Seasonal Variations in Spread of Arthropod-Borne Disease Agents of Man and Animals: Implications for Control [and Discussion]

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Seasonal variations in spread of arthropod-borne disease agents of man and animals: implications for control

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The disease agents under discussion include viruses and protozoans that undergo a cycle in animals as hosts and in insects as vectors. Such agents are found in climatic zones ranging from tropical rains to cool. Outside the tropical rain forests there are periods during which conditions are unsuitable for the cycle of infection to continue, because the activity of the insects is inhibited at temperatures above 35 °C and below 15 °C. During this 'overwintering' period the virus may survive by persistence in the host, or in the adult insect, larva or egg, or the virus may be introduced or reintroduced into the area by movement or migration of the host or by carriage of infected insects on the wind.

Examples are given of the introduction of disease or infection by insects carried on the wind. Such examples include (i) between the southerly and northerly limits of the Inter-Tropical Convergence Zone, the wind carriage or possible wind carriage of insects infected with bluetongue or yellow fever in West Africa and Sudan and with Rift Valley fever in Egypt, (ii) the wind carriage of midges infected with bluetongue in the eastern Mediterranean and (iii) the introduction of Japanese encephalitis to Japan on monsoon winds by infected mosquitoes.

Analysis of the host–vector cycles of the agents in the particular zones enables appropriate control measures to be devised. In endemic areas this would involve importation and vaccination of introduced animals at the appropriate time. In epidemic and sporadic areas, the uncertainty of the timing of outbreaks has to be recognized and the times of breeding of animals and the need for vaccination of animals assessed. During an epidemic the questions of eliminating insect breeding sites, and dipping and spraying of animals have to be considered in relation to possible reintroduction of infection at a later date.

Introduction

The diseases under discussion, in which aerial transmission occurs by dispersal of infected insects, are caused by viruses or protozoans that undergo a cycle in vertebrates as hosts and in insects as vectors.

Host–vector cycles

The vertebrates involved – mammals and birds – are warm-blooded and in some instances they may suffer disease; often, however, infection can only be detected by the presence of the infective agent or of antibody. Many of the vertebrates can be migratory. The insects include mosquitoes, midges and other biting flies. Their activity, flight, feeding, mating and oviposition are influenced by temperature. For most of the cycles discussed, the lower limit is 15 °C and the upper 35 °C. Moisture is essential for development of the intermediate stages in breeding sites. Flight is unaided over short distances at winds of less than 1.5 m s⁻¹; at winds faster than this, the insects may be carried for tens and hundreds of kilometres (Pedgley 1982, and this symposium; Sellers 1980 .

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The infectious agents multiply in the host and are present in the blood and blood cells or associated with the skin, sometimes with lesions on the skin. With the most agents that multiply in the blood and blood cells of the host, transmission is biological, i.e. they undergo multiplication in the insect vector and are transmitted when that insect bites a fresh host. With those agents multiplying in the skin and with some of those present in the blood, transmission is mechanical, i.e. the virus or protozoan survives on the mouthparts of the insect at biting until the insect bites a fresh host.

Thus in the host–vector cycle the viruses or protozoans are parasites of the insects, which in turn are dependent on the vertebrate host as a source of nutrient.

TABLE 1. DISEASES CAUSED BY VIRUSES AND PROTOZOANS INVOLVED IN SPREAD BY CARRIAGE OF INFECTED INSECTS ON THE WIND

method of transmission	host		disease agent	vector
biological	mammal	viruses	yellow fever	
			Rift Valley fever	mosquito
			vesicular stomatitis	
			Venezuelan equine encephalitis	
			bluetongue/Ibaraki	
			African horse sickness	*Culicoides*
			Akabane	
			bovine ephemeral fever	
		protozoan	trypanosomiasis	*Glossina*
biological	bird	viruses	encephalitides: Japanese, Murray Valley, St Louis, eastern equine, western equine, west Nile	mosquito
mechanical	mammal	virus	myxomatosis	mosquito
		protozoan	trypanosomiasis	tabanid

Table 1 gives a list of the viruses and protozoans and their various vectors in which wind carriage of infected insects may occur. There are also a number of other arthropod-borne virus diseases in which carriage of infected insects on the wind may be responsible for spreading disease, e.g. biologically transmitted by mosquitoes (dengue, Rocio) or mechanically transmitted by biting flies (lumpy skin disease, sheep and goat pox, pseudo lumpy skin disease, bovine herpes mammillitis).

Climatic zones

The areas and climatic conditions where such agents are found can be divided into a number of zones (see table 2). An agent can be found in more than one zone and may be transmitted in different cycles with variation in the species of insect as vector or of vertebrate as host. In the jungle (zone A), yellow fever virus circulates in monkeys and in *Aedes africanus* mosquitoes; in the savannah (zone B) the mosquitoes involved are *Aedes furcifer-taylori* and *Aedes luteocephalus*.

Western equine encephalitis virus, which circulates in all zones in birds and *Culex tarsalis*, may in zones C–E spill over into a mosquito–man or mosquito–horse cycle.

Throughout the zones, mammals and birds, because they are warm-blooded, are not affected by the variation in temperature. However, the activity of insects above 35 °C or below 15 °C is inhibited.

In zone A conditions are suitable for the activity of insects for most of the year, but in zones

C–F there are periods during which it is too hot, too dry or too cold. Insects may survive these adverse conditions as eggs, larvae or adults.

Disease or evidence of infection is found at different times in the zones. In Kuching, Sarawak (zone A: 1° 32′ N, 110° 20′ E), Simpson *et al.* (1976) found serum antibody conversions in pigs to Japanese encephalitis every month of the year except June. In zone B (Vom, Nigeria: 9° 45′ N, 8° 50′ E) Taylor and his collaborators (Taylor & McCausland 1976; Sellers 1980; Herniman *et al.* 1983) found peaks of seroconversion to bluetongue in calves in May–June,

TABLE 2. CLIMATIC ZONES IN WHICH DISEASES LISTED IN TABLE 1 OCCUR

A	temperatures all year > 15 °C	rain all year > 50 mm
B	temperatures all year > 15 °C	wet and dry season
C	temperatures 9 months and more > 15 °C	rain, summer or winter
D	temperature 6–8 months > 15 °C	rain, summer or winter
E	temperature 6 months or more < 15 °C	rain, summer or winter
F	temperatures < 15 °C most of year	

TABLE 3. PERCENTAGES OF ANIMALS SHOWING SEROCONVERSION

	A: Kuching	B: Vom	C: Khartoum
April	3	4	0
May	2	42	0
June	0	24	0
July	7	0	7
August	2	17	30
September	7	81	62
October	6	59	55
November	16	60	0
December	9	22	0
January	14	0	0
February	6	0	0
March	5	24	0

A, Pigs in Kuching, Sarawak (1° 32′ N, 110° 20′ E) (Simpson *et al.* 1976).
B, Calves in Vom, Nigeria (9° 45′ N, 8° 50′ E) (Herniman *et al.* 1983).
C, Calves in Shambat, Khartoum, Sudan (15° 39′ N, 32° 34′ E) (unpublished results).

September–November and March. In Khartoum (extreme north of zone B: 15° 39′ N, 32° 34′ E) work at present being carried out in calves has shown one period of the year for seroconversion to bluetongue (see table 3).

In Cyprus and Israel (zones C–D) outbreaks of bluetongue usually occur between August and December, with most cases in September, October and November. In Cyprus disease may not occur every year; from 1943 to 1977 there were 16 years in which no bluetongue was reported and from 1970 to 1976 no evidence of infection was found in sentinel herds (Sellers 1975; Sellers *et al.* 1979).

Disease may only be introduced into Portugal or Spain on occasion, e.g. bluetongue in 1956, African horse sickness in 1966.

In instances where infection has been found most of the year, the term 'endemic' has been used; where there are outbreaks or incursions every few years, 'epidemic'; where irregular incursions occur, 'sporadic'.

Overwintering

There is speculation on how the virus persists during those periods when virus or evidence of virus infection cannot be found. The term 'overwintering' is used, although survival during

hot dry seasons is also involved (Reeves 1974). Several hypotheses have been put forward to account for this, including the reintroduction by infected insects on the wind (see table 4). Often two or more strategies for overwintering may be apparent; for example, in some zones the period of overwintering is so short that bluetongue virus can persist in the blood cells of cattle or in the adult midge, as well as being introduced afresh from outside the zone. With Japanese encephalitis it has been suggested that virus may be taken by migrating birds or mosquitoes or may be transmitted transovarially. The quantitative aspects of these various strategies have still to be assessed.

TABLE 4. OVERWINTERING: METHODS BY WHICH VIRUSES SURVIVE CONDITIONS
ADVERSE FOR VECTOR

within zone

animal:	survival in tissues, blood cells
	transplacental transmission
	change to different host (bat, reptile)
insect:	survival in adults, larvae, eggs
	change to different vector (flea, tick)

from outside

animal:	migrations
	movements
insects:	wind carriage

However, where there is an abundance of uninfected hosts and where mosquitoes breed during most months of the year, that will suffice to maintain the cycle over the whole period. In the more temperate climates, as well as the possibility of reintroduction, there is a tendency towards persistence in a host, transovarial transmission in an insect or persistence in an arthropod that survives the adverse conditions, e.g. tick or rabbit flea. Thus a gradation can be seen with yellow fever (zones A and B) being present in the host for a comparatively short time, bluetongue (zones A–E) being present in cattle blood for up to 100 days, and members of the California group of viruses (zones D and E) showing transovarial transmission.

WINDBORNE CARRIAGE OF INFECTED INSECTS

The main interest of the discussion is the spread of disease by wind carriage of insects infected with disease agents. The attributions to this method of spread have been based on circumstantial evidence, back-tracking from later developments and arrivals with weather systems (Pedgley 1982, and this symposium). The insects responsible have been caught at various heights (Johnson 1969; Bowden & Gibbs 1973) and over the sea (Hayashi *et al.* 1979), but isolation of viruses from insects captured in the air has not been reported. Evidence has come from analysis of outbreaks of disease or infection where, because spread took place across seas or deserts, the possibility of host movement could be excluded. Often the epidemiological evidence is incomplete and it is necessary to use historical judgement on the reports. Nevertheless, the meteorological data are sufficient to show where or where not suitable winds were available. Techniques of 'fingerprinting' are being developed for identifying viruses and insects at the source and at the site of infection, and these should assist analysis in the future.

Several situations involving introduction of viruses by infected insects on the wind have been analysed.

Mammal–insect

Bluetongue: West Africa and Sudan

Taylor and his colleagues have studied the numbers of midges and seroconversion to bluetongue in calves at Vom, Nigeria (9° 45′ N, 8° 50′ E). A peak in the numbers of midges caught (*Culicoides imicola*, the main vector of bluetongue) was found from August to October; smaller peaks occurred in February and May–June. Seroconversion in calves was found in nine months of the year, the exceptions being July, January and February. Peaks in seroconversion were seen in September–November, in March and in May–June. These peaks can be correlated with the peaks in numbers of midges found. However, the failure to find seroconversion in July, during the wet season when midges were comparatively plentiful, needs explanation.

In similar studies at Khartoum (15° 39′ N, 32° 34′ E), seroconversion to bluetongue in calves occurred in July, August, September and October. Catches of *Culicoides imicola* can be associated with these peaks.

These results suggest that, although there may be a background of infection most of the year in Nigeria, the main peaks are due to the carriage of infected *Culicoides* on winds resulting from the northward or southward movement of the Inter-Tropical Convergence Zone (I.T.C.Z.). At Vom there are two passages of the I.T.C.Z. Khartoum is near its northerly limit, so that the effects of its northward and southward passage cannot be separated and one peak of seroconversion over 2–3 months was found. The peak of seroconversion in March in Vom may

TABLE 5. SEQUENCE OF VIRUS INFECTION IN MAMMAL–MIDGE CYCLE IN WEST AFRICA

(1) northward movement of I.T.C.Z.
(2) growth of herbage as result of rains
(3) movement of ruminants to feed on herbage
(4) movement of insects on southern winds for feeding on blood, for oviposition and for breeding sites
(5) virus carried northwards in ruminants and in insects

have been due to oscillations of the I.T.C.Z. similar to those described by Magor & Rosenberg (1980).

In Sudan, in some years the I.T.C.Z. moves further north than usual. The outbreak of Rift Valley fever in the Aswan district in 1977 was preceded by an unusual northward surge of the I.T.C.Z. This remained for 6 days over Aswan (24° 05′ N, 32° 56′ E). The southerly winds could have been responsible for bringing infected insects into that area or infecting animals just to the south, which then arrived in Aswan district (Sellers *et al.* 1982).

Where cattle, wild ruminants and insects all migrate, the movement of the I.T.C.Z., the rains, the movement of cattle and that of insects interrelate (see table 5). The virus thus moves in a cycle between the host and vector; the vector is carried forward by the I.T.C.Z. as its source of food – the host – moves forward to feed on the herbage. Where settlement of cattle has occurred, movement of the insect still continues.

Yellow fever: West Africa and Sudan

Windborne dispersal of insects may be responsible for spread of yellow fever. In recent years, outbreaks of yellow fever in the savannah areas of West Africa have been investigated (Germain *et al.* 1981). Jungle yellow fever involving monkeys and *Aedes africanus* occurs in the forests, while

at the edges of the forest and north in the savannah a cycle involving monkeys and *Aedes furcifer-taylori* and *A. luteocephalus* is found. Man is also affected. Spread has been attributed to the movement of monkeys along the river gallery forests or to movements of man. Outbreaks of yellow fever were reported in West Africa as follows (Chambon *et al.* 1971): 1964, Portuguese Guinea; October 1965, Senegal; 1967, Liberia; September 1969, Mali, Upper Volta, Ghana, Togo, Nigeria; 1970, Cameroon, equatorial Guinea.

The time of year of the outbreaks suggests that movements of the I.T.C.Z. could have been involved. The virus moved eastwards through the forest in a mosquito–monkey cycle. At the edge of the forest, besides monkeys, southwest winds could have been responsible for carrying infected mosquitoes. The outbreaks in Nigeria in 1969 were at Jos, where previous outbreaks of yellow fever had been reported in October and November 1951 (Monath 1972). Outbreaks some distance from known sources occurred in the Nuba mountains, Sudan, in May–November 1940 (Kirk 1941) and western Ethiopia and eastern Sudan in August–November 1959 (Berdonneau *et al.* 1961; Satti & Haseeb 1966). In 1958 yellow fever had been reported from northeastern Congo. The 1958 and 1959 outbreaks were followed by an epidemic in Ethiopia (1960–2) (Série *et al.* 1968). Whether these outbreaks were associated with windborne carriage of infected insects is unknown but it is a possibility.

In recent years there has been evidence of yellow fever in Senegal in 1976–8, cases in Ivory Coast in 1971 and an epidemic in Gambia in 1978 (Salaun *et al.* 1981).

Other examples of dispersal on the wind in these areas include *Glossina* and trypanosomiasis in West Africa (Molyneux *et al.* 1979) and tabanids and trypanosomiasis in Sudan (Bowden 1976).

Bluetongue: eastern Mediterranean

Outside the limits of the I.T.C.Z., investigations have been made of bluetongue and bluetongue infection in Cyprus and the neighbouring countries: Turkey, Syria, Israel and Jordan (Sellers *et al.* 1979; Yonguc *et al.* 1982). Most outbreaks of bluetongue in Cyprus occur between August and December, but not every year (Sellers 1975). The intervals between epidemics vary from 1 to 8 years. It appears that the virus is unlikely to overwinter in the island, because there are so few cattle as hosts and the winter in most years is too severe. Persistence of virus in sheep and goats for long periods has not been found (Gibbs *et al.* 1979). There has been no correlation between imports of ruminants and occurrence of disease. Outbreaks of bluetongue in September 1965, December 1969 and August 1977 were preceded by southeasterly, easterly, northeasterly and northerly winds 6–14 days previously (Sellers *et al.* 1979; Sellers & Herniman 1981). These winds could have brought infected midges from Israel, Syria and eastern Turkey. Northerly and northeasterly winds in August 1960 were responsible for bringing midges infected with African horse sickness virus from Turkey to Cyprus (Sellers *et al.* 1977). Further investigations over the past few years indicate that there may be a circulation of bluetongue virus in northern Israel, Syria (Orontes valley), Turkey (Adana region) which, under certain climatic conditions between August and December, may involve Cyprus and – as happened in 1977 – western Turkey (Aydin, 37° 50′ N, 27° 30′ E). In Turkey and Israel there is a large cattle population in which the virus can survive the winter.

At the western end of the Mediterranean, wind carriage of infected *Culicoides* may have been responsible for introducing bluetongue into Portugal in 1956 and African horse sickness into Spain in 1966 (Sellers *et al.* 1977, 1978).

Bird–mosquito cycle

Japanese encephalitis: SE Asia and Japan

Another example of possible spread of virus by carriage of infected insects on the wind is found with Japanese encephalitis outbreaks in Japan. Japanese encephalitis virus is endemic in Indonesia and Sarawak, where there is a cycle involving *Culex tritaeniorhyncus* and *C. gelidus* and pigs and birds (Simpson *et al.* 1966). The disease is also found throughout southeast Asia, India and southern China, where it occurs seasonally (Sellers 1980).

In Japan the virus circulates in *C. tritaeniorhyncus* and in birds and pigs, occasionally affecting horses and man. In southwest Japan infection occurs most years, the first signs of infection in animals or mosquitoes occurring in June and July. Evidence of infection disappears in October.

Overwintering of virus has been attributed to survival of infected adult mosquitoes, transovarial transmission or reintroduction by migrating birds. However, overwintering mosquitoes and migrating birds appear at the end of March but evidence of virus is not found until May or early June.

TABLE 6. SEQUENCE OF VIRUS INFECTION INVOLVING BIRD–MOSQUITO CYCLE IN EASTERN ASIA

March	birds migrate northwards to nesting areas
March–April	female insects (overwintered) appear
May–June	increase in newly emerged insects nestlings are hatched
June	warm southerly winds (monsoon) bring insects infected with virus cycle of virus in nestlings, piglets, insects and local insects
September–October	winds from north bird migration

The time of appearance of virus coincides with the advance of the southwest monsoon to Japan in June and early dates of appearance of infection can be correlated with early advance of fronts associated with the monsoon (Sellers 1980). Some specimens of *C. tritaeniorhyncus* have been caught at sea (Hayashi *et al.* 1979).

Thus infection may follow (see table 6) the introduction of virus by infected mosquitoes coinciding with the optimum time for spread of virus in antibody-free nestlings and young pigs through large numbers of insects.

A similar cycle of infection may occur with Western equine encephalitis, Eastern equine encephalitis and St Louis encephalitis in North America.

In Japan the dates of introduction of Akabane, bovine ephemeral fever and Ibaraki viruses are associated with advance of the southwest monsoon or with tropical storms.

In Australia, spread of bovine ephemeral fever and of Akabane disease has been attributed to infected insects carried on the wind (Murray 1970; Della-Porta *et al.* 1976).

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Control

The analysis of the host–vector cycle in a particular zone, the involvement of movement of the host, and wind carriage of the insect in spread of infection and persistence of the infective agent during unfavourable periods enables a risk assessment to be made and the appropriate control measures to be devised before, during and after outbreaks. Risk assessment is probably easier in endemic areas than in epidemic or sporadic areas, where fringe or edge effects are seen.

Endemic areas

When animals or genetic material (semen and embryos) are being imported into endemic areas, it is possible to choose the time of importation to undergo the least risk (Sellers & Taylor 1980). With bluetongue in West Africa, this is the period in January and February before the northerly advance of the I.T.C.Z., when no seroconversion has been found. The animals can also be vaccinated at the same time. When such precautions are omitted, severe losses can occur. For example, when Shire horses were imported into northern Nigeria in September 1974 they were rapidly struck down by African horse sickness and succumbed (Best *et al.* 1975). This was the period of the year when one would expect the greatest weight of infection.

Where export of animals is required, it would be possible to move young animals at certain periods of the year after they had lost maternal antibodies and before fresh infection occurred.

Epidemic and sporadic areas

In epidemic or sporadic areas, introduction of infection by infected insects may occur every 1, 2, 5, 10 or 20 years, depending on the coincidence in time and place of the presence of virus, vectors and hosts, together with suitable winds and temperatures (Sellers *et al.* 1977).

Pregnant animals

Some of the insect-borne viruses affect the pregnant animal. Rift Valley fever causes abortion in ruminants; Japanese encephalitis causes abortion in sows as well as death of newborn pigs; Akabane and bluetongue viruses give rise to abortions, foetal malformations, arthrogryposis and hydranencephaly in cattle, sheep and goats. Where animals become infected in the first year of life, antibodies develop so that the breeding female is protected. Where infection is introduced every few years, the viruses could affect a number of susceptible animals during pregnancy. For example, in New South Wales and in southwestern Japan, Akabane disease causes problems when infected mosquitoes are introduced on the wind. Rift Valley fever caused great losses due to abortions in Sudan in 1973 and in Egypt in 1977, when the virus was carried northwards beyond its usual area.

To avoid some of these losses, changes in animal husbandry have been made. In Japan the occurrence of early pregnancy in pigs from July to September is avoided. Similarly, in Cyprus the first 50 days of pregnancy in sheep are between May and July, times of the year when infection with bluetongue or Akabane viruses is unlikely (Sellers & Herniman 1981).

Import, export and disease outbreaks

In epidemic and sporadic areas the introduction of infection, its extent and duration varies from year to year. It is therefore necessary in exporting animals, semen or embryos from these areas to test for freedom from infection each year. In the north-central and northeastern states

of the U.S.A., bluetongue is infrequent. It could be introduced by animal movement or by infected midges brought in by unusual warm winds. Cattle, in which the virus may persist, do not show clinical signs and the infection is only apparent in serological tests. To avoid the introduction of bluetongue, exports may be carried out during the winter season in the absence of midges, the animals or those providing semen or embryos having previously been shown to be free of infection. In epidemic and sporadic areas, people and animals can be protected by vaccines. For people who intend to visit or live in West Africa there is an effective yellow fever vaccine that gives protection for 20 years or more. However, with regard to animal diseases that may only be introduced every 3 or 4 years, there may be a reluctance to vaccinate, especially if there are 20 or more serological types (as in bluetongue) or where, as with an inactivated vaccine, the immunity conferred is of short duration. Vaccination in the face of an outbreak may also produce problems, since a live vaccine may have to be given to pregnant animals.

At the time of an outbreak, dipping or spraying of animals may give protection for a short period. Elimination of insect breeding sites may be difficult if recolonization occurs. However, control by spraying would give relief for a period sufficient to delay the virus cycle in the vector.

Attempts could be made to develop predictive methods. In the inter-epidemic period assessments of the likelihood of incursion could be made. During an epidemic, the likelihood of spread of insects and the areas at risk could be assessed and vaccination and spraying carried out in those areas.

Often it might be better to discover the source of the infection and carry out preventive measures there. However, in a country with endemic bluetongue, with few problems in cattle and sheep, there would be no incentive on the part of that country to vaccinate or attempt other control measures.

The possibilities of wind carriage of infected insects have not been recognized to the same extent for human and animal diseases as for plant pathogens and insect pests, perhaps because spread of such diseases is attributed to movement of the host or to overwintering. However, movement by carriage of infected insects may be becoming more important; forests are being cut down and spread of disease through savannah insects, which can move over longer distances than forest mosquitoes, is increasing. New irrigation schemes attract colonization by insects for breeding sites. It is more than ever important to take into account the possibility of windborne movement of infected insects when assessing spread of disease.

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Discussion

W. H. G. REES (*M.A.F.F., Tolworth, Surbiton, U.K.*). Would Dr Sellers comment on the factors that may influence the competence of *Culicoides* to transmit infection? *Culicoides variipennis*, the vector of bluetongue in North America, transmitted disease in the southern states but although present in the northeastern states did not appear to be competent to maintain infection, although periodic incursions of the disease into the area occurred.

R. F. SELLERS. Workers in Denver, Colorado, U.S.A., have described populations of *Culicoides variipennis* caught in the northeastern states that have a low infection rate to bluetongue virus by the oral route. They have also tested populations from California, where bluetongue is endemic, and found higher infection rates. There may also be differences in infection rates due to the different strains of virus used. In my opinion, changes in infection rates in *Culicoides* in the northeastern states might occur because of a change in weather conditions more suitable for *Culicoides* and/or *Culicoides* being introduced on the wind from further south.

J. D. GILLETT (*London School of Hygiene and Tropical Medicine, U.K.*). I was a little surprised at the suggestion (in the discussion that followed Dr Sellers's interesting paper) that perhaps no tests had been made on the possible effect of infection with yellow fever virus on mosquitoes. In the 1930s infected and normal *Aedes aegypti* were compared, mainly for longevity it is true, and no difference was detected between them, except of course that the infected insects remained capable of transmitting the virus when fed on a susceptible host. Both groups survived, as far as I recall, for more than 5 months, during which time they must have passed through many (at least a dozen?) ovarian cycles. Today, new and more sophisticated work has disclosed that viruses may indeed affect the mosquito host, as Dr Tinsley pointed out with Semliki Forest virus and its enhancement of flight activity (which, I suggest, may be supposed to provide a selective advantage to the virus, complementing that provided by the rise in temperature that may occur in the viraemic vertebrate host (Gillett & Connor 1976)). The main point I wish to make, however, is that we should at least be aware of what was done in earlier days.

Reference

Gillett, J. D. & Connor, J. 1976 Host temperature and the transmission of arboviruses by mosquitoes. *Mosq. News.* **36**, 472–477.

J. P. T. BOORMAN (*Animal Virus Research Institute, Pirbright, U.K.*). Further to the discussion on the effect of virus infection upon the insect vector, there is no evidence that infection has any effect upon biting midges either. However, the possible effects have usually been measured by effect upon longevity in the insect. By this same criterion one could say that the common cold has no effect upon humans. Infection may have some effect upon behaviour of the insect and this could be very important epidemiologically, but so far no really satisfactory methods have been worked out to measure the possibility. Although levels of virus in the infected insect are usually high, we do not know how many cells are infected and it may be a small proportion of the whole. This might not be sufficient to cause any change in behaviour.

R. C. RAINEY, F.R.S. (*formerly Centre for Overseas Pest Research, U.K.*) In support of Dr Sellers' point on tropical wind systems and vector distribution, we have taken *Culicoides* by aircraft-trapping in the intertropical convergence zone in the Sudan, up to 150 m above the ground in traverses of the sharply defined intertropical front over the Gezira in October.

Further to his suggestion of increases in the more mobile insects after the widespread clearance of tropical forest to savannah, this same increase of grass may well be a factor in the menacing and probably unprecedented severity of recent armyworm attacks in a number of countries, such as by *Spodoptera exempta* in Sierra Leone, where in sample areas 33 % of forest was lost between 1958 and 1975. This migrant moth pest of cereals and rangeland feeds exclusively on Gramineae and Cyperaceae.