations (143). This conclusion enables the construction of tsetse fly population models as an input into dynamic models of trypanosome transmission.

The basic minimum inputs into tsetse population models are temperature-dependent fertility and developmental rates, and abiotic and/or biotic mortalities operating on both soil-dwelling puparia and free-living adults. The models require a set of observations on the appropriate tsetse species in order to extract some measure of the seasonally-changing abiotic mortalities operating on the adults (the assumption that all mortality falls on the adults, rather than on the adults and puparia equally, does not qualitatively affect the outcome, as demonstrated by the models themselves) (130). Given the seasonal variation in temperatures and adult abiotic mortality rates, the basic tsetse population model may then be adjusted by adding density dependence to the adult or puparial stages, and additional density independence to both adults and puparia (the latter acting essentially to scale the output). Both the threshold density at which density dependence comes into action and the rate at which it changes with density (i.e. the slope of the graph relating mortality to density) are adjusted until the model output shows seasonal variation and timing of seasonal population peaks which best describe the observed result (136).

One might argue that very little may be gained from a modelling exercise in which many of the model inputs are derived from the data which the model seeks to describe. Experience shows, however, that model outputs are extremely sensitive to the characteristics of the density-dependent factors operating on the tsetse populations, and these have only rarely been quantified. The models may therefore be used to predict the degree of population regulation (i.e. density dependence) operating on tsetse species locally; thus, in turn, they may be used to predict the likely impact of control measures or environmental changes on fly numbers. The stronger the influence of regulatory mortality *vis-à-vis* density-disturbing (i.e. density-independent) mortality, the less significant will be the impact of any particular level of control or environmental change (131).

Figures 2 and 3 show some examples of models for *G. palpalis* Robineau-Desvoidy and *G. morsitans*. Figure 2 shows the significant difference to the observed population changes made by applying a range of density-dependent mortalities to the *G. palpalis* model. Figure 3 illustrates the effect of density-independent 'bottlenecks' of different severities, operating only during the teneral stage of *G. morsitans*, on predicted changes in numbers of this species in the Yankari Game Reserve, Nigeria. None of the other formulations of this model, in which either density dependence or density independence operated equally on all adult stages, was able to give such a satisfactory fit to the observed data (136).

Having earlier dismissed population movement as a factor considered by tsetse population modellers, it is important to realise that fly movement can be very extensive and there is now considerable evidence to suggest that it is density-dependent. *G. palpalis* populations in Côte d'Ivoire showed rates of population recovery (as estimated by trapping) after insecticide applications which were inversely related to population size and (at low density) far in excess of the reproductive capabilities of the populations concerned (122). On a much shorter time scale, a similar effect was observed in female *G. morsitans* and both male and female *G. pallidipes* in Zimbabwe (168). These results may be explained by invoking density-dependent regulation of adult fly populations via behavioural interactions.

Transmission coefficients from vertebrate to vector and vector to vertebrate

The transmission coefficient b is the proportional efficiency with which a single infected vector, taking a single blood meal, establishes an infection in a susceptible host, while the coefficient c is the analogous measure when a single uninfected fly takes a

blood meal from an infected vertebrate. Very few attempts have been made to measure these coefficients directly, and the results are listed in Table I.

Table I shows that the values of b increase, whilst the values of c tend to decrease in the sequence *T. vivax*, *T. congolense*, *T. brucei*, i.e. in the order of increasing complexity of the developmental pathways of the trypanosomes within the fly (*T. vivax* develops directly within the proboscis, *T. congolense* develops in the midgut and then moves up the gut to the proboscis, and *T. brucei* develops in the midgut and matures in the salivary glands after invasion from the gut or via the haemocoel). Younger flies are more easily infected with *T. congolense* than older flies, and this effect is even more pronounced in the case of *T. brucei*, where flies are almost refractory to infection after the very first blood meal (175) unless they are much older (41) or starved (42) (the figure in Table I refers to the first meal only; the average over several meals would be lower). In Table I, the values for most of the transmission coefficients are remarkably low in comparison with some tick-borne diseases, where coefficients may approach 1.0. Recent laboratory work on insects suggests that the values of the transmission coefficients reflect a balance of selective forces. Both mosquitoes and tsetse flies can be selected either for increased efficiency of transmission, or for refractoriness (84). In the case of tsetse, susceptibility to *T. brucei* infection is maternally inherited and is apparently a function of the abundance of cytoplasmic rickettsia-like organisms, which appear to be universally present in many tsetse species. Flies are particularly susceptible to midgut infections with trypanosomes when abundant rickettsia-like organisms produce high concentrations of chitinase enzymes, reducing the concentration of midgut lectins in the flies through the generation of *N*-acetyl-D-glucosamine, a lectin inhibitor (94, 174). Curiously, the maturation of these midgut infections to salivary gland forms requires different lectins, possibly of haemocoelic origin (85), and is also sex-linked (46). Recent work has shown that the rickettsia-like organisms are phylogenetically distinct from the midgut symbionts essential for blood meal digestion in tsetse, and from organisms, probably belonging to the genus *Wolbachia*, that are found in the ovaries (111).

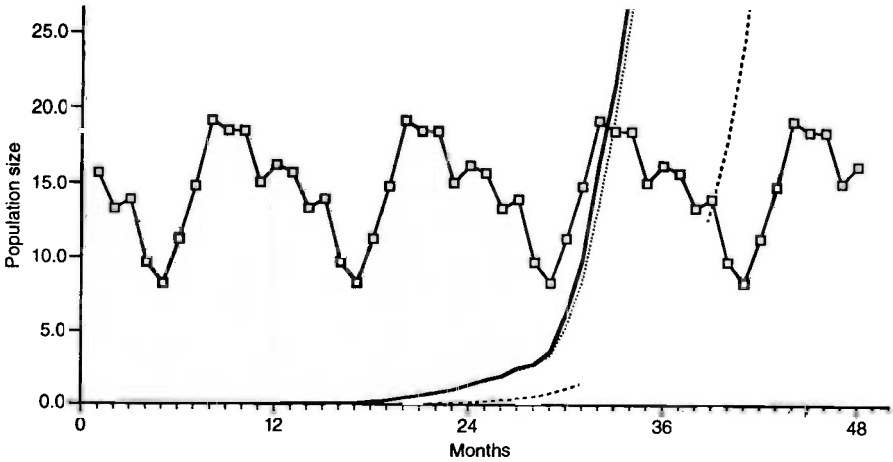
Temperature is another factor affecting the infection rate of flies, especially with *T. brucei*. Fly puparia incubated at higher temperatures produce flies which are more easily infected than flies incubated at lower puparial temperatures (104, 173), and this may explain a rough but significant correlation, first noted by Ford and Leggate (37), between average fly population infection rates and either mean latitude south of the equator or mean environmental temperature.

Another obvious factor affecting the values of the transmission coefficients is the nature of the hosts and the parasites. The range of tolerance of domestic cattle to

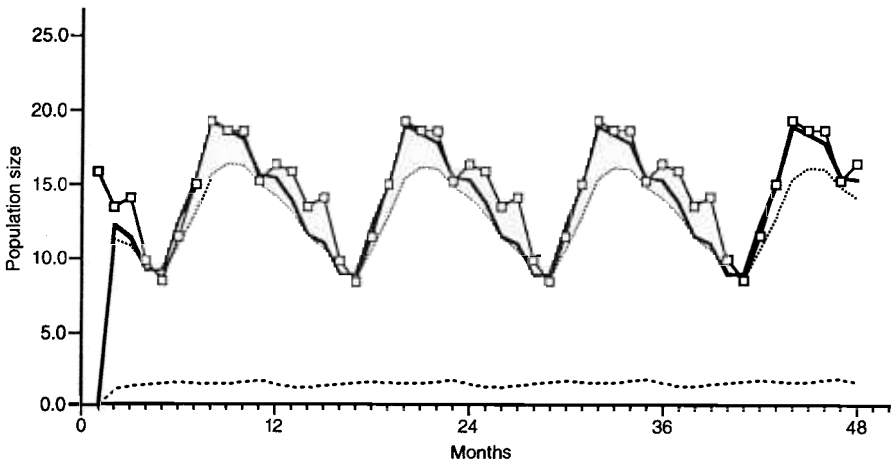
TABLE I

Values for the transmission coefficients of the African trypanosomiases
(134)

Trypanosome	Transmission from infected vector to susceptible vertebrate	Transmission from infected vertebrate to vector
<i>Trypanosoma vivax</i>	0.29	0.177
<i>T. congolense</i>	0.46	0.025 (average)
<i>T. brucei</i>	0.62	0.065 (first meal only)



a) Model including only the seasonally-varying, density-independent mortalities estimated from Moran curve analysis of the observed data set, and no density dependence. It should be noted that this model, with no regulation, is unstable



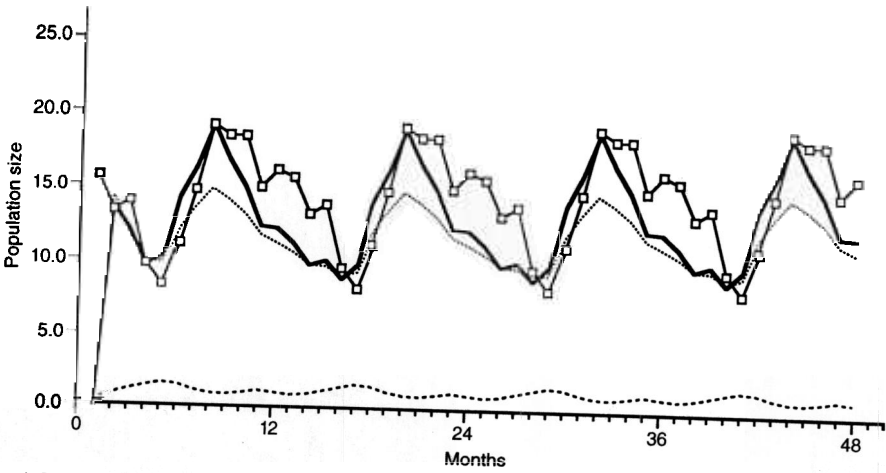
b) Model as in a), but incorporating density-dependent regulation in both the puparial and adult fly populations. Minimum, threshold and slope of the puparial density-dependent relationship were 0.005, 10 and 0.05, respectively (minimum value and slope refer to the logarithmic plot of mortality against density and hence are both logarithmic measures; the threshold is the number of flies per unit area below which the mortality is set at the minimum value). The minimum, threshold and slope of adult density dependence were 0, 200 and 0.01, respectively. This is the best fit to the observed data set

—□— Observed adults Model puparia - - - - - Model teneral ——— Model adults

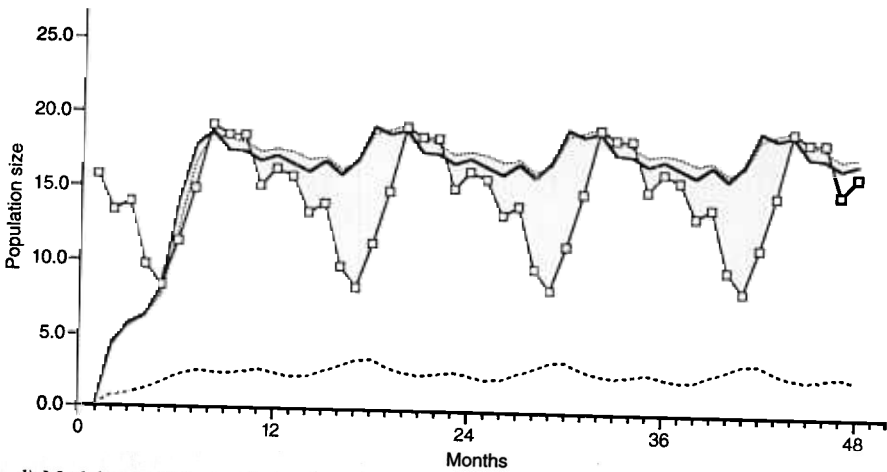
FIG. 2

Population models for *Glossina palpalis palpalis* in Katabu, Nigeria (136)

In all four models, the observed average monthly population count of flies is repeated four times, and model results (for puparia, teneral adults and mature adults) are super-imposed. For models b), c) and d), the outputs were scaled so that the maximum values equalled those observed. No other scaling was employed



c) Model as in b), but with the slope of puparial density dependence increased from 0.05 to 0.25. It should be noted that the numbers decline more rapidly after the annual peak than in b)



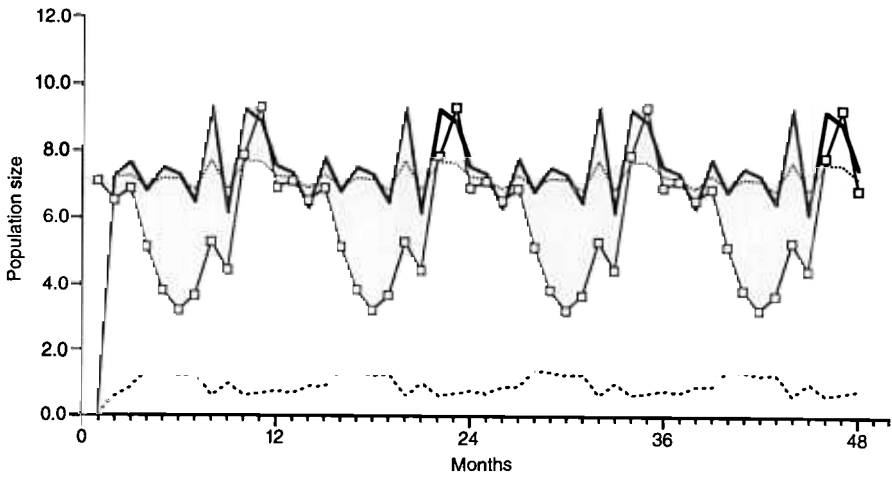
d) Model as in b), but with the threshold for puparial density dependence raised from 10 to 100, and the slope of adult density dependence raised from 0.01 to 0.50. The increased density dependence on the adult population stabilises the model population within much narrower limits than the observed population

Observed adults Model puparia Model tenerals ——— Model adults

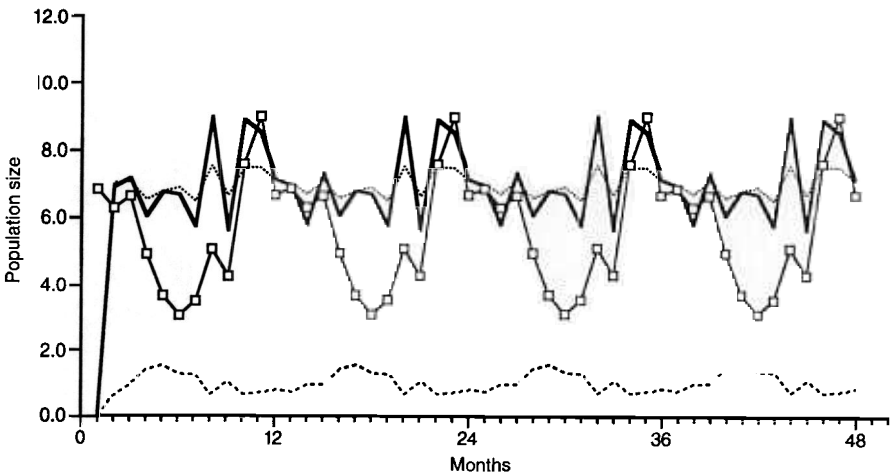
FIG. 2 (contd)

**Population models for *Glossina palpalis palpalis* in Katabu, Nigeria
(136)**

In all four models, the observed average monthly population count of flies is repeated four times, and model results (for puparia, teneral adults and mature adults) are super-imposed. For models b), c) and d), the outputs were scaled so that the maximum values equalled those observed. No other scaling was employed



a) Model with constant teneral density-independent mortality set at 0.00007. Minimum threshold and slopes of puparial and adult mortalities were 0.00130, 0.40 and 0.20, respectively



b) Model as in a), but with teneral density-independent mortality made equal to that of the adults (i.e. varying seasonally)

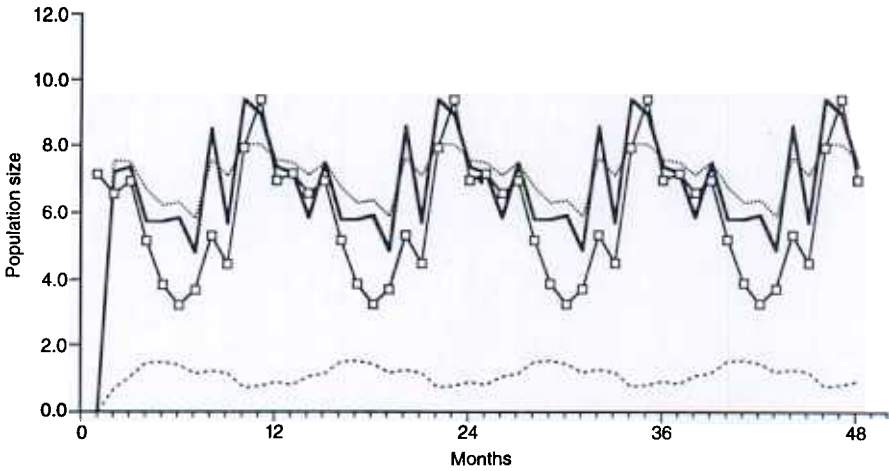
—□— Observed adults Model puparia - - - - - Model tenerals ——— Model adults

FIG. 3

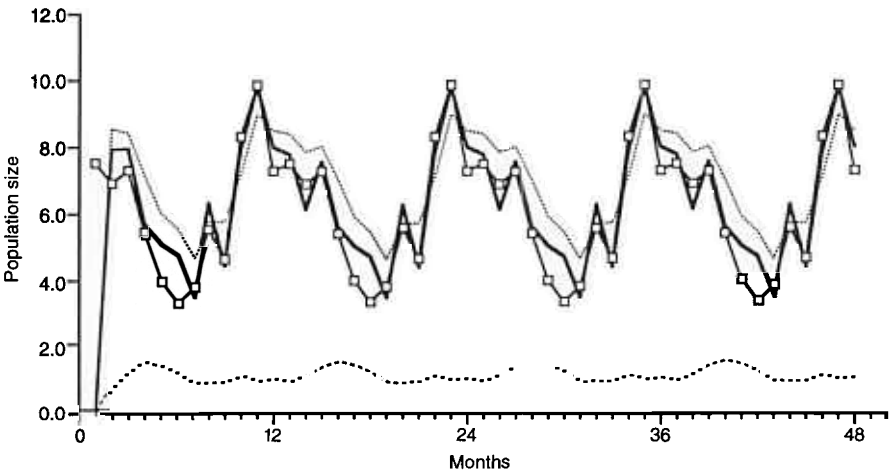
Population models for *Glossina morsitans submorsitans* in Yankari Game Reserve, Nigeria (136)

This series of models uses the observed seasonal variation in adult density-independent mortality (as estimated by Moran curve analysis of the field data) and explores the impact of changing the mortality rate of the teneral flies.

For all models, the outputs were scaled so that the maximum values equalled those observed. No other scaling was employed



c) Model as in a), but with teneral density-independent mortality set at twice that of the adults



d) Model as in a), but with teneral density-independent mortality set at three times that of the adults. This model provides the best fit to the observed data

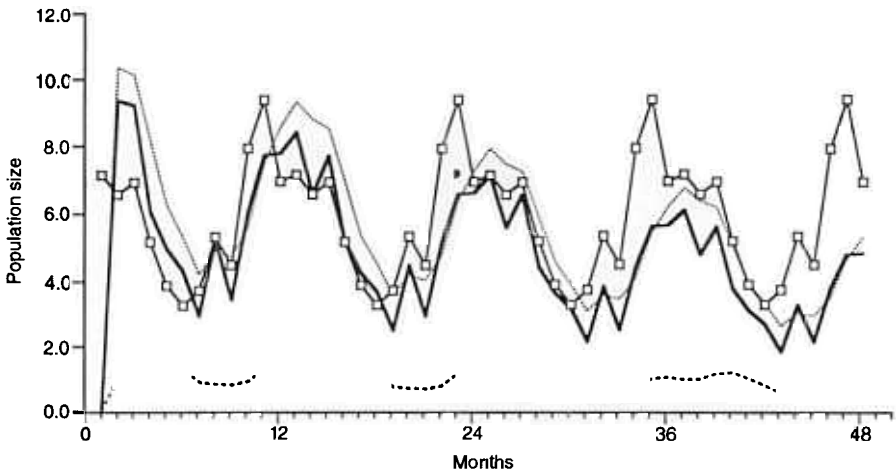
—□— Observed adults Model puparia - - - - - Model tenerals ——— Model adults

FIG. 3 (contd)

Population models for *Glossina morsitans submorsitans* in Yankari Game Reserve, Nigeria (136)

This series of models uses the observed seasonal variation in adult density-independent mortality (as estimated by Moran curve analysis of the field data) and explores the impact of changing the mortality rate of the teneral flies.

For all models, the outputs were scaled so that the maximum values equalled those observed. No other scaling was employed



e) Model as in a), but with teneral density-independent mortality set at four times that of the adults. Here, the additional mortality puts the population in long-term decline to extinction

—□— Observed adults Model puparia Model teneral — Model adults

FIG. 3 (contd)

**Population models for *Glossina morsitans submorsitans* in
Yankari Game Reserve, Nigeria
(136)**

This series of models uses the observed seasonal variation in adult density-independent mortality (as estimated by Moran curve analysis of the field data) and explores the impact of changing the mortality rate of the teneral flies.

For all models, the outputs were scaled so that the maximum values equalled those observed. No other scaling was employed

trypanosome infection has already been mentioned, and this may well be associated with differences in transmission coefficients. In addition, sexual recombination between trypanosome strains occurs within the tsetse vectors, generating new varieties in species which are already antigenically complex (62).

The demonstration that transmission coefficients which are normally low may be artificially selected to reach higher levels implies that infection of flies with trypanosomes necessarily entails some cost to the fly in terms of survival or reproductive output. Such penalties have rarely been investigated experimentally, and the evidence is somewhat equivocal (4, 82, 90, 106). Another important observation is that laboratory maintenance of flies creates quite different sets of selective pressures from those operating in the field, and care should therefore be taken when using, in models or other calculations, estimates of transmission coefficients from tsetse stocks which have been kept for many generations in artificial laboratory conditions. Similar reservations must apply to the trypanosome stocks used in such experiments.

The importance of the transmission coefficients cannot be over-emphasised, despite the very small number of experiments which have attempted to measure these using stocks with minimal laboratory history. Trypanosome transmission is as sensitive to

changes in these coefficients as to changes in the ratio of vectors to hosts, and both factors therefore merit equal study.

Fly mortality rates

The ways in which fly mortality rates affect tsetse population size are discussed above. These rates are also important in transmission dynamics, determining the fraction of newly-acquired infections in the fly population which will mature (i.e. will not be lost through premature fly death). The various means of estimating these rates are described below.

Direct estimation

The probability of adult fly death is independent of age for most realistic situations, and death therefore results from one of a number of hazards faced during everyday life. While it is possible to record the impact of particular sets of climatic conditions on fly survival both in the laboratory (16) and in the field (103), or to observe individual flies being captured by vertebrate (151) and invertebrate predators (66, 128) or hosts killing flies which attempt to feed on them (12, 44), it is difficult to convert any of these observations into realistic estimates of the resultant population mortality rates.

Indirect estimation from changes in fly population size

Changes in relative fly population size (measured by fly-rounds or stationary traps) may be used to estimate population mortality rates operating from one sampling occasion to the next (variation in fertility rates will also contribute to changes, but these are thought to be much less pronounced than changes in mortality rate). One simple method uses the difference between the logarithmic values of populations in consecutive months. Fixed fertility and mortality rates operating on the population in the interval between the two sampling occasions will result in a constant difference between the two logarithmic measures of population size, and any net variation in these rates causes changes in the difference, which can then be used as a measure of change in net population loss (or gain) rates. This method can reveal striking seasonal differences in the effective density dependence operating on tsetse populations (140). Both *G. palpalis* and *G. morsitans* populations in Nigeria showed greatest amounts of regulatory (i.e. density-dependent) mortality in the middle of the dry and wet seasons (December and July, respectively).

A variation of this approach draws on the experience of fisheries biologists who were the first to try to comprehend the dynamics of populations with overlapping generations and seasonal breeding (127). This method, when modified and applied to tsetse, involves plotting the population density of flies in one month (on the *y*-axis) against the density in the previous month (on the *x*-axis), and then constructing a population growth curve which best describes the maximal change of population size from one month to the next over the period of observation. It is assumed that this curve describes the way in which populations would grow if constrained only by density-dependent factors, and that departures from this curve are the result of density-independent factors operating (in addition to the density-dependent factors) from one month to the next. At least several years' sampling data are required for this technique to work properly, and the logarithmic version of this plot (the Moran curve) may be used to make direct estimates of density-independent mortality rates in terms of *k*-values (170), which may later be related to environmental conditions (130) and used in constructing tsetse population models (136).

With either of the above two methods, it is preferable to separate the population data for each sex, as mortality rates of males are generally higher than those of females.

Indirect estimation from changes in fly population age structure

Vertebrate and mosquito ecologists first developed techniques to calculate population mortality rates by analysing the age structure of the study populations. The assumptions involved, and some of the pitfalls to be avoided, are described by Caughley (17) and, for tsetse, by Van Sickle (169). Female tsetse flies have two ovaries, each with two ovarioles, in which the eggs mature in a predictable sequence. On maturation, each egg ruptures the follicular stalk attaching it to the ovariole wall and passes into the uterus, where it hatches as a larva about halfway through the inter-larval period. The ruptured follicular stalk remains as an indication of a previous ovulation and can be seen under a dissection microscope. Only one follicular relict is visible in any one ovariole, regardless of how many eggs have been ovulated from it. The predictable sequence of ovulation, and the absence or presence of follicular relicts in the ovarioles, allow dissected females to be assigned to one of eight ovarian age categories: four unique categories (0, 1, 2 and 3) and four repeated categories (4+, 5+, 6+ and 7+). The relative frequencies of these categories in the sampled population enable an estimate to be made of the population mortality rate, assuming this to be constant throughout the lifetime of the oldest individuals in the sample (an unfortunate but unavoidable assumption) (141). Studies on *G. palpalis* in West Africa showed a clear seasonal cycling of population mortality rates estimated in this way, and data from an extensive campaign of fly suppression in 340 settlements and villages in the region demonstrated quite clearly that apparently 'similar' control efforts had spectacularly different impacts from one village to the next, as a result of the rather different ways in which flies lived in and around each village (122). Even in villages where the control campaign was judged to have been successful (i.e. in which a significant reduction in fly numbers resulted from a dramatic increase in fly mortality rates), the impact of several cycles of aerial applications of deltamethrin and endosulfan in aerosol formulation on the target populations, and on their age structures, lasted for less than four months (122).

Incubation period of trypanosomes within the tsetse vectors

The interval between the moment when an uninfected fly takes an infected blood meal and the stage when this fly is able to transmit mature trypanosomes to another host, varies between approximately 10 days (for *T. vivax*) and 25 days (for *T. brucei*). Therefore, fewer immature infections are lost in *T. vivax* than in *T. brucei* during development within the fly, and this factor helps to explain the much higher infection rates of tsetse with the former trypanosome species.

Rate of recovery of the vertebrate host from infection

This rate is the reciprocal of the average duration of infection in the vertebrate host and obviously varies from one host species to another. Even within host species, values for the duration of infection may vary by one order of magnitude (or more) (59) and this, at least in domestic animals, is related *inter alia* to the general condition of the individual host (i.e. whether the host is anaemic, infected with other parasite species, etc.) (158).

The modelling of trypanosome transmission is complicated even further by the parasitaemic pattern within each host. Initially, after first infection of a previously uninfected host, blood parasitaemias may reach high levels. However, after the immune response develops in the host, high parasitaemias may become episodic or even absent. Hosts which are infected, but in which the blood parasitaemias are immeasurably small, may be rather poor sources of new fly infections, although they are not altogether ineffective, as these infections can sometimes be revealed by xeno-diagnosis (an important diagnostic tool).

Due to the enormous complexity of the transmission cycle of trypanosomes and the great variation in the fate of parasites within individual fly and vertebrate hosts, the general trypanosome transmission models referred to above have not been applied with any great success to any particular epidemiological or epizootiological situation. While it is possible to model the mean trypanosome prevalence within each of the components – the tsetse, the (generic) wild vertebrate and the human or domestic host (e.g. 135) – it has not proved possible to describe seasonal variation accurately. Especially in the case of trypanotolerant cattle, the time interval between the seasonal peak of tsetse numbers and the peak of animal infections is highly variable. In some cases, the animal infection peak appears to precede the tsetse population or challenge peak! Careful study of the seasonal grazing patterns of village-based cattle in The Gambia, and of their contact with populations of flies in patches of bush between grazing sites, suggests that fixed trap-based estimates of fly populations in such situations may provide only a poor measure of cattle biting rates (171). Clearly, many research challenges still lie ahead.

VARIATION IN SPACE

Figure 1 shows the need to define the local and regional distributional limits of the vector species and, together with equation 1, shows that vector abundance must also be predicted. Much progress has been made recently, with both grid-based sampling of tsetse populations and interpretation of the distributional data arising from these samples, using multivariate techniques such as linear discriminant analysis (145). The idea underlying these analyses is that each tsetse species can be imagined to exist in an n -dimensional environmental 'space', where the axes represent variables such as temperature, relative humidity and rainfall. Geographical areas translate to different regions of this n -dimensional space, depending on the environmental conditions. Areas containing flies can be characterised within environmental space, by calculating the means and covariances of their environmental variables. Areas outside the range of the flies can be similarly characterised. Representative sets of data points drawn from tsetse and non-tsetse areas, used to calculate means and covariances, are called 'training sets'. When training sets have been selected, it is possible to predict the probability of any particular point in geographical space belonging to the groups of points characterising tsetse and non-tsetse areas (it is assumed any point must belong to one or the other of these groups). One of the outputs of the analysis is a map of the probability of suitability for flies of countries and geographical regions from which the training sets were drawn, and of countries with broadly similar climatic conditions, i.e. a 'risk map' for tsetse flies. There are several measures of the accuracy of such spatial predictions of distributions. These include the percentage of correct predictions of fly presence and absence, the percentage of false positives (i.e. a false prediction of fly presence) and false negatives (i.e. a false prediction of fly absence), and the sensitivity (ability to predict fly presence correctly) and specificity (ability to predict fly absence correctly) of the final map; each measure may be put to different uses. In general, there tend to be proportionately fewer false-negative than false-positive predictions, suggesting both that the technique appears to provide a good description of the observed distributions (i.e. does not often predict that flies are absent when, in fact, they are present) and that either the maps are incomplete descriptions of fly distribution (i.e. flies are present in some areas but have not yet been caught there) or the region contains areas suitable for, but presently uninhabited by the flies. The linear discriminant analysis approach to describing arthropod distributions has already been applied successfully to the analysis of the

distribution of *G. morsitans* in Zimbabwe (with 85% correct predictions), of *G. morsitans* and *G. pallidipes* in Kenya and Tanzania (84% and 79%, respectively), and of the tick *Rhipicephalus appendiculatus* Neumann in Zimbabwe (72%) and in Kenya and Tanzania (73%) (145).

Tsetse flies in Togo

In recent years, the *Projet Trypanosomose* in Togo (GCP/TOG/013/BEL) has developed a country-wide, grid-based survey system at a resolution of 0.125°, and has sampled both the tsetse and the domestic animal populations. The project measured a number of important variables in each grid square (cattle numbers, blood packed-cell volume and infection rates; tsetse fly species and abundance as flies/trap/day). Because fly populations were estimated in a rolling sampling programme, it was necessary to adjust for seasonal differences between sampling occasions in the different grid squares, based on the monthly average tsetse populations across all grid squares for the region. This produced a seasonally-adjusted measure of fly abundance for each grid square. The analysis presented here is based on a logarithmic transformation of the abundance data (i.e. $\log. [\text{numbers} + 1]$, to avoid negative numbers).

Meteorological variables and altitude were later added to the Togo database using interpolations made from point data, and the level of agricultural activity in each grid square was read from a map of the agricultural sector of Togo (119). In characterising the environmental conditions within each grid square, the analysis also made use of satellite-derived data on vegetation cover and rainfall. The 'normalised difference vegetation index' (NDVI) was originally suggested as a measure of photosynthetic activity in plants (159), and is derived from readings from Channel 1 (visible red) and Channel 2 (near infrared) of the Advanced Very High Resolution Radiometer (AVHRR) instrument on board the National Oceanographic and Atmospheric Administration (NOAA) meteorological satellites which circle the globe in polar orbit, capturing data at approximately 1 km resolution in five wavebands:

$$\text{NDVI} = \frac{\text{Channel 2} - \text{Channel 1}}{\text{Channel 2} + \text{Channel 1}} \quad (\text{equation 2}).$$

NDVIs have been related to plant biomass production in the Sahel (5), and are now routinely used for vegetation monitoring as part of famine early-warning systems (120). Extreme NDVIs associated with high rainfall also coincided with outbreaks of Rift Valley fever in Kenya (80). The further potential importance of NDVIs for vector studies was suggested by the demonstration of correlations of NDVIs throughout Africa with rainfall and saturation deficit (144), two variables to which tsetse are particularly sensitive. This study also showed correlations of NDVI with tsetse fly mortality rates in West and East Africa, fly abundance in an area of approximately 140,000 km² in Côte d'Ivoire and fly size (a correlate of fly mortality rate) across a 700 km transect running approximately North/South in West Africa. Further studies revealed correlations of NDVI with both the incidence and the prevalence of human trypanosomiasis in Kenya and Uganda (137).

For the present study, NDVIs for Togo for 1989 and 1990 were made available by the Food and Agriculture Organisation of the United Nations (FAO) ARTEMIS programme, in the form of ten-day maximum value composites (the product of amalgamating a series of images taken over a ten-day period and eliminating the effect of cloud cover, by selecting for each pixel the maximum NDVI recorded in that time) at a spatial resolution of approximately 8 km. These ten-day composites were further

processed to provide monthly composites, and information was extracted from these for each of the approximately 14×14 km grid squares of the Togo data set. This involved centering a 2×2 pixel array as nearly as possible on the centre of each grid square; this array was then averaged, and from these averages were calculated the monthly mean, maximum and minimum values for the year. At a later stage, the monthly composite images for Togo for the period 1987-1989, at approximately 8 km spatial resolution, were processed using temporal Fourier analysis (147). This analysis effectively describes the vegetation 'signal' from each site as a series of sinusoidal curves of vegetation activity with frequencies of one to six cycles per year. The annual cycle tends to dominate in most areas, with the bi-annual cycle (i.e. with a period of six months) being an important modulator in many areas, and the tri-annual cycle (period of four months) providing a final adjustment of occasional importance. Cycles at higher frequency are usually ignored, as they appear to contribute very little to the description of the data. Previous work has shown how Fourier-analysed NDVI may be used to investigate ecological patterns and processes in unique and valuable ways. For example, the amplitude of the first Fourier 'component' (describing the annual cycle of vegetation activity) was shown to be very high in regions of deciduous savannah woodland throughout Africa, and hence to correlate well with the distribution of *G. morsitans*, which essentially inhabits savannah areas (147). The outputs from Fourier analysis used in the present study were the mean value, amplitude and phase (i.e. seasonal timing) of each of the annual, bi-annual and tri-annual cycles. These outputs were stored in the form of data layers and were sampled in the same way as the NDVI images, to yield average values for each of the Togo study grid squares.

In recent years, images derived from the geo-stationary weather satellite METEOSAT have also been processed to provide information on the frequency and duration of cold-cloud cover in various parts of the world. Studies in West Africa have shown that the abundance of clouds with cloud-top temperatures of between -30°C and -60°C or less (varying seasonally and latitudinally) is correlated with rainfall at ground level (149). Average monthly 'cold-cloud duration' images from the FAO ARTEMIS programme for the period 1989-1990 were sampled to give monthly mean, maximum and minimum values for inclusion in the analyses. Later, a set of monthly images averaged over the period 1988-1992 were processed in the same way as the NDVI images, and provided further variables for analysing the Togo fly data.

When all the data for each of the grid squares were available, these were analysed by a variety of methods. Fly distribution was analysed using step-wise linear discriminant analysis, in which each predictor variable was entered into the analysis in turn, and the discriminating power (i.e. ability to distinguish tsetse and non-tsetse areas) tested by calculating the resulting 'Mahalanobis distance' (a covariance-adjusted measure of separation in multivariate space) between the tsetse and non-tsetse area data; with the first variable, this test reduces to a simple comparison between variance-adjusted means [readers are referred to the work by Green (51) which provides further details]. At each stage, the variable resulting in the greatest Mahalanobis distance between training sample centroids was selected for inclusion in the next round of analysis. No more than ten variables were selected for each fly species (from a total of more than 25 in the database) and, when distribution predictions were made, it was usually found that the first four or five variables performed almost as well as all ten. As there was a total of only 311 grid squares in the Togo data set, all of these were used as training set data.

When the variables had been selected in order of their importance, it was possible to produce maps predicting the probability of fly occurrence in each grid square

(or, strictly speaking, the probability that each grid square belonged to the group of squares characterising fly presence, based on its environmental and other characteristics). These probability maps were based on from one to ten variables, and the progressive improvement of the fit of the map to the original data was assessed as described above. As the Mahalanobis distance provides a measure of separation of tsetse and non-tsetse areas in multivariate (not geographical) space, it cannot be guaranteed that the order of variables determined in the training exercise will lead to incremental improvement of the predicted maps. In general, however, there is an excellent correspondence between the aspatial training exercise and the resulting spatial predictions.

A variation on the above approach was introduced when it was appreciated that within Togo there is a fairly sharp division between the drier northern areas and the more humid southern areas. This led to separate analyses on either side of a dividing line shown in Figure 4.

Fly abundance in Togo (i.e. flies per trap per day) was initially analysed using step-wise multiple regression methods. While the broad patterns of fly distribution were captured by the final regressions, the extremes (both high and low) were only poorly described; this problem is common to all linear regression methods, as extreme values tend to be outliers from the regression line or plane. Instead of applying regression analysis therefore, the linear discriminant analysis program was modified to describe the abundance data as a series of abundance classes, each with approximately equal numbers of observations. During the training exercise, the predictor variables were tested and included in a step-wise fashion, as before. The criterion for inclusion was the incremental value of the summed Mahalanobis distances for all abundance categories (the 'Mahalanobis index'). Once again, the predictions arising from this training exercise could include any number of the variables which were identified as important, and the progressive improvements in the description of the original data could then be assessed.

Analyses of the distribution in Togo of five species of tsetse – namely *G. tachinoides*, *G. palpalis*, *G. morsitans*, *G. fusca* Walker and *G. longipalpis* Wiedemann – are shown in Figure 4. The predictor variables used in each and their order of importance in making the predictions are given in Table II, together with figures for the accuracy of the predictions. Analyses of the logarithmic abundance of two of the species, *G. palpalis* and *G. tachinoides*, are shown in Figure 5, and details are given in Table III. Here, the accuracy of prediction is assessed as the percentage of each abundance class which is correctly identified by the discriminant analysis exercise. As abundance classes are somewhat arbitrarily defined during analysis (i.e. chosen to give equal sample sizes), a further measure of accuracy was employed, namely the percentage of predictions which were correct to plus or minus one abundance class. This last measure, also given in Table III, shows how many 'near misses' were made when predicting fly abundance.

Figure 4 and Table II show that fly distributions are predicted with accuracies (percentage of correct figures) of between 72% (*G. palpalis* and *G. tachinoides*) and 98% (*G. fusca*), with sensitivities of between 56% (*G. morsitans*) and 76% (*G. palpalis*) and specificities of between 70% (*G. palpalis*) and 99% (*G. fusca*). In general, species with more restricted distributions are better described than more widespread species. The distribution of *G. tachinoides* was the most difficult to describe accurately. This species extends far south of the southerly limit shown in the map given by Ford and Katondo (38), and its range has also recently extended southwards elsewhere in West Africa, as droughts have become more frequent. *G. tachinoides* is also the only one of the five species in which separate fitting of the fly distributions in the north and south of

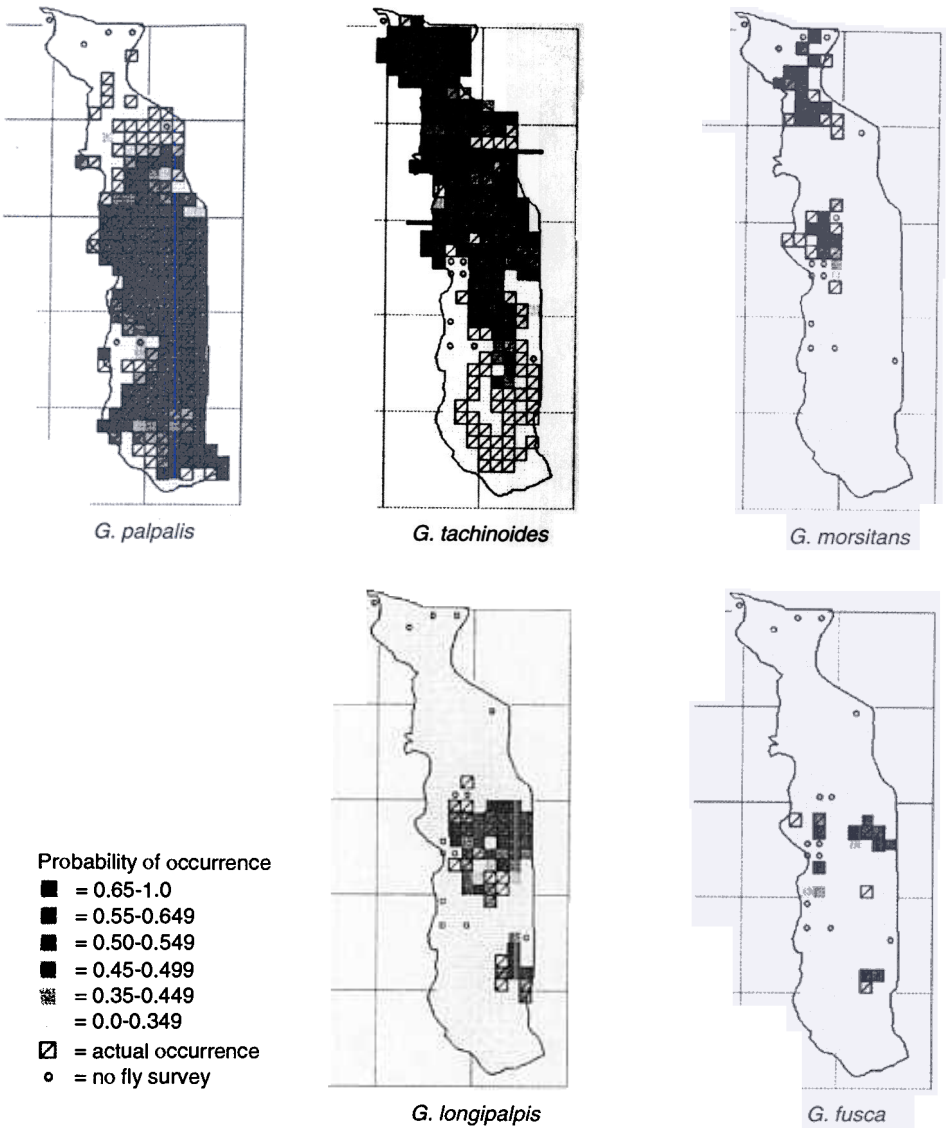


FIG. 4

Results of applying discriminant analysis to the distribution of five species of tsetse flies (*Glossina* spp.) in Togo, using the ten most important predictor variables for each, from a total of 25 available variables

The output of the analysis is the probability of occurrence of each species in each of the 311 grid squares, and these probabilities are shaded according to the key shown. The recorded distributions are also indicated. A few grid squares have yet to be surveyed (see text and Table II for more details)

TABLE II

Variables used to describe the distribution (i.e. presence/absence) of tsetse in Togo, together with the accuracy of the resulting maps (see also Fig. 4)

Species	Var. 1 *	Var. 2	Var. 3	Var. 4	Var. 5	Var. 6	Var. 7	Var. 8	Var. 9	Var. 10	n	Accuracy of predictions					
												% Correct	% False +ve	% False -ve	Sensitivity	Specificity	
<i>Glossina palpalis</i>	NDp2	NDa1	NDx	NDp1	NDa3	%Agric	CCDx	CCDp3	CCDp1	CCDa0		63	15	23	0.613	0.642	
											5	73	9	18	0.688	0.789	
											10	73	13	14	0.757	0.699	
<i>G. tachinoides</i>	NDa1	CCDp1	NDp3	%Agric	Rain	NDn	NDa0	NDp2	CCDp3	CCDa1		1	60	14	26	0.597	0.600
											5	72	9	19	0.702	0.743	
											10	73	9	18	0.717	0.743	
<i>G. morsitans</i>	Tx	%Agric	CCDx	CCDa0	NDa0	CCDa2	CCDn	NDp2	CCDp3	Tm		1	75	21	4	0.594	0.769
											5	91	6	3	0.719	0.932	
											10	95	0	5	0.563	0.996	
<i>G. longipalpis</i>	NDa3	CCDa3	NDp3	NDn	NDm	CCDa2	CCDx	CCDp3	CCDa0	Tm			59	38	3	0.737	0.566
											5	84	10	5	0.579	0.884	
											10	91	5	4	0.711	0.942	
<i>G. fusca</i>	%Agric	CCDx	CCDa2	NDp3	NDp2	CCDa0	Rain	NDx	NDm	NDn		1	66	33		0.75	0.651
											5	92	6	2	0.583	0.933	
											10	98		1	0.750	0.933	

* Variables 1 to 10 are those used in making the predictions, in order of importance

n : no. of variables taken into consideration

NDVI : normalised difference vegetation index

NDm : mean NDVI for 1989-1990

NDx : maximum NDVI for 1989-1990

NDn : minimum NDVI for 1989-1990

NDa : amplitudes (NDa0 = annual mean; NDa1-3 = annual to tri-annual values) of the cycles from the Fourier-processed NDVIs for the period 1987-1989

NDp1-3: phases corresponding to amplitudes (NDa1-3) (i.e. the months in which each cycle first reaches its maximum value in the year)

CCDm

mean of the monthly cold-cloud durations for the period 1989-1990

CCDx

maximum of the monthly cold-cloud durations for the period 1989-1990

CCDn

minimum of the monthly cold-cloud durations for the period 1989-1990

CCDa0-3

variables from the Fourier-processed cold-cloud duration images (naming analogous to NDVI images), based on mean monthly images for the period 1988-1992

%Agric

mean percentage area given to agricultural activity

Rain

rainfall

Tm

mean monthly temperature (from ground-based stations)

Tx

maximum monthly temperature (from ground-based stations)

Tn

minimum monthly temperature (from ground-based stations)

TABLE III

Percentage accuracy of predictions of abundance for Glossina palpalis and Glossina tachinoides in Togo

Accuracy is given after fitting the prediction to observed abundance, using 1, 5 or 10 predictor variables.

Predictor variables, in estimated order of importance, were as follows (see Table II for key to abbreviations):

– for *Glossina palpalis*: NDp2, CCDp1, CCDa3, CCDa2, NDa3, CCDa1, Tn, Tx, Tm and CCDa0

for *Glossina tachinoides*: CCDp3, NDp2, NDa1, %Agric, NDp3, CCDa3, Elevation, NDa0, CCDa1 and NDn

No. of variables used	<i>Glossina palpalis</i>					<i>Glossina tachinoides</i>				
	log. (abundance + 1)					log. (abundance + 1)				
	0.0-0.04	0.04-0.12	0.12-0.22	0.22-0.34	0.34-1.01	0.0-0.08	0.08-0.14	0.14-0.23	0.23-0.34	0.34-0.97
1	70.5	53.2	25.8	45.2	4.7	55.6	30.0	4.8	4.8	67.2
5	67.2	46.8	37.1	71.0	40.6	73.0	41.7	32.3	30.6	64.1
10	72.1	71.0	66.1	64.5	60.9	82.5	46.7	46.8	56.5	64.1
10*	91.8	91.9	98.4	100.0	89.1	93.7	86.7	87.1	83.9	92.2

* accuracy to \pm one abundance class

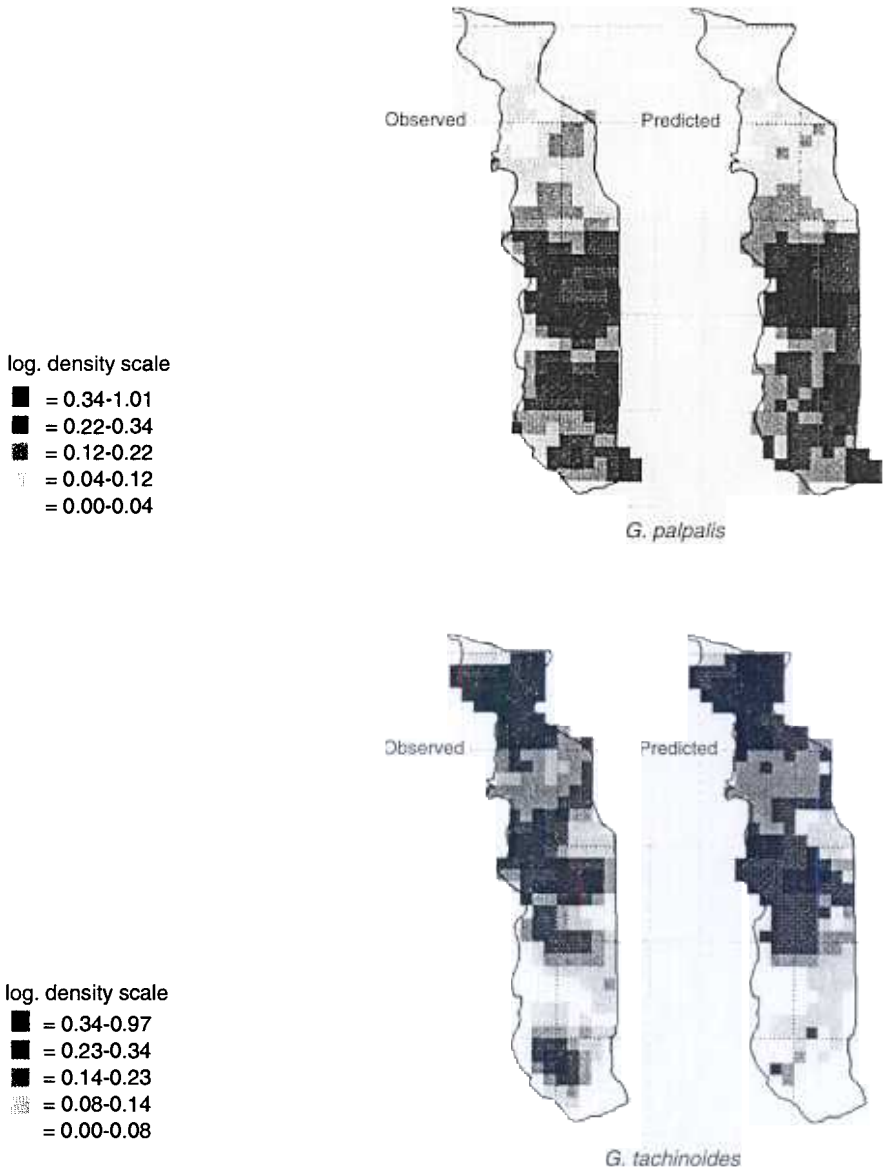


FIG. 5

Results of applying discriminant analysis to the abundance data for *Glossina palpalis* and *G. tachinoides* in Togo
(see Tables III and IV for further details)

Five categories of abundance were used for each species, giving approximately equal sample sizes. The survey results are shown on the left of each pair of maps, and the prediction on the right

the country leads to an overall improvement in the predicted distribution; for the other species, there is an improvement in one area but a worsening fit in the other area, so the overall results are no better. Table II suggests that a different mixture of the ground-based and satellite-derived measurements of climate or habitat is important for each species. Too much emphasis should not be placed on the precise ordering shown in Table II, however, as many of the variables are correlated with each other, and therefore may substitute for each other in the listings shown. Approximately one quarter (6/25) of the variables available to the analysis were derived from ground-based measurements and the same proportion (12/50) appeared in the list of important variables. This suggests that the remaining, satellite-derived variables predict tsetse distribution just as accurately as the more traditional meteorological and other variables measured at ground level.

Figure 5 and Table III show that the abundance classes of *G. palpalis* and *G. tachinoides* can be predicted with an accuracy of 47-83%. Taking account of 'near misses' (i.e. categories either side of the predicted category), these figures rise to 84-100%. The maps show once again that *G. tachinoides* in the south of Togo presents the more difficult challenge to the analysis. Finally Table IV shows the average values of all ten of the predictor variables for each abundance category of *G. tachinoides*, to demonstrate the small differences often seen between the mean values of the predictor variable for each category of presence/absence or abundance. Table IV shows that the first category, zero fly abundance, tends to be more distinct from the other categories than these are from each other, but even in this case the differences are not large. This suggests that fly distributions are very sensitive to subtle changes in the natural environment, and that continuous monitoring is required, especially at the edges of tsetse distributions which are likely to change rapidly, even before environmental change is appreciated.

VARIATION IN TIME

While rapid progress has been made recently in understanding the spatial distribution of tsetse and trypanosomiasis in Africa, the understanding of temporal variation in disease risk is still poor. The annual variation of fly numbers with the seasons is well appreciated, but the way in which this translates to seasonal variation of trypanosomiasis risk is unclear. Variations of tsetse populations and disease prevalence on longer time scales are only rarely recorded and even more rarely analysed. Long-term records of the abundance of *G. swynnertoni* Austen in Tanzania suggest that inter-annual variation in both rainfall and temperature affects fly abundance (32) and, in the same region, the numbers of cases of human sleeping sickness can be related to inter-annual variation in rainfall levels for part of the period 1922-1980 (146).

The epidemic nature of human sleeping sickness in East Africa is open to a number of interpretations, involving factors ranging from genetic mutation or re-arrangement in the trypanosome parasites to the whims of explorers or dictators who, by their actions, have introduced either old strains of trypanosomes into new areas or non-immune human populations into ancient foci of trypanosome transmission (146). There is little evidence for similar epizootics of trypanosomiasis among either wild animals or cattle, and, indeed, theoretical reasons suggesting why this should be so (135). The widespread nature of cattle trypanosomiasis (arising from the high basic reproductive number of this disease) ensures that most animals are infected at an early age and thus they either die or develop a certain degree of protective immunity. Less widespread diseases such as

TABLE IV

Mean values of the predictor variables for the five abundance classes of Glossina tachinoides in Togo
(See Table II for key to variable names and Table III for accuracy of predictions of abundance using the variables listed)

Abundance	CCDp3	NDp2	NDa1	% Agric	NDp3	CCDa3	Elevation	NDa0	CCDa1	NDn	n
0.0-0.08	2.51	3.90	0.05	43.52	2.03	12.13	248.7	0.36	49.35	0.21	64
0.08-0.14	2.99	3.64	0.07	41.78	1.86	15.08	290.4	0.36	51.86	0.16	59
0.14-0.23	3.30	3.60	0.07	37.82	1.78	16.60	295.2	0.36	54.73	0.16	62
0.23-0.34	3.32	3.40	0.07	37.02	1.72	18.32	302.9	0.36	56.40	0.16	62
0.34-0.97	3.59	2.89	0.08	26.64	1.70	18.19	262.4	0.35	59.30	0.15	64
Average	3.14	3.48	0.07	37.28	1.82	16.06	279.5	0.36	54.35	0.17	311

human sleeping sickness (with low reproductive numbers) are much less prevalent and hosts (in this case human beings) can therefore build up large populations of susceptible individuals before the disease is able to take hold and spread during epidemic outbreaks. Between outbreaks, the disease persists in a very small proportion of individuals, or in a non-human reservoir.

TSETSE CONTROL

With a few notable exceptions, the history of tsetse control in Africa is a record of increasing sophistication of the methodology, increasingly targeted specifically at tsetse. General methods of tsetse control are published by the FAO (34, 35), specific case studies are described by Jordan (63) and more recent articles have reviewed insecticides (1), traps (78), traps and screens (2), and baits (both visual and olfactory) (50, 76). A review of the control of trypanosomiasis from a veterinary perspective is given by Murray *et al.* (100).

To be effective, each method of tsetse and trypanosomiasis control must affect one or other of the parameters or variables of equation 1. Tsetse control affects both fly numbers and fly mortality rates, and the duration and degree of impact determine the amount of suppression achieved. The initial attraction of residual insecticides applied throughout the tsetse habitat – their persistence long after spray teams or aeroplanes had departed – is now regarded as an environmentally harmful consequence of tsetse control. Despite the lack of hard evidence, it is judged that the damage inflicted outweighs the obvious benefits of residual sprays (it should be noted that the amounts of persistent insecticides used for tsetse control are only a fraction of the amounts applied to crops such as cotton). Two major developments arose from the criticisms of these early attempts at large-area fly eradication. The first was the deployment of less environmentally-harmful chemicals, such as the synthetic pyrethroids, often in aerosol formulations. Aerosols affect fly mortality rates for a limited period of time only, and must therefore be applied several times to be effective against flies that are in the puparial stages during the early applications (at any time, the number of puparia present in the soil is approximately equal to the number of adult tsetse in the air, as shown in Figures 2 and 3). This undoubtedly increases costs without obviously increasing benefits. The second major development involved the use of traps and target screens for fly control (69). When supplemented with appropriate odours, both impregnated and non-impregnated traps and insecticide-impregnated screens achieve remarkable reductions of fly populations, usually within the space of less than a year. Table V provides details of some recent examples. As the insecticides are localised in the environment, these may be applied to the targets at high doses, and may thus remain active for many months. The addition of ultraviolet inhibitors to treated material further prolongs the action of the insecticides. The fact that screens and traps can be tailored to be especially attractive to tsetse rather than to other invertebrates makes this form of tsetse control both precisely targeted and environmentally acceptable. The same traps or targets (untreated with insecticides) may also be used to deliver doses of chemosterilants or insect hormone mimics which interfere with the normal development of tsetse. These chemicals are picked up by flies visiting the targets and are later transferred to other tsetse during mating (56, 72, 73).

The rapid development of tsetse control methods in recent years has partly obscured the objectives of the control programmes in which they are used. Long-term fly

TABLE V
Recent examples of tsetse control operations
 (see Green [50] for earlier examples)

Control method	Country/region	Tsetse species	Type/density of traps/targets	Insecticide and dose rate	Odour baits /dose rate	Operational area	Reduction in tsetse density	Time taken	Comments (Reference)
Aerial spraying	Uganda, Busoga	<i>Glossina fuscipes fuscipes</i>		Five sequential aerosol applications of endosulfan at flow rates of 5.6-6.7 l/min, vmd 28-30 µm		1,525 km ²		Dry season applications, Jan.-Mar. and Jun.-Aug.	Cases of sleeping sickness decreased by >50% in treated areas (65)
Ground spraying	Uganda	<i>G.f. fuscipes</i>		10% wettable powder lambda-cyhalothrin at 11.6 g ai/ha		28 km ²	100%	1-2 months	Insecticide applied selectively to fly resting sites. Electrolyt applications were also tried at 0.9 g ai/ha over 30 km ² , and reduced flies to very low levels. Sleeping sickness cases in the study area fell from 4-12/month to <1/month during the campaign (108)
	Kenya, Kisumu	<i>G.f. fuscipes</i>		0.075% deltamethrin + 0.3% cypermethrin		4 km 'linear' habitat	95%	4-5 weeks	Cypermethrin eliminated the flies after three 5-week cycles (110)
Traps	Congo, Niari focus	<i>G. palpalis</i>	1,263 pyramidal traps in 55 villages				100% in 9 villages 90% in 38 more villages	3 years	Human seroprevalence fell to 0.41%, and sentinel animals confirmed the cessation of transmission. Community participation was involved, but interest tended to decline with reduction of the tsetse population (47)
	Congo, Nioki	<i>G. palpalis</i>	101 mono-pyramidal traps in 14 villages				95%	9 months	Higher apparent densities were found in villages with higher incidences of sleeping sickness (83)
Traps + insecticides	Côte d'Ivoire	All	mono-conical traps	a-cypermethrin (300-400 mg/m ²)		60,000 km ²	95-100%	1 year	(52, 2)

Traps + insecticides (contd)	Côte d'Ivoire, Boundiali	<i>G. palpalis</i> <i>G. tachinoides</i>	542 bi-conical traps at 300 m intervals in gallery forest + 2 trap barriers 10 km long, with traps at 100 m intervals	cypermethrin		<i>G. palpalis</i> 95% <i>G. tachinoides</i> 99%	7 months	No corresponding reduction in trypanosome prevalence in livestock (animals were untreated and able to move freely in and out of the tsetse control areas) (23)	
	Uganda, Busoga	<i>G.f. fuscipes</i>	pyramidal traps at 10/km ²	deltamethrin	Busoga District	>95%, up to 100%	2 years	In the initial phase, 4,000 traps were set in 1,000 km ² . Since 1990, the project has extended into the neighbouring Tororo district, with similar results. Here, monthly treatment of cattle with deltamethrin pour-on was also applied in a small area (71, 69)	
	Uganda	<i>G.f. fuscipes</i>	pyramidal traps at ~ 7/km ²	lambda-cyhalothrin 100 mg/m ²	32 km ²	>95%	6 months	(108)	
Traps + odours	Kenya, Nguruman	<i>G. pallidipes</i> <i>G. longipennis</i>	100 Nguruman traps		Acetone and cow urine at 150 and 1,000 mg/h, respectively	100 km ²	<i>G. pallidipes</i> 98-99% <i>G. longipennis</i> ~ 90%	9 months	90 additional traps were added later to enlarge the suppression zone and reduce re-invasion (29)
Targets + insecticides + odours	Rwanda	All	Zimbabwe-style, black cloth flanked by mosquito netting	0.1% deltamethrin then 0.05% every 2 months	Acetone and 1-octen-3-ol		98%	6 months	(105)
	Zimbabwe	<i>G. morsitans</i> <i>G. pallidipes</i>	1.5 x 0.5 m targets at 4/km ²	500 ml of 0.1% deltamethrin per target; re-sprayed and re-baited every 3 months	Acetone, 1-octen-3-ol, 4-methylphenol and 3-n-propylphenol at 500, 400, 800 and 100 mg/h, respectively	600 km ²	>99%	6 months	(9)

TABLE V (contd)
Recent examples of tsetse control operations

Control method	Country/region	Tsetse species	Type/density of traps/targets	Insecticide and dose rate	Odour baits /dose rate	Operational area	Reduction in tsetse density	Time taken	Comments (Reference)
Targets + insecticides + odours (contd)	Kenya, Galana	<i>G. pallidipes</i> <i>G. austeni</i> <i>G. longipennis</i> <i>G. brevipalpis</i>	Zimbabwe-style targets at 5/km ²	0.1% deltamethrin then 0.05% at 3-monthly intervals	Acetone and 1-octen-3-ol	25 km ²	Immediate reduction noted, followed by eradication	1 year	Flies later re-invaded from outside the suppression zone (112)
	Zambia, Western Province	<i>G.m. centralis</i>	1.7 × 1.0 m black cloth targets (with or without flanking netting) at 4/km ²	deltamethrin suspension concentrate	Acetone, 1-octen-3-ol at 130 and 0.5 mg/h, respectively	500 km ²	Fly population initially fell by 3%/day and was eradicated in one year	1 year	Control activities were later extended into a further area of 2,550 km ² with targets at <1.6-3.7/km ² (see below) (176)
	Zambia	<i>G.m. centralis</i>	1.8 × 1.0 m targets at 0.2-2.3/km ²	deltamethrin suspension concentrate	Butanone and/or acetone at 40-130 mg/h and 1-octen-3-ol at 0.5 mg/h		Fly population initially fell by 3%/day, but reached only 99% reduction	1 year	Experimental trial of reduced target densities. The authors conclude that the prolonged period required for eradication made this alternative financially unattractive (67)
Traps + targets + insecticides	Togo	<i>G. palpalis</i> <i>G. tachinoides</i>	22 bi-conical traps in 2.2 km gallery forest + 32 screens, re-sprayed every 3 months	deltamethrin		32 ha	97.8% fly reduction, 88.1% reduction of trypanosomiasis in trypano-tolerant cattle	1 year	Both the experimental herd and a herd kept in an uncontrolled area were treated monthly with Berenil. Productivity increased in the experimental herd relative to the other herd, with fewer abortions, reduced calf mortality and increased calving rate (86, 87)
	Côte d'Ivoire	<i>G. palpalis</i> <i>G. tachinoides</i>	Insecticide-impregnated screens and traps			12,000 km ²	95%	Maintained over 6-year period	(27)

Traps + targets + insecticides (contd)	Côte d'Ivoire	<i>G.p. palpalis</i>	38,412 black-blue-black screens + 464 impregnated Vavoua traps at village margins	K-Othrine 50	,500 km ²	99.96%	7 months	Scheme involved allocating screens to farmers who maintained and re-impregnated these every 6 months. Project employee maintained and monitored the traps (75, 77)
Dips	Zimbabwe, Chesa	<i>G. pallidipes</i>		deltamethrin at 37.9 ppm; weekly dips	331 cattle	Trypanosomiasis incidence in treated cattle declined, but doubled in untreated animals	4 months	(154)
	Zimbabwe	<i>G. morsitans</i>		deltamethrin at 37.5 ppm; fortnightly dips	26,244 cattle	Trypanosomiasis incidence fell to zero	3 months	(154)
Pour-ons	Burkina Faso, Satiri	<i>G.p. gambiensis</i> <i>G. tachinoides</i> <i>G.m. submorsitans</i>		1 mg ai flumethrin/kg body weight/month	2,000 zebu cattle	Trypanosomiasis prevalence reduced from 20-77% to 0-5% after third month (cf. up to 30% or more in an untreated herd 30 km away)	1 year	A tsetse population disappeared from a heavily-infested habitat after six treatments. Trypanosomiasis prevalence in 200 monitored cattle fell from 40% to zero. Tick burdens also fell by more than 80% within 4 weeks (6, 89)
	Burkina Faso, Satiri	<i>G.p. gambiensis</i> <i>G. tachinoides</i> <i>G.m. submorsitans</i>		1 mg ai flumethrin/kg body weight/ 2 months	3,282-8,624 cattle	Highest infection rate in sentinel animals was 1.4%	1 year	Control supplemented with 130 insecticide impregnated moniconical traps deployed in habitats inaccessible to the cattle. In monitored village animals, trypanosomiasis prevalence dropped to 4.8% (6)
	Zanzibar	<i>G. austeni</i>		1% deltamethrin pour-on every 15-18 days	700 cattle, 200 goats and some donkeys	100% fly reduction	37 days	(154)

TABLE V (contd)
Recent examples of tsetse control operations

Control method	Country/region	Tsetse species	Type/density of traps/targets	Insecticide and dose rate	Odour baits/dose rate	Operational area	Reduction in tsetse density	Time taken	Comments (Reference)
Pour-ons (contd)	Zimbabwe, North	<i>G. morsitans</i>		1% deltamethrin pour-on every 4 weeks		11,667 cattle	Trypanosomiasis incidence fell to zero	6 months	(154)
SIT	Burkina Faso, Sideradougou	<i>G.p. gambiensis</i> <i>G. tachinoides</i>				3,000 km ²	100%	2 years	Populations were first reduced by 88.1% and 93.0% using deltamethrin-impregnated screens. Tsetse re-invasion was prevented with insecticide screens and traps (21)
	Nigeria	<i>G.p. palpalis</i>				1,500 km ²	100%	4-6 months	Populations were first reduced by 90% using traps and impregnated screens. SIT required at least 10 sterile:1 fertile (wild) fly (109)
Other integrated methods	Zimbabwe, Umfurdzi	<i>G.m. morsitans</i>	Targets at 4 /km ²	deltamethrin		1,500km ²	100%	9 months	Combined with aerial spraying, selective ground spraying, treatment of cattle with non-residual pesticides, chemoprophylaxis and the introduction of trypanotolerant N'Dama cattle (3, 153)

vmd : volume mean diameter

ai : active ingredient

SIT : sterile insect technique

eradication has been achieved in very few areas in Africa, most notably in northern Nigeria. It has been argued elsewhere (130) that this part of Nigeria is quite close to the distributional limits of tsetse in West Africa and therefore that fly suppression and eradication were therefore relatively easy in an ecological (but not logistical) sense. The increasing impact of humans in the north of Nigeria (undoubtedly exacerbated by severe droughts) has maintained eradication through the progressive elimination of the vegetation cover on which flies depend. In the absence of this 'agricultural prophylaxis' (36) it is quite possible that flies would have re-invaded the cleared areas from the south, where they still occur. Re-invasion is a persistent problem elsewhere, where local eradication has been claimed, but where more extensive areas cannot be cleared for logistical or political reasons (e.g. Zimbabwe).

The response to the re-invasion problem is two-fold. Firstly, fly-proof barriers are erected to protect the cleared areas. In this scheme, the cleared areas themselves are no longer protected by other means, as maintenance of eradication is believed to be possible using barriers alone. The second response accepts that re-invasion is an almost inevitable phenomenon, thus requiring continual suppression within the protected area. Under this scheme, targets and/or traps are permanently deployed both within and around the edge of the protected area, and these must therefore be maintained on a permanent basis.

In recent years, the acceptance that eradication is rarely achieved, or permanently maintained, has led to a re-examination of the role of hard-pressed governments in tsetse control; in many areas, this has involved effective privatisation of control activities. Privatisation usually passes under the guise of community participation schemes, where the local population is responsible (and in some cases actually pays) for the traps or targets in the area. The greatest progress in community schemes has been made by the poorest countries of Africa (or by the poorest areas in richer countries), where the option of government support is not available (30, 76, 77, 107). A notable feature of such schemes is that they do not occur spontaneously, but arise only through the long-term dedication of one individual or a very small group of research workers becoming involved in the problems of the local community. Whether the schemes will remain effective when the initiators have withdrawn remains to be seen (75). These examples of community participation schemes provide an opportunity for governments to devolve responsibility for tsetse control to those most affected by trypanosomiasis. However, effecting this change without totally abandoning peasant farmers to their fate remains a challenge. Regardless of the success of a community participation scheme, the community is unlikely to be sufficiently motivated to erect and maintain any protective barriers around a protected area. This will remain the responsibility of government services, which should not only monitor the success of each scheme but also select sites and erect barriers which most efficiently prevent re-invasion.

A further problem arises when responsibility for chemotherapy and chemoprophylaxis of cattle trypanosomiasis passes from the government to the private sector. Drug-resistant strains of trypanosomes are an increasingly widespread consequence of unregulated drug use.

Devolution of tsetse and trypanosomiasis control to individuals gradually favours new alternatives to traditional approaches. One of these is the use of 'pour-on' insecticides/acaricides applied directly to individual livestock animals. In the short term, these chemicals repel or kill flies attempting to feed on the treated animals, and in the long-term they reduce the fly population (6, 89, 154, 155). It may not be necessary to

apply the pour-on to all animals in a herd. The readiness of the individual cattle-owner to invest in control must determine the level of control used (perhaps varying seasonally with trypanosomiasis risk).

The examples in Table V show that almost all possible forms of tsetse control have been used in recent years. Although the frequency of aerial applications of insecticides has fallen considerably (see Fig. 4 presented by Vale [165]), there are still some situations (e.g. epidemic outbreaks of human sleeping sickness) where aerial applications have been the only means to reduce transmission quickly and effectively (65).

Understanding of the limits to tsetse distribution in Africa derived from the application of the methods described in this paper may also be used to 'tailor' fly control schemes to local conditions. As shown by the example of northern Nigeria, there will be some areas where only a small degree of control may be sufficient to limit fly numbers. In such areas, less than the presently recommended density of four screens or targets per square kilometre will be required against *morsitans* group species. Elsewhere, especially in areas where the flies obviously thrive (e.g. the Lambwe Valley in Kenya), more than the recommended target density will be required to reduce populations quickly. Another useful insight from the mapping exercise is the identification of areas which are apparently suitable for tsetse but which do not presently contain flies. These areas should be carefully surveyed, using modern methods incorporating bait technology, to confirm the absence of flies, and these areas should also be considered when barrier lines are being set out, as they may act as invasion corridors along which flies could move to re-occupy cleared areas.

CONCLUSION

Trypanosomiasis remains a threat to human and animal health and welfare in Africa. Much of the present poor performance of the agricultural sector can be attributed to the ever-present threat of animal trypanosomiasis in most of tropical Africa. Unfortunately, one of the solutions to the problem of poor agricultural production – an increase in the volume of livestock for milk, meat, manuring and draught power – is also often seen as part of the problem. In the past, livestock herds have been accused of causing displacement of settled arable farmers, land degradation and desertification. Livestock projects in Africa have met with so little economic success in the past that few are contemplated for the future.

These problems need to be re-addressed urgently. Careful field surveys over 1.5 million km² of Africa show that cattle herds belonging to nomadic pastoralists are associated with both habitation density and the percentage of land used for cultivation at all levels of rainfall (178). Far from competing with settled agriculturalists, cattle appear to be an integral part of the way in which humans survive in such marginal areas. Debate on the question of whether drought or cattle cause land degradation has recently been reanimated (58, 60, 179). Both sets of correspondents in this exchange have made pleas for a better understanding of the ecosystem dynamics of the areas where cattle are traditionally kept. No consensus exists among active field workers on the relationship between climate, vegetation, cattle and human populations in Africa.

Tsetse flies undoubtedly play an important role in determining the present distribution of cattle in Africa. The impact of flies is obviously changing, however, as humans change their own natural environments. On the one hand, population pressure on already occupied land is pushing humans and domestic animals towards marginal

areas, many of which are threatened by tsetse and trypanosomiasis; in the short term, disease risk will increase. On the other hand, the same population pressure is changing land-cover types, thereby reducing tsetse abundance; in the longer term, disease risk will decrease. Therefore, one certain prediction for the future is that occurrence of trypanosomiasis will change in both space and time in Africa. There is an urgent need to monitor these changes and alleviate their harmful effects. Careful, standardised data collection (as shown by the project in Togo) and sound interpretation should reveal the processes causing change. Rational resource management could then be implemented by exploring alternative developmental scenarios within predictive models of environmental development. Clearly, these models will be several orders of magnitude more complex than those for trypanosomiasis transmission or for tsetse fly populations, but the underlying principles remain the same: to describe the past, to explain this in terms of quantifiable processes and thence to predict the future. Neither the past nor the future is best served by misunderstanding or by ignorance.

ACKNOWLEDGEMENTS

The authors wish to thank the FAO/ARTEMIS programme for the provision of satellite imagery used in the analyses presented in these papers, especially F. Snijders and Dr J. Hielkema. The research work of D.J. Rogers is financed by the Overseas Development Administration through the Natural Resources Institute (Project X0239), with the support and encouragement of R. Allsopp, which is much appreciated. Other image-processing activities were carried out with equipment provided by the United Kingdom Natural Environment Research Council (Project GR3/7524). Dr S.E. Randolph kindly read and commented on the manuscript.

The authors also wish to acknowledge the Belgian Government, which finances the project GCP/TOG/013/BEL through the FAO, Rome, and especially Prof. J. Mortelmans, Dr R. De Deken (Tropical Institute, Antwerp), B. Hursey and Drs P. Marchot (FAO), M. Denke and T.B. Kagnaya (*Direction de l'Élevage et des Pêches*, Lomé).

The field results presented in this paper were gathered by an efficient and enthusiastic team including Drs A. Napala and B. Dao, and D. Batawui, A. Aliti, K. Amedji, S. Yacnambé, K. Agrignan, A. Body, L. Lakougnon and K. Kpanto.

LES GLOSSINES ET LEUR CONTRÔLE. – D.J. Rogers, G. Hendrickx et J.H.W. Slingenbergh.

Résumé : Les auteurs utilisent un modèle quantitatif pour décrire et étudier les caractéristiques biologiques de la mouche tsé-tsé (Glossina spp.) qui sont importantes pour déterminer le taux de transmission des trypanosomoses africaines entre les hôtes. Ils donnent des exemples de la contribution des recherches antérieures sur les glossines à une connaissance plus précise de l'épidémiologie et de l'épizootiologie, et ils indiquent les domaines où les connaissances sont encore insuffisantes, en vue d'études ultérieures.

Les variations spatiales et temporelles des risques sont des facteurs importants (mais rarement étudiés) de l'impact des trypanosomoses sur

l'homme, les animaux domestiques et les activités agricoles. Des enquêtes basées sur une grille d'échantillons, effectuées récemment au Togo, fournissent un précieux ensemble de données sur les glossines, le bétail et les trypanosomoses dans tout le pays.

Une combinaison de données météorologiques au sol et de télédétection par satellite dans le cadre de modèles linéaires d'analyse discriminante permet la description des distributions observées des cinq espèces de glossines existant au Togo, avec une précision allant de 72 % (Glossina palpalis et G. tachinoides) à 98 % (G. fusca). L'abondance des deux espèces les plus répandues, G. palpalis et G. tachinoides, est décrite avec une précision allant de 47 % à 83 %. Cela est particulièrement remarquable compte tenu des différences relativement modestes entre les valeurs moyennes des variables prédites par le modèle dans les zones où l'abondance des mouches varie.

Des analyses similaires pourraient être utilisées pour prévoir la présence et l'abondance des mouches dans d'autres zones non encore étudiées à ce jour, afin de programmer des campagnes de lutte contre les glossines ou d'étudier des options de développement.

Enfin, les auteurs donnent un bref compte-rendu des récentes campagnes de lutte contre les glossines. Le passage d'une stratégie basée sur l'éradication des mouches vers une stratégie basée sur leur simple contrôle est liée, selon les auteurs, au transfert de responsabilité de ces activités du gouvernement central aux régions et aux communautés locales, voire aux individus. Le rôle des gouvernements centraux restera cependant crucial pour déterminer les zones dans lesquelles seront mises en œuvre les différentes stratégies. Ces dernières auront pour objectif d'aider les communautés locales à contrôler les mouches puis à protéger les zones ainsi contrôlées d'une nouvelle invasion en provenance d'autres zones.

MOTS-CLÉS : Abondance des vecteurs de maladies – Analyse discriminante – Distribution des agents vecteurs – Epidémiologie – Glossina – Lutte contre les agents vecteurs – Modèles de population – Modèles de transmission des maladies – Mouche tsé-tsé – Télédétection par satellite – Togo – Trypanosomose.

LAS MOSCAS TSETSE Y SU CONTROL. – D.J. Rogers, G. Hendrickx y J.H.W. Slingenbergh.

Resumen: Los autores utilizan un cuadro de modelización cuantitativa para describir y estudiar las características biológicas de las moscas tsetse (Glossina spp.) que son importantes para determinar la tasa de transmisión de las tripanosomiasis africanas entre los huéspedes. Los autores presentan ejemplos del modo en que investigaciones anteriores sobre las moscas tsetse son utilizadas para un estudio epidemiológico y epizootiológico cuantitativo, pero también identifican los ámbitos en los que los conocimientos son insuficientes y que precisan ser estudiados en el futuro.

Las variaciones espaciales y temporales de los factores de riesgo han sido poco estudiadas hasta ahora, a pesar de su importancia, pues determinan notablemente el impacto de las tripanosomiasis en el hombre, los animales domésticos y las actividades agrícolas. Una encuesta reciente fue realizada en el

Togo; los muestreos provenían de todo el país dividido en cuadros geográficos, lo que permitió coleccionar una serie de datos de suma importancia sobre las moscas tsetse, el ganado y la tripanosomiasis.

*Combinando los datos meteorológicos obtenidos en el terreno y los obtenidos por teledetección satélite, en el marco de modelos analíticos lineares discriminantes, se puede describir la distribución observada de cinco especies de moscas tsetse presentes en el Togo, con un índice de exactitud comprendido entre 72% (*Glossina palpalis* y *G. tachinoides*) y 98% (*G. fusca*). Las clases de abundancia de las dos especies más difundidas, *G. palpalis* y *G. tachinoides*, son descritas con un índice de exactitud comprendido entre 47% y 83%. Estos resultados son sumamente notables si se considera las diferencias relativamente insignificantes entre los valores medios de las variables predichas en las zonas donde los niveles de abundancia fluctúan.*

Análisis similares podrían ser desarrollados para predecir la frecuencia y la abundancia de moscas en otras regiones que no han sido hasta ahora objeto de investigaciones, de manera a poder planificar programas de control de moscas tsetse o examinar alternativas de desarrollo.

Finalmente, los autores pasan revista a algunos programas recientes de control de moscas tsetse. Los autores explican que la tendencia actual de remplazar las estrategias de erradicación de las moscas por estrategias de control está ligada al hecho que el gobierno central ha delegado la responsabilidad de estas actividades a las regiones, a las comunidades locales e incluso a los individuos. El papel del gobierno central seguirá sin embargo siendo crucial, pues le incumbirá determinar las zonas en las que se aplicarán las distintas opciones de control, facilitar las actividades de control asumidas por las comunidades locales y proteger las zonas controladas contra reinvasiones de moscas desde otras zonas.

PALABRAS CLAVE: Abundancia de vectores – Análisis discriminante – Control de vectores – Distribución de vectores – Epidemiología – *Glossina* – Modelos de poblaciones – Modelos de transmisión de enfermedades – Moscas tsetse – Teledetección por satélite – Togo – Tripanosomiasis.

REFERENCES

1. ALLSOPP R. (1984). – Control of tsetse flies (Diptera: Glossinidae) using insecticides: a review and future prospects. *Bull. entomol. Res.*, **74**, 1-23.
2. ANON. (1992). – Assainir les zones infestées de glossines. Une lutte économique et non polluante. *Afr. Agric.*, **190**, 34-40.
3. ANON. (1992). – Lutte intégrée contre les trypanosomes et les vecteurs. *Afr. Agric.*, **197**, 44-45.
4. BAKER J.R. & ROBERTSON D.H.H. (1957). – An experiment on the infectivity to *Glossina morsitans* of a strain of *Trypanosoma rhodesiense* and of a strain of *T. brucei*, with some observations on the longevity of infected flies. *Ann. trop. Med. Parasitol.*, **51** (2), 121-135.
5. BARTHOLOME E. (1988). – Radiometric measurement and crop yield forecasting: some observations over millet and sorghum experimental plots in Mali. *Int. J. remote Sensing*, **9**, 1539-1552.

6. BAUER B., KABORE I., LIEBISCH A., MEYER F. & PETRICH-BAUER J. (1992). – Simultaneous control of ticks and tsetse flies in Satiri, Burkina Faso, by the use of flumethrin pour on for cattle. *Trop. Med. Parasitol.*, **43** (1), 41-46.
7. BOGNER F. (1992). – Response properties of CO₂-sensitive receptors in tsetse flies (Diptera: *Glossina palpalis*). *Physiol. Entomol.*, **17** (1), 19-24.
8. BOURN D. (1983). – Tsetse control, agricultural expansion and environmental change in Nigeria. D.Phil. Thesis, Oxford, 250 pp.
9. BRADY J. (1991). – Successful control of tsetse flies using attractants. *Am. J. trop. Med. Hyg.*, **45** (Suppl. 3), 288-289.
10. BRADY J. & GRIFFITHS N. (1993). – Upwind flight responses of tsetse flies (*Glossina* spp.) (Diptera: Glossinidae). *Bull. entomol. Res.*, **83** (3), 329-333.
11. BURSELL E. (1960). – The effect of temperature on the consumption of fat during pupal development in *Glossina*. *Bull. entomol. Res.*, **51**, 583-598.
12. BURSELL E. (1961). – The behaviour of tsetse flies (*Glossina swynnertoni* Austen) in relation to problems of sampling. *Proc. R. entomol. Soc. Lond.*, (A), **36**, 9-20.
13. BURSELL E. (1990). – The effect of host odour on the landing responses of tsetse flies (*Glossina morsitans morsitans*) in a wind tunnel with and without visual targets. *Physiol. Entomol.*, **15** (4), 369-376.
14. BURSELL E., BILLING K.C., HARGROVE J.W., MCCABE C.T. & SLACK E. (1974). – Metabolism of the bloodmeal in tsetse flies (a review). *Acta trop.*, **31**, 297-320.
15. BURSELL E. & TAYLOR P. (1980). – An energy budget for *Glossina* (Diptera: Glossinidae). *Bull. entomol. Res.*, **70**, 187-196.
16. BUXTON P.A. & LEWIS D.J. (1934). – Climate and tsetse flies: laboratory studies upon *Glossina submorsitans* and *tachinoides*. *Phil. Trans. R. Soc. Lond.*, (B), **224**, 175-240.
17. CAUGHLEY G. (1977). – Analysis of vertebrate populations. John Wiley & Sons, Chichester, 234 pp.
18. CHALLIER A. & LAVEISSIERE C. (1973). – Un nouveau piège pour la capture des glossines (*Glossina* : Diptera, Muscidae) : description et essais sur le terrain. *Cah. ORSTOM, Série Entomol. méd. Parasitol.*, **11** (4), 251-262.
19. CHORLEY C.W. (1933). – Traps for tsetse flies of the 'crinoline' and 'ventilator' forms. *Bull. entomol. Res.*, **24**, 315-317.
20. CHORLEY T.W. (1948). – *Glossina pallidipes* Austen attracted by the scent of cattle-dung and urine (Diptera). *Proc. R. entomol. Soc. Lond.* (A), **23**, 9-11.
21. CLAIR M., CUISANCE D., POLITZAR H., MEROT P. & BAUER B. (1990). – Tsetse fly eradication in Burkina Faso and evaluation of traps and targets. In Sterile insect technique for tsetse control and eradication. Vom, Plateau State, Nigeria, 6-10 June 1988. Joint FAO/IAEA Division of Nuclear Techniques in Food and Agriculture, Vienna, 31-43.
22. COLVIN J. & GIBSON G. (1992). – Host-seeking behavior and management of tsetse. *Annu. Rev. Entomol.*, **37**, 21-40.
23. COULIBALY L., DIARRASOUBA I., D'ETEREN G.D.M., GNIHAN N., HECKER P., ITY P., KUPPER W., LEAK S.G.A., NAGDA S.M., RARIEYA J.M., SCHUETTERLE A., THORPE W., TRAIL J.C.M. & TRAUB D. (1991). – Effect of tsetse control by means of insecticide impregnated biconical traps on health and productivity of livestock in northern Côte d'Ivoire. In Twentieth Meeting of the International Scientific Council for Trypanosomiasis Research and Control. Mombasa, Kenya, 10-14 April 1989. Organisation of African Unity/Scientific, Technical and Research Commission, Nairobi, 445-447.

24. CUISANCE D., GOUTEUX J.P., CAILTON P., KOTA-GUINZA A., NDOKOUE F., POUNEKROZOU E. & DEMBA D. (1992). – Problématique d'une lutte contre les glossines pour la protection d'élevage zébu en République centrafricaine. *Mémoires Soc. roy. belge d'Entomol.*, **35**, 103-110.
25. D'AMICO F., MOUSSA A., SARDA J. & GOUTEUX J.P. (1992). – Distribution et importance des gîtes à *Glossina fuscipes* Newstead, 1910 dans l'agglomération de Bangui (République centrafricaine). *Bull. Soc. Pathol. exot. Filiales*, **85** (1), 64-68.
26. DOUA F. & BOA YAPO F. (1993). – Human trypanosomiasis in the Ivory Coast: therapy and problems. *Acta trop.*, **54** (3-4), 163-168.
27. DOUATI A. (1988). – Bilan de quatre années de lutte antiglossinaire aux moyens de pièges et écrans au nord de la Côte d'Ivoire. In Nineteenth Meeting of the International Scientific Council for Trypanosomiasis Research and Control. Lomé, Togo, 30 March-3 April 1987. Organisation of African Unity/Scientific, Technical and Research Commission, Nairobi, 506-508.
28. DRANSFIELD R.D., BRIGHTWELL R., KIILU J., CHAUDHURY M.F. & ADABIE D.A. (1989). – Size and mortality rates of *Glossina pallidipes* in the semi-arid zone of southwestern Kenya. *Med. vet. Entomol.*, **3**, 83-95.
29. DRANSFIELD R.D., BRIGHTWELL R., KYORKU C. & WILLIAMS B. (1990). – Control of tsetse fly (Diptera: Glossinidae) populations using traps at Nguruman, south-west Kenya. *Bull. entomol. Res.*, **80**, 265-276.
30. DRANSFIELD R.D., WILLIAMS B.G. & BRIGHTWELL R. (1991). – Control of tsetse flies and trypanosomiasis: myth or reality? *Parasitol. Today*, **7** (10), 287-291.
31. DUGGAN A.J. (1970). – An historical perspective. In The African trypanosomiasis (H.W. Mulligan, ed.). Ministry of Overseas Development, London; George Allen & Unwin, London, xli-lxxxviii.
32. FAIRBAIRN H. & CULWICK A.T. (1950). – Some climatic factors influencing populations of *Glossina swynnertoni*. *Ann. trop. Med. Parasitol.*, **44**, 27-33.
33. FOOD AND AGRICULTURE ORGANISATION OF THE UNITED NATIONS (FAO) (1992). – Programme for the control of African animal trypanosomiasis and related development. Proceedings of the FAO Panel of Experts, Harare, Zimbabwe, 24-26 June 1991. FAO, Rome, 203 pp.
34. FOOD AND AGRICULTURE ORGANISATION OF THE UNITED NATIONS (FAO) (1992). – Training manual for tsetse control personnel, Vol. 4. Use of attractive devices for tsetse survey and control. FAO, Rome, 196 pp.
35. FOOD AND AGRICULTURE ORGANISATION OF THE UNITED NATIONS (FAO) (1993). – Training manual for tsetse control personnel, Vol. 5. Insecticides for tsetse and trypanosomiasis control using attractive bait techniques. FAO, Rome, 88 pp.
36. FORD J. (1971). – The role of the trypanosomiasis in African ecology: a study of the tsetse fly problem. Clarendon Press, Oxford, 568 pp.
37. FORD J. & LEGGATE B.M. (1961). – The geographical and climatic distribution of trypanosome infection rates in *G. morsitans* group of tsetse-flies. *Trans. R. Soc. trop. Med. Hyg.*, **55** (4), 383-397.
38. FORD J. & KATONDO K.M. (1977). – The distribution of tsetse flies in Africa. Organisation of African Unity, Nairobi; Cook, Hammond & Kell, London, 3 sheets.
39. FREZIL J.-L., EOUZAN J.-P., ALARY J.-C., MALONGA J.-R. & GINOUX P.Y. (1980). – Epidémiologie de la trypanosomiase humaine en République populaire du Congo. *Cah. ORSTOM, Série Entomol. méd. Parasitol.*, **18**, 329-346.

40. GARRETT-JONES C. (1964). – The human blood index of malaria vectors in relation to epidemiological assessment. *Bull. Wld Hlth Org.*, **30**, 241-261.
41. GINGRICH J.B., WARD R.A., MACKEN L.M. & ESSER K.M. (1982). – African sleeping sickness: new evidence that mature flies (*Glossina morsitans*) can become potent vectors. *Trans. R. Soc. trop. Med. Hyg.*, **76**, 479-481.
42. GINGRICH J.B., WARD R.A., MACKEN L.M. & SCHOENBECHLER M.J. (1982). – *Trypanosoma brucei rhodesiense* (Trypanosomatidae): factors influencing infection rates of a recent human isolate in the tsetse *Glossina morsitans* (Diptera, Glossinidae). *J. med. Entomol.*, **19**, 268-274.
43. GLASGOW J.P. (1961). – The feeding habits of *Glossina swynnertoni* Austen. *J. Anim. Ecol.*, **30**, 77-85.
44. GLASGOW J.P. (1963). – The distribution and abundance of tsetse. Pergamon Press, Oxford, 241 pp.
45. GLASGOW J.P. & POTTS W.H. (1970). – Control by hand-catching and traps. In *The African trypanosomiasis* (H.W. Mulligan, ed.). George Allen & Unwin, London, 456-463.
46. GOODING R.H. (1992). – Genetic variation in tsetse flies and implications for trypanosomiasis. *Parasitol. Today*, **8** (3), 92-95.
47. GOUTEUX J., NOIREAU F., SINDA D. & FREZIL J.L. (1991). – L'utilisation du piège pyramidal par les communautés rurales dans la lutte contre la maladie du sommeil. Bilan des essais réalisés au Congo. In *Twentieth Meeting of the International Scientific Council for Trypanosomiasis Research and Control*. Mombasa, Kenya, 10-14 April 1989. Organisation of African Unity/Scientific, Technical and Research Commission, Nairobi, 428-435.
48. GOUTEUX J.P. & LE GALL F. (1992). – Piège bipyramidal à tsé-tsé pour la protection de l'élevage en République centrafricaine. *Wld Anim. Rev.*, **70/71**, 37-43.
49. GOUTEUX J.P., KOUNDA GBOUMBI J.C., NOUTOUA L., D'AMICO F., BAILLY C. & ROUNGOU J.B. (1993). – Man-fly contact in the Gambian trypanosomiasis focus of Nola-Bilolo (Central African Republic). *Trop. Med. Parasitol.*, **44** (3), 213-218.
50. GREEN C.H. (1994). – Bait methods for tsetse control. *Adv. Parasitol.*, **34**, 229-291.
51. GREEN P.E. (1978). – Analyzing multivariate data. The Dryden Press, Hinsdale, Illinois, 519 pp.
52. GRUNDLER G.H.M. & DOUATI A. (1993). – Méthode pluridisciplinaire de lutte antiglossinaire en Côte d'Ivoire. In *Twenty-first Meeting of the International Scientific Council for Trypanosomiasis Research and Control*. Yamoussoukro, Côte d'Ivoire, 21-25 October 1991. Organisation of African Unity/Scientific, Technical and Research Commission, Nairobi, 207.
53. GUEDEGBE B., VERHULST A., VAN MEIRVENNE N., PANDEY V.S. & DOKO A. (1992). – Indications sérologiques de l'existence d'un réservoir sauvage du *Trypanosoma brucei gambiense* dans la réserve de la biosphère de la Pendjari en République du Bénin. *Ann. Soc. belge Méd. trop.*, **72** (2), 113-120.
54. HABTEMARIAM T., RUPPANNER R., RIEMANN H.P. & THEIS J.H. (1983). – An epidemiologic systems analysis model for African trypanosomiasis. *Prev. vet. Med.*, **1**, 125-136.
55. HABTEMARIAM T., RUPPANNER R., RIEMANN H.P. & THEIS J.H. (1983). – Epidemic and endemic characteristics of trypanosomiasis in cattle: a simulation model. *Prev. vet. Med.*, **1**, 137-145.
56. HARGROVE J.W. & LANGLEY P.A. (1990). – Sterilizing tsetse (Diptera: Glossinidae) in the field: a successful trial. *Bull. entomol. Res.*, **80**, 397-403.

57. HARGROVE J.W. & PACKER M.J. (1993). – Nutritional states of male tsetse flies (*Glossina* spp.) (Diptera: Glossinidae) caught in odour-baited traps and artificial refuges: models for feeding and digestion. *Bull. entomol. Res.*, **83** (1), 29-46.
58. HIERNAX P. (1994). – Hiernaux's answer. *ILCA Newsl.*, **13** (2), 5-6.
59. HOARE C.A. (1972). – The trypanosomes of mammals: a zoological monograph. Blackwell Scientific Publications, Oxford, 749 pp.
60. INTERNATIONAL LIVESTOCK CENTRE FOR AFRICA (ILCA) (1993). – Droughts, not animals, degrade Sahelian rangelands. *ILCA Newsl.*, **12** (4), 6.
61. JACKSON C.H.N. (1944). – The analysis of a tsetse fly population. II. *Ann. Eugenics, Camb.*, **12**, 176-205.
62. JENNI L., MARTI S., SCHWEITZER J., BETSCHAT B., LE PAGE R.W.F., WELLS J.M., TAIT A., PAINDOVOINE P., PAYS E. & STEINERT M. (1986). – Hybrid formation between African trypanosomes during cyclical transmission. *Nature*, **322**, 173-175.
63. JORDAN A.M. (1986). – Trypanosomiasis control and African rural development. Longman, London, 357 pp.
64. KAGERUKA P. (1992). – Concepts sur l'existence du réservoir animal de *Trypanosoma brucei gambiense*. In Rapport final du séminaire de modélisation appliquée pour l'optimisation des prises de décisions et du suivi des programmes de contrôle de la maladie du sommeil (J.D.F. Habbema & A.D. Muynck, eds). Daloa, Côte d'Ivoire, 28-30 October 1991. Erasmus University, Rotterdam, 39-44.
65. KANGWAGYE T.N. & LATIGO A.A. (1991). – *Glossina fuscipes* control by aerial spraying in the E. Uganda Rhodesian trypanosomiasis epidemic. In Twentieth Meeting of the International Scientific Council for Trypanosomiasis Research and Control. Mombasa, Kenya, 10-14 April 1989. Organisation of African Unity/Scientific, Technical and Research Commission, Nairobi, 415.
66. KEMP P.B. (1951). – Field observations on the activity of *Pheidole*. *Bull. entomol. Res.*, **42** (1), 201-206.
67. KNOLS B.G.J., WILLEMSE L., FLINT S. & MATE A. (1993). – A trial to control the tsetse fly, *Glossina morsitans centralis*, with low densities of odour-baited targets in west Zambia. *Med. vet. Entomol.*, **7** (2), 161-169.
68. KUPPER W., SPATH J. & KROBER T. (1991). – Attractiveness of chemicals to *Glossina tachinoides* Westwood (Diptera, Glossinidae) in Côte d'Ivoire. *Trop. Pest Manag.*, **37** (4), 436-438.
69. LANCIEN J. (1991). – Lutte contre la maladie du sommeil dans le sud-est Ouganda par piégeage des glossines. *Ann. Soc. belge Méd. trop.*, **71** (Suppl. 1), 35-47.
70. LANCIEN J., EOZAN J.-P., FREZIL J.-L. & MOUCHET J. (1981). – Elimination des glossines par piégeage dans deux foyers de trypanosomiase en République populaire du Congo. *Cah. ORSTOM, Série Entomol. méd. Parasitol.*, **19** (4), 239-246.
71. LANCIEN J., MUGUWA J., BOUTES B. & LANNES C. (1991). – Contraintes scientifiques et logistiques pour un vaste programme national réaliste de piégeage des glossines riveraines : l'exemple de l'Ouganda. In Twentieth Meeting of the International Scientific Council for Trypanosomiasis Research and Control. Mombasa, Kenya, 10-14 April 1989. Organisation of African Unity/Scientific, Technical and Research Commission, Nairobi, 413-414.
72. LANGLEY P.A., FELTON T., STAFFORD K. & OOUCHI H. (1990). – Formulation of pyriproxyfen, a juvenile hormone mimic, for tsetse control. *Med. vet. Entomol.*, **4**, 127-133.

73. LANGLEY P.A., HARGROVE J.W., MAUCHAMP B., ROYER C. & OOUCHI H. (1993). – Prospects for using pyriproxifen-treated targets for tsetse control. *Entomol. exp. Appl.*, **66** (2), 153-159.
74. LAVEISSIERE C. (1977). – Ecologie de *Glossina tachinoides* Westwood, 1850, en savane humide d'Afrique de l'Ouest. III. Etat alimentaire d'une population. *Cah. ORSTOM, Série Entomol. méd. Parasitol.*, **15** (4), 331-337.
75. LAVEISSIERE C., GREBAUT P., LEMASSON J., MEDA H., COURET D., DOUA F. & BROU N. (1990). – Les communautés rurales et la lutte contre la maladie du sommeil en forêt de Côte d'Ivoire. OCCGE/ORSTOM, Institut Pierre-Richet, Paris, 135 pp.
76. LAVEISSIERE C., VALE G.A. & GOUTEUX J.-P. (1991). – Bait methods for tsetse control. *In* Control of disease vectors in the community (C.F. Curtis, ed.). Wolfe Publishing, London, 47-74.
77. LAVEISSIERE C., GREBAUT P. & LEMASSON J. (1991). – Trypanosomiase humaine africaine : la lutte antivectorielle confiée aux paysans. *In* Twentieth Meeting of the International Scientific Council for Trypanosomiasis Research and Control. Mombasa, Kenya, 10-14 April 1989. Organisation of African Unity/Scientific, Technical and Research Commission, Nairobi, 223-232.
78. LAVEISSIERE C. & MEDA H.H. (1992). – La lutte par piégeage contre la maladie du sommeil : pas aussi simple que l'on croit ! *Ann. Soc. belge Méd. trop.*, **72** (Suppl. 1), 57-68.
79. LEAK S.G.A., COLARDELLE C., D'ETEREN G., DUMONT P., FERON A., JEANNIN P., MNENGU M., MULUNGU M., NGAMUNA S., ORDNER G., SAUVEROCHE B., TRAIL J.C.M. & YANGARI G. (1991). – *Glossina fusca* group tsetse vectors of cattle trypanosomiasis in Gabon and Zaïre. *Med. vet. Entomol.*, **5** (1), 111-120.
80. LINTHICUM K.J., BAILEY C.L., TUCKER C.J., ANGLEBERGER D.R., CANNON T., LOGAN T.M., GIBBS P.H. & NICKESON J. (1991). – Towards real-time prediction of Rift Valley fever epidemics in Africa. *Prev. vet. Méd.*, **11** (3-4), 325-334.
81. MACDONALD G. (1957). – The epidemiology and control of malaria. Oxford University Press, London, 201 pp.
82. MAKUMI J.N. & MOLOO S.K. (1991). – *Trypanosoma vivax* in *Glossina palpalis gambiensis* do not appear to affect feeding behaviour, longevity or reproductive performance of the vector. *Med. vet. Entomol.*, **5** (1), 35-42.
83. MANSINGA D.M., MILORD F., ETHIER L. & POCHEA A. (1991). – Essai d'élimination des glossines par piégeage à Nioki, au Zaïre. *In* Twentieth Meeting of the International Scientific Council for Trypanosomiasis Research and Control. Mombasa, Kenya, 10-14 April 1989. Organisation of African Unity/Scientific, Technical and Research Commission, Nairobi, 427.
84. MAUDLIN I. (1985). – Inheritance of susceptibility to trypanosomes in tsetse flies. *Parasitol. Today*, **1**, 59-60.
85. MAUDLIN I. (1991). – Transmission of African trypanosomiasis: interactions among tsetse immune system, symbionts, and parasites. *Adv. Dis. Vector Res.*, **7**, 117-148.
86. MAWUENA K. & YACNAMBE S. (1988). – L'utilisation des pièges et écrans imprégnés d'insecticides pour la lutte contre la trypanosomiase animale. *Trypanotolérance et Production*, **5**, 85-92.
87. MAWUENA K. & YACNAMBE M.S. (1990). – Emploi de pièges et d'écrans dans la lutte contre la trypanosomiase animale au Togo. *Tropicicultura*, **8** (1), 40-43.
88. MEROT P. & FILLEDIER J. (1993). – Attractifs olfactifs pour les glossines riveraines : bilan de cinq années de recherches. *In* Twenty-first Meeting of the International Scientific

Council for Trypanosomiasis Research and Control. Yamoussoukro, Côte d'Ivoire, 21-25 October 1991. Organisation of African Unity/Scientific, Technical and Research Commission, Nairobi, 197.

89. MEYER F., BAUER B., KABORE I. & LIEBISCH A. (1991). – Simultaneous control of tsetse flies and ticks by the application of flumethrin pour-on on cattle in Satiri, Burkina Faso. *Trop. Med. Parasitol.*, **42** (4), 451.
90. MIHOK S., OLUBAYO R.O. & WESONGA D.F. (1991). – Infection rates in *Glossina morsitans morsitans* fed on waterbuck and Boran cattle infected with *Trypanosoma congolense*. *Acta trop.*, **49** (3), 185-191.
91. MILLIGAN P.J.M. & BAKER R.D. (1988). – A model of tsetse-transmitted animal trypanosomiasis. *Parasitology*, **96**, 211-239.
92. MOLOO S.K. (1994). – The distribution of *Glossina* species in Africa and their natural hosts. *Insect Sci. Appl.*, **14** (4), 511-527.
93. MOLYNEUX D.H. & ASHFORD R.W. (1983). – The biology of *Trypanosoma* and *Leishmania* parasites of man and domestic animals. Taylor & Francis, London, 294 pp.
94. MOLYNEUX D.H. & STILES J.K. (1991). – Trypanosomatid-vector interactions. *Ann. Soc. belge Méd. trop.*, **71** (Suppl. 1), 151-166.
95. MORRIS K.R.S. (1946). – The control of trypanosomiasis by entomological means. *Bull. entomol. Res.*, **37**, 201-250.
96. MORRIS K.R.S. & MORRIS M.G. (1949). – The use of traps against tsetse in West Africa. *Bull. entomol. Res.*, **39**, 491-528.
97. MORTELMANS J. (1986). – Quelques aspects économiques en rapport avec la parasitologie vétérinaire. *Tropicultura*, **4** (3), 112-116.
98. MUIRHEAD-THOMSON R.C. (1991). – Trap responses of flying insects: the influence of trap design on capture efficiency. Academic Press, London, 287 pp.
99. MURRAY M., MORRISON W.I. & WHITELAW D.D. (1982). – Host susceptibility to African trypanosomiasis: trypanotolerance. *Adv. Parasitol.*, **21**, 1-68.
100. MURRAY M., STEAR M.J., TRAIL J.C.M., D'ETEREN G.D.M., AGYEMANG K. & DWINGER R.H. (1990). – Trypanosomiasis in cattle: prospects for control. In *Breeding for disease resistance in farm animals* (J.B. Owen & R.F.E. Axford, eds). Bangor, United Kingdom, 13-14 September. CAB International, Wallingford, United Kingdom, 203-223.
101. MWANGELWA M.I., DRANSFIELD R.D. & OTIENO L.H. (1991). – The response of *Glossina fuscipes fuscipes* Newstead to odour baits and trap types on Rusinga Island, Kenya. In *Twentieth Meeting of the International Scientific Council for Trypanosomiasis Research and Control*. Mombasa, Kenya, 10-14 April 1989. Organisation of African Unity/Scientific, Technical and Research Commission, Nairobi, 519-519.
102. MWANGI E.K., STEVENSON P., MURRAY M. & GETTINBY G. (1993). – Susceptibility of three cattle breeds to trypanosomiasis under natural tsetse challenge in Nguruman, south western Kenya. In *Twenty-first Meeting of the International Scientific Council for Trypanosomiasis Research and Control*. Yamoussoukro, Côte d'Ivoire, 21-25 October 1991. Organisation of African Unity/Scientific, Technical and Research Commission, Nairobi, 163-164.
103. NASH T.A.M. (1937). – Climate, the vital factor in the ecology of *Glossina*. *Bull. entomol. Res.*, **28**, 75-127.

104. NDEGWA P.N., IRUNGU L.W. & MOLOO S.K. (1992). – Effect of puparia incubation temperature: increased infection rates of *Trypanosoma congolense* in *Glossina morsitans centralis*, *G. fuscipes fuscipes* and *G. brevipalpis*. *Med. vet. Entomol.*, **6** (2), 127-130.
105. NEILL G.E. (1987). – Campagne pour combattre la mouche tsé-tsé dans la région de Nasho. *Bull. agric. Rwanda*, **20** (3), 217-221.
106. NITCHEMAN S. (1988). – Comparaison des longévités des glossines (*Glossina morsitans morsitans* Westwood, 1850) infectées par les trypanosomes (*Trypanosoma nannomonas congolense* Broden, 1904) et des glossines saines. *Ann. Parasitol. hum. comp.*, **63** (2), 163-164.
107. OKOTH J.O., KIRUMIRA E.K. & KAPAATA R. (1991). – A new approach to community participation in tsetse control in the Busoga sleeping sickness focus, Uganda. A preliminary report. *Ann. trop. Med. Parasitol.*, **85** (3), 315-322.
108. OKOTH J.O., OKETHI V. & OGOLA A. (1991). – Control of tsetse and trypanosomiasis transmission in Uganda by applications of lambda-cyhalothrin. *Med. vet. Entomol.*, **5** (1), 121-128.
109. OLADUNMADE M.A., FELDMAN U., TAKKEN W., TENABE S.O., HAMANN H., ONAH J., DENGWAT L., VLOEDT A.M.V. VAN DER & GINGRICH R.E. (1990). – Eradication of *Glossina palpalis palpalis* (Robineau-Desvoidy) (Diptera: Glossinidae) from agropastoral land in central Nigeria by means of the sterile insect technique. In Sterile insect technique for tsetse control and eradication. Vom, Plateau State, Nigeria, 6-10 June 1988. Joint FAO/IAEA Division of Nuclear Techniques in Food and Agriculture, Vienna, 5-23.
110. OLOO F.P. & KINYANJUI K. (1983). – Field trials of deltamethrin and cypermethrin for tsetse control. *Kenya Vet.*, **7** (2), 21-22.
111. O'NEILL S.L., GOODING R.H. & AKSOY S. (1993). – Phylogenetically distinct microorganisms reside in *Glossina* midgut and ovary tissues. *Med. vet. Entomol.*, **7** (4), 377-383.
112. OPIYO E., DOLAN R.B., NJOGU A.R., SAYER P.D. & MGUTU S.P. (1988). – Tsetse control on Galana Ranch. In Nineteenth Meeting of the International Scientific Council for Trypanosomiasis Research and Control. Lomé, Togo, 30 March-3 April 1987. Organisation of African Unity/Scientific, Technical and Research Commission, Nairobi, 434-437.
113. OTESILE P.A., AKPOKODJE J.U. & EKWURUKE J.O. (1991). – Performance of a herd of Ndama cattle during and after cessation of chemoprophylaxis for trypanosomiasis. *Trop. Vet.*, **9** (1-2), 51-54.
114. OTTER C.J. DEN (1991). – Olfactory responses of tsetse flies to phenols from buffalo urine. *Physiol. Entomol.*, **16** (4), 401-410.
115. OTTER C.J. DEN & GOES VAN NATERS W.M. VAN DER (1992). – Single cell recordings from tsetse (*Glossina m. morsitans*) antennae reveal olfactory, mechano- and cold receptors. *Physiol. Entomol.*, **17** (1), 33-42.
116. OTTER C.J. DEN & GOES VAN NATERS W.M. VAN DER (1993). – Responses of individual antennal olfactory cells of tsetse flies (*Glossina m. morsitans*) to phenols from cattle urine. *Physiol. Entomol.*, **18** (1), 43-49.
117. PAYNTER Q. & BRADY J. (1993). – Flight responses of tsetse flies (*Glossina*) to octenol and acetone vapour in a wind-tunnel. *Physiol. Entomol.*, **18** (1), 102-108.
118. PHELPS R.J. & CLARKE G.P.Y. (1974). – Seasonal elimination of some size classes in males of *Glossina morsitans morsitans* Westw. (Diptera, Glossinidae). *Bull. entomol. Res.*, **64**, 313-324.

119. PNUD (PROGRAMME DES NATIONS UNIES POUR LE DEVELOPPEMENT) (1984). – Atlas de développement régional du Togo. Editog, Lomé, 207 pp.
120. PRINCE S.D., JUSTICE C.O. & LOS S.O. (1990). – Remote sensing of the Sahelian environment: a review of the current status and future prospects. Commission of the European Communities. Directorate General VIII. Joint Research Centre, Ispra Establishment. Technical Centre for Agricultural and Rural Cooperation, Brussels, 128 pp.
121. RANDOLPH S.E. & ROGERS D.J. (1978). – Feeding cycles and flight activity in field populations of tsetse (Diptera: Glossinidae). *Bull. entomol. Res.*, **68**, 655-671.
122. RANDOLPH S.E., ROGERS D.J. & KUZOE F.A.S. (1984). – Local variation in the population dynamics of *Glossina palpalis palpalis* (Robineau-Desvoidy) (Diptera: Glossinidae). II. The effect of insecticidal spray programmes. *Bull. entomol. Res.*, **74**, 425-438.
123. RANDOLPH S.E., DRANSFIELD R.D. & ROGERS D.J. (1989). – Effect of host odours on trap catch composition of *Glossina pallidipes* in Kenya. *Med. vet. Entomol.*, **3**, 297-306.
124. RANDOLPH S.E., ROGERS D.J., DRANSFIELD R.D. & BRIGHTWELL R. (1991). – Trap-catches, nutritional condition and the timing of activity of the tsetse fly *Glossina longipennis* (Diptera: Glossinidae). *Bull. entomol. Res.*, **81** (4), 455-464.
125. RANDOLPH S.E., ROGERS D.J. & KIILU J. (1991). – The feeding behaviour, activity and trappability of wild female *Glossina pallidipes* in relation to their pregnancy cycle. *Med. vet. Entomol.*, **5** (3), 335-350.
126. RANDOLPH S.E., WILLIAMS B.G., ROGERS D.J. & CONNOR H. (1992). – Modelling the effect of feeding-related mortality on the feeding strategy of tsetse (Diptera: Glossinidae). *Med. vet. Entomol.*, **6**, 231-240.
127. RICKER W.E. (1954). – Stock and recruitment. *J. Fish. Res. Bd Can.*, **11**, 559-623.
128. ROGERS D.J. (1974). – Ecology of *Glossina*: natural regulation and movement of tsetse fly populations. In Paper presented at a Symposium on Tsetse Flies and Trypanosomiasis organised by IEMVT (French Institute for Breeding and Veterinary Medicine in Tropical Countries). Maisons-Alfort, 12-15 March. IEMVT, Paris, 35-38.
129. ROGERS D.J. (1977). – Study of a natural population of *Glossina fuscipes fuscipes* Newstead and a model of fly movement. *J. Anim. Ecol.*, **46**, 281-307.
130. ROGERS D.J. (1979). – Tsetse population dynamics and distribution: a new analytical approach. *J. Anim. Ecol.*, **48**, 825-849.
131. ROGERS D.J. (1983). – Interpretation of sample data. In Pest and vector management in the tropics (A. Youdeowei & M.W. Service, eds). Longman, London & New York, 139-160.
132. ROGERS D.J. (1984). – The estimation of sampling biases for male tsetse. *Insect Sci. Appl.*, **5** (5), 369-373.
133. ROGERS D.J. (1985). – Trypanosomiasis 'risk' or 'challenge': a review. *Acta trop.*, **45**, 5-23.
134. ROGERS D.J. (1988). – A general model for the African trypanosomiasis. *Parasitology*, **97**, 193-212.
135. ROGERS D.J. (1988). – The dynamics of vector-transmitted diseases in human communities. *Phil. Trans. R. Soc. Lond., (B)*, **321**, 513-539.
136. ROGERS D.J. (1990). – A general model for tsetse populations. *Insect Sci. Appl.*, **11** (3), 331-346.

137. ROGERS D.J. (1991). – Satellite imagery, tsetse and trypanosomiasis in Africa. *Prev. vet. Med.*, **11** (3-4), 201-220.
138. ROGERS D.J. & HUBBARD S. (1974). – How the behaviour of parasites and predators promotes population stability. In *Ecological stability* (M.B. Usher & M.H. Williamson, eds). Chapman & Hall, London, 99-119.
139. ROGERS D.J. & RANDOLPH S.E. (1978). – Metabolic strategies of male and female tsetse (Diptera: Glossinidae) in the field. *Bull. entomol. Res.*, **68**, 639-654.
140. ROGERS D.J. & RANDOLPH S.E. (1984). – A review of the density-dependent processes in tsetse populations. *Insect Sci. Appl.*, **5** (5), 397-402.
141. ROGERS D.J., RANDOLPH S.E. & KUZOE F.A.S. (1984). – Local variation in the population dynamics of *Glossina palpalis palpalis* (Robineau-Desvoidy) (Diptera: Glossinidae). I. Natural population regulation. *Bull. entomol. Res.*, **74**, 403-423.
142. ROGERS D.J. & RANDOLPH S.E. (1985). – Population ecology of tsetse. *Annu. Rev. Entomol.*, **30**, 197-216.
143. ROGERS D.J. & RANDOLPH S.E. (1990). – Estimation of rates of predation on tsetse. *Med. vet. Entomol.*, **4**, 195-204.
144. ROGERS D.J. & RANDOLPH S.E. (1991). – Mortality rates and population density of tsetse flies correlated with satellite imagery. *Nature*, **351**, 739-741.
145. ROGERS D.J. & RANDOLPH S.E. (1993). – Distribution of tsetse and ticks in Africa: past, present and future. *Parasitol. Today*, **9** (7), 266-271.
146. ROGERS D.J. & WILLIAMS B.G. (1993). – Monitoring trypanosomiasis in space and time. *Parasitology*, **106** (Suppl.), S77-S92.
147. ROGERS D.J. & WILLIAMS B.G. (1994). – Tsetse distribution in Africa: seeing the wood and the trees. In *Large-scale ecology and conservation biology*, Chapter 11. 35th Symposium of the British Ecological Society with the Society for Conservation Biology (P.J. Edwards, R.M. May & N.R. Webb, eds). University of Southampton, United Kingdom, 14-16 April 1993. Blackwell Scientific Publications, Oxford, 249-273.
148. SAINI R.K. & HASSANALI A. (1992). – Olfactory sensitivity of tsetse to phenolic kairomones. *Insect Sci. Appl.*, **13** (1), 95-104.
149. SNIJDERS F.L. (1991). – Rainfall monitoring based on Meteostat data – a comparison of techniques applied to the western Sahel. *Int. J. remote Sensing*, **12** (6), 1331-1347.
150. SPATH J. & KUPPER W. (1993). – The trap-oriented behaviour of *G. tachinoides* (Diptera: Glossinidae). In *Twenty-first Meeting of the International Scientific Council for Trypanosomiasis Research and Control*. Yamoussoukro, Côte d'Ivoire, 21-25 October 1991. Organisation of African Unity/Scientific, Technical and Research Commission, Nairobi, 283.
151. SWYNNERTON C.F.M. (1936). – The tsetse flies of East Africa. *Trans. R. entomol. Soc. Lond.*, **84**, 1-579.
152. TCHICAYA T. (1990). – Morphogenèse antennaire et sensibilité olfactive chez la glossine (Diptera, Glossinidae). Ph.D. Thesis, University of Montpellier II, France.
153. THAKERSI H. (1993). – Umfurudzi large-scale target operation. In *Twenty-first Meeting of the International Scientific Council for Trypanosomiasis Research and Control*. Yamoussoukro, Côte d'Ivoire, 21-25 October 1991. Organisation of African Unity/Scientific, Technical and Research Commission, Nairobi, 104.
154. THOMSON J.W., MITCHELL M., REES R.B., SHERENI W., SCHOENFELD A.H. & WILSON A. (1991). – Studies on the efficacy of deltamethrin applied to cattle for the control of tsetse flies (*Glossina* spp.) in Southern Africa. *Trop. Anim. Hlth Prod.*, **23** (4), 221-226.

155. THOMSON J.W. & WILSON A. (1992). – The control of tsetse flies and trypanosomiasis by the application of deltamethrin to cattle. *Bull. Anim. Hlth Prod. Afr.*, **40** (1), 5-8.
156. TORR S.J. (1989). – The host-orientated behaviour of tsetse flies (*Glossina*): the interaction of visual and olfactory stimuli. *Physiol. Entomol.*, **14**, 325-340.
157. TOURE S.M. & MORTELMANS J. (1991). – Impact de la trypanosomose animale africaine (TAA). *Bull. Séances, Acad. roy. Sci. d'Outre-Mer* (1990), **36** (2), 239-257.
158. TRAIL J.C.M., D'IETEREN G.D.M., VIVIANI P., YANGAR G. & NANTULYA V.M. (1992). – Relationships between trypanosome infection measured by antigen detection enzyme immunoassays, anaemia and growth in trypanotolerant N'Dama cattle. *Vet. Parasitol.*, **42** (3-4), 213-223.
159. TUCKER C.J. & SELLERS P.J. (1986). – Satellite remote sensing of primary production. *Int. J. remote Sensing*, **7** (11), 1395-1416.
160. VALE G.A. (1974). – The responses of tsetse flies (Diptera, Glossinidae) to mobile and stationary baits. *Bull. entomol. Res.*, **64**, 545-588.
161. VALE G.A. (1977). – Feeding responses of tsetse flies (Diptera: Glossinidae) to stationary hosts. *Bull. entomol. Res.*, **67**, 635-649.
162. VALE G.A. (1977). – The flight of tsetse flies (Diptera: Glossinidae) to and from a stationary ox. *Bull. entomol. Res.*, **67**, 297-303.
163. VALE G.A. (1979). – Field responses of tsetse flies (Diptera: Glossinidae) to odours of men, lactic acid and carbon dioxide. *Bull. entomol. Res.*, **69**, 459-467.
164. VALE G.A. (1980). – Field studies of the responses of tsetse flies (Glossinidae) and other Diptera to carbon dioxide, acetone and other chemicals. *Bull. entomol. Res.*, **70**, 563-570.
165. VALE G.A. (1993). – Development of baits for tsetse flies (Diptera: Glossinidae) in Zimbabwe. *J. med. Entomol.*, **30** (5), 831-842.
166. VALE G.A. (1993). – Visual responses of tsetse flies (Diptera: Glossinidae) to odour-baited targets. *Bull. entomol. Res.*, **83**, 277-289.
167. VALE G.A. & HARGROVE J.W. (1979). – A method of studying the efficiency of traps for tsetse flies (Diptera: Glossinidae) and other insects. *Bull. entomol. Res.*, **69**, 183-193.
168. VALE G.A., HURSEY B.S., HARGROVE J.W., TORR S.J. & ALLSOPP R. (1984). – The use of small plots to study populations of tsetse (Diptera: Glossinidae). *Insect Sci. Appl.*, **5** (5), 403-410.
169. VAN SICKLE J. (1988). – Invalid estimates of rate of population increase from *Glossina* (Diptera: Glossinidae) age distributions. *Bull. entomol. Res.*, **78**, 155-161.
170. VARLEY G.C., GRADWELL G.R. & HASSELL M.P. (1973). – Insect population ecology: an analytical approach. Blackwell Scientific Publications, Oxford, 212 pp.
171. WACHER T.J., MILLIGAN P.J.M., RAWLINGS P. & SNOW W.F. (1994). – Tsetse-trypanosomiasis challenge to village N'Dama cattle in The Gambia: field assessments of spatial and temporal patterns of tsetse-cattle contact and the risk of trypanosomiasis infection. *Parasitology*, **109**, 149-162.
172. WARNES M.L. (1992). – Activation of three species of tsetse (*Glossina* spp.) in response to host derived stimuli. *Med. vet. Entomol.*, **6** (4), 349-354.
173. WELBURN S.C. & MAUDLIN I. (1991). – Rickettsia-like organisms, puparial temperature and susceptibility to trypanosome infection in *Glossina morsitans*. *Parasitology*, **102** (2), 201-206.

174. WELBURN S.C., ARNOLD K., MAUDLIN I. & GOODAY G.W. (1993). – Rickettsia-like organisms and chitinase production in relation to transmission of trypanosomes by tsetse flies. *Parasitology*, **107** (2), 141-145.
175. WIJERS D.J.B. (1958). – The importance of the age of *G. palpalis* at the time of the infective feed with *T. gambiense*. In Seventh Meeting of the International Scientific Committee for Trypanosomiasis Research. Brussels, 25-30 August. Commission for Technical Co-operation in Africa South of the Sahara, London, 319-320.
176. WILLEMSE L. (1991). – A trial of odour-baited targets to control the tsetse fly, *Glossina morsitans centralis* (Diptera: Glossinidae) in west Zambia. *Bull. entomol. Res.*, **81** (3), 351-357.
177. WILLETT K.C. (1965). – Some observations on the recent epidemiology of sleeping sickness in Nyanza Region and its relation to the general epidemiology of Gambian and Rhodesian sleeping sickness in Africa. *Trans. R. Soc. trop. Med. Hyg.*, **59**, 374-386.
178. WINT W. & BOURN D. (1994). – Livestock and land-use surveys in sub-Saharan Africa. Oxfam, 36 pp.
179. WINTER W.H. & BROWN J. (1994). – Letter to the Editor. *ILCA Newsl.*, **13** (2), 4-5.
180. WORLD BANK (1993). – World development report: investing in health. Oxford University Press, Oxford, 292 pp.