J. Hyg., Camb. (1980), **85**, 65
Printed in Great Britain

Weather, host and vector – their interplay in the spread of insect-borne animal virus diseases

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(*Received* 5 *November* 1979)

SUMMARY

The spread of insect-borne animal virus diseases is influenced by a number of factors. Hosts migrate, move or are conveyed over long distances: vectors are carried on the wind for varying distances in search of hosts and breeding sites; weather and climate affect hosts and vectors through temperature, moisture and wind. As parasites of host and vector, viruses are carried by animals, birds and insects, and their spread can be correlated with the migration of hosts and the carriage of vectors on winds associated with the movements of the Intertropical Convergence Zone (ITCZ) and warm winds to the north and south of the limits of the ITCZ. The virus is often transmitted from a local cycle to a migratory cycle and back again.

Examples of insect-borne virus diseases and their spread are analysed. Japanese, Murray Valley, Western equine, Eastern equine and St Louis encephalitis represent viruses transmitted by mosquito–bird or pig cycles.

The areas experiencing infection with these viruses can be divided into a number of zones: A, B, C, D, E and F. In zone A there is a continuous cycle of virus in host and vector throughout the year; in zone B, there is an upsurge in the cycle during the wet season, but the cycle continues during the dry season; there is movement of infected vectors between and within zones A and B on the ITCZ and the virus is introduced to zone C by infected vectors on warm winds; persistence may occur in zone C if conditions are right. In zone D, virus is introduced each year by infected vectors on warm winds and the arrival of the virus coincides with the presence of susceptible nestling birds and susceptible piglets. The disappearance of virus occurs at the time when migrating mosquitoes and birds are returning to warmer climates. The virus is introduced to zone E only on occasions every 5–10 years when conditions are suitable. Infected hosts introduced to zone F do not lead to circulation of virus, since the climate is unsuitable for vectors. Zones A, B and C correspond to endemic and zones D and E to epidemic conditions.

Similar zones can be recognized for African horse sickness, bluetongue, Ibaraki disease and bovine ephemeral fever – examples of diseases transmitted in a midge-mammal cycle. In zones A and B viruses are transported by infected midges carried on the wind in association with the movement of ITCZ and undergo cycles in young animals. In these zones and in zone C there is a continual movement of midges on the warm wind between one area and another, colonizing new sites or reinforcing populations of midges already present. Virus is introduced at

0022-1724/80/0100-1979 $01.00

times into fringe areas (zones D and E) and, as there is little resistance in the host, gives rise to clinical signs of disease. In some areas there is persistence during adverse conditions; in others, the virus is carried back to the endemic zones by infected midges or vectors.

Examples of viruses maintained in a mosquito/biting fly–mammal cycle are Venezuelan equine encephalitis and vesicular stomatitis. These viruses enter a migratory cycle from a local cycle and the vectors in the migratory cycle are carried over long distances on the wind. Further examples of virus spread by movement of vectors include West Nile, Rift Valley fever, yellow fever, epizootic haemorrhagic disease of deer and Akabane viruses.

In devising means of control it is essential to decide the relationship of host, vector and virus and the nature of the zone in which the area to be controlled lies. Because of the continual risk of reintroduction of infected vectors, it is preferable to protect the host by dipping, spraying or by vaccination rather than attempting to eliminate the local population of insects.

INTRODUCTION

Animal viruses transmitted by insects have often been found to spread over great distances especially just before and during epidemics. This spread has been attributed to involvement of people or animals carrying the virus and to infected insects carried on planes, ships or vehicles. Recently a number of papers have been published on the spread of insect-borne animal viruses by infected insects carried on the wind. The virus diseases include bovine ephemeral fever (Seddon, 1938; Murray, 1970; Newton & Wheatley, 1970), African horse sickness (Sellers, Pedgley & Tucker, 1977), bluetongue (Sellers, Pedgley & Tucker, 1978; Sellers *et al.* 1979), myxomatosis (Ratcliffe *et al.* 1952) and Japanese encephalitis (Hayashi *et al.* 1978*a*). In some of these papers examples were given and analyses made of the spread of virus by infected insects during outbreaks or epidemics of a disease when other methods of spread could be excluded.

In the present paper the spread of insect-borne viruses carried on the wind will be related to the spread of such viruses by the movement of animals. A scheme involving a dynamic system which includes migration of hosts and vectors, local cycles of hosts and vectors, the persistence of virus, climate and winds will be put forward. The spread of a number of insect-borne viruses in various parts of the world will be analysed. Zones will be delineated based on the climate and the behaviour and persistence of the viruses in different areas, and the usefulness of such zones in determining the epidemiology and control of virus diseases will be discussed.

VIRUS CYCLES

Virus

In insect-borne virus diseases the virus is transmitted from one host to another by the bite of a vector. The virus multiplies in both host and vector. After introduction into the host by a bite from an infected insect, the virus multiplies locally

and is then distributed through the host giving rise to a viraemia. During or after viraemia signs of disease may appear or there may be inapparent infection. The insect vector biting a host with viraemia may become infected. The virus multiplies in the insect and after 7–10 days can be transmitted to new hosts; the insect remains infective for the rest of its life. Spread of the virus over distance depends on the movements of host and vector and these movements are related to the environment, climate, trade and migration as well as other factors.

A number of host–vector cycles have been found. These are based on location – for example, with yellow fever there is an urban cycle, a jungle or sylvan cycle and a savannah cycle (Gayral & Cavier, 1971) – or based on the vector, e.g. a mosquito–bird cycle or a bug–bird cycle, as with Western equine encephalitis, or based on the host, e.g. a mosquito–bird or mosquito–pig cycle or a mosquito–man or mosquito–horse cycle as with Japanese encephalitis. Some of these cycles involve static populations of hosts and vectors, others migratory hosts and vectors. In endemic areas, adaptation of the virus to the host has occurred and infected hosts frequently show no clinical signs. When the virus is introduced to unfamiliar hosts or to areas where it has not been before or for some time, edge or fringe effects occur and often disease is seen in the unfamiliar host or in the usual host in the new areas.

The virus is essentially a parasite and its survival depends on the maintenance of the host–vector cycle and the availability of sufficient numbers of hosts and vectors.

After infection, an animal develops antibodies and no longer acts as a source of infection. However, young are born regularly and after loss of maternal antibody become new hosts. Insects are dependent on a suitable temperature for survival and where this is maintained throughout the year, the cycle can continue. However, in other areas, owing to adverse conditions, the insect migrates or goes into diapause as adult, larva or egg.

During the period of adverse conditions for the vector, 'overwintering' of the virus may take place (Reeves, 1974). The virus may survive in the host as a chronic infection associated with certain tissues or it may be carried across the placenta and thus be transmitted to the newborn animal. In the insect the virus may persist in the surviving adults or be transmitted transovarially. In addition, the virus may be carried out of an area by migrating hosts or vectors and reintroduced in the next or later years.

Hosts

The hosts in which the viruses multiply include man, domestic and wild mammals and birds. The numbers will be influenced by the availability of food, which depends ultimately on the soil and climate.

Migratory movements of man and his domestic animals are well known; for example, transhumance in the Middle East of owners with their herds of sheep, goats and donkeys; nomadic movement of Fulani tribesmen and their cattle in West Africa; movement of nomads with sheep and goats in the Sudan and the migrations of zebra, wildebeeste and Thomson's gazelle in the Serengeti region

of Central Africa. The distance travelled by these animals is unlikely to exceed 30–50 km a day. Far greater distances varying from tens and hundreds to thousands of kilometres are travelled by migrating bats and by migrating birds.

As the hosts are warm-blooded, low ambient temperatures are not a factor in restricting spread.

Vectors

The vectors of these viruses are mainly mosquitoes and midges although sandflies and other biting flies are sometimes involved. After passing through egg, larval and pupal stages, the newly emerged female takes a blood meal and mates. Thereafter it can be fertile for life, but will need to take a blood meal every three to five days for eggs to develop. Eggs are laid in various sites and warmth is necessary for the hatching and development of larvae.

The metabolism of the insect is dependent on temperature. Under optimal conditions of temperature (13–35 °C) and humidity and with an abundance of sites for egg laying, development of larvae and pupation, the number of adults can reach high levels. During adverse conditions the insects go into diapause (hibernation or aestivation) or migrate. Survival during diapause is as adults, eggs or larvae depending on the species. With migrating insects, adults die out completely after a season in an area or they migrate to other sites; recolonization of the original site takes place when suitable conditions return.

Some of the blood-sucking insects to be discussed are regarded as static and fly short distances. Such are *Aedes aegypti*, a vector of yellow fever and dengue viruses, *Culiseta melanura*, a vector of Eastern equine encephalitis virus, and *Culex aikenii*, a vector of Venezuelan equine encephalitis virus. Others, often the occupiers of temporary habitats, are regarded as migratory, being dispersed on the wind (see Johnson, 1969). Flights of 5–100 km have been recorded for *Aedes taeniorhynchus* (a vector of Venezuelan equine encephalitis virus) and of 175 km for *Aedes sollicitans* (a vector of Venezuelan and Eastern equine encephalitis viruses.) *Aedes vigilax* (a vector of Murray Valley encephalitis) has been found 100 km from source in Queensland. *Culex tarsalis* (a vector of Western equine encephalitis and St Louis encephalitis viruses) has been recaptured 15 km from source and *Culex tritaeniorhynchus* (a vector of Japanese encephalitis virus) is thought to fly at least 8·4 km (Wada *et al.* 1969). *Culicoides* species have been carried for 5–6 km on winds (see Sellers *et al.* 1977). In addition, there is circumstantial evidence (Murray, 1970; Sellers *et al.* 1977, 1978, 1979) that midges have been carried in the air for distances of 40–700 km.

Such flights were completed at temperatures of 15–35 °C at heights up to 1·5 km. *Culicoides* species have been captured at heights up to 1·7 km and mosquitoes have been captured at heights up to 1·5 km (Glick, 1939). *Culex tarsalis* were caught at heights between 10 and 600 m and *Aedes vexans* (a vector of Venezuelan and Eastern equine encephalitis viruses), *Aedes sollicitans* and *Aedes taeniorhynchus* at heights up to 30 m (see Johnson, 1969).

Climate

Three aspects of climate have an effect: temperature, winds and moisture. Insects are cold-blooded and the species discussed here are active at temperatures between 13 and 35 °C. Winds are important in two ways: in the absence of wind or at wind speeds below 2 m s^{-1}, insects can fly unaided. At higher speeds insects can be carried on the wind for long distances. Moisture is important for the growth of herbage or crops on which animals feed, and for breeding sites for insects. Moisture is present as a result of rain, rivers, irrigation schemes or boreholes.

In the carriage of insects over long distances warm winds are important and these occur between and outside the tropics. Between the tropics the extent of the warm winds is determined by the Intertropical Convergence Zone (see Boucher, 1975). This is a zone where southerly winds from the southern hemisphere meet northerly winds from the northern hemisphere. The position of the zone varies with the season, moving to the northern hemisphere in the northern summer and to the southern hemisphere in the southern summer. The position in January and July is shown in Fig. 1. The position varies from day to day and from month to month. In certain years the zone may be further north or further south than the average. The extent of the north-south movement is greater over land than over sea. Other zones of convergence also occur where the wind systems meet, examples being the Rift Valley convergence zone in East Africa and the Red Sea convergence zone (Brown, Betts & Rainey, 1969). Movement of the zone is also associated with rains either at the zone of convergence itself or some distance on the equator side of it. Outside the limits of the zone, warm winds are represented by the southern or northern carriage of warm tropical air as the result of the movement of low or high pressure areas in the mid-latitudes.

Types of climate. As a result of the movement of the ITCZ, the rains and warm winds, a number of climates suitable for blood-sucking insects during all or part of the year can be defined. These are based on the seasonal climates (*Oxford World Atlas*, 1973).

(i) Summer, no winter, all months with rain over 50 mm (Indonesia, Malaysia, Congo basin, Amazon basin).

(ii) Summer, no winter, with rain during the summer season for at least 5 months (Central America, Brazil, West Africa, Central Africa).

(iii) Full summer with mild winter, rain during the summer (Japan, South Africa, Australia, Texas).

(iv) Full summer with mild winter, rain during the winter (Mediterranean, California).

(v) Full summer, cold winter (China, N. America).

In these climates there is a period for part or all of the year during which temperature and moisture are suitable for colonization and breeding of the insects concerned.

Table 1. *Association of winds with movement of insects within and beyond the boundaries of the Intertropical Convergence Zone*

Wind system and location	Insect	References
ITCZ – Africa, Western Asia, India	*Schistocerca gregaria*	Rainey (1973, 1974)
Red Sea Convergence Zone	*S. gregaria*	Rainey (1973, 1974)
ITCZ – West Africa	*Agrius convolvuli*, *Nezara viridula*	Bowden (1973)
	Dysdercus voelkeri	Duviard (1977)
	Culicoides spp	Sellers *et al.* (1977).
ITCZ – Sudan	*Homoptera*	Bowden & Gibbs (1973)
	Ceratopogonidae	
	Tabanidae	Bowden (1976)
ITCZ – East Africa	*Spodoptera exempta*	Brown *et al.* (1969)
Rift Convergence Zone – East Africa	*S. exempta*	Haggis (1971)
ITCZ – Rhodesia, Southern Africa	*S. exempta*	Brown *et al.* (1969)
S.W. Winds (ITCZ) – Japan	*Sogata furcifera*	Kisimoto (1971)
	Nilaparvata lugens	
	C. tritaeniorhynchus	Hayashi *et al.* (1978*a*)
S.W. Winds (ITCZ) – China	*Pseudaletia separata*	Li *et al.* (1964)
Southerly winds – N. Africa Iberian Peninsula	*Schistocerca gregaria*	Johnson (1969)
	Culicoides spp	Sellers *et al.* (1977), Sellers *et al.* (1978)
Southeasterlies – Middle East	*Schistocerca gregaria*	Rainey (1974)
	Agrotis ipsilon	Rainey (1974)
	Culicoides spp	Sellers *et al.* (1977)
Westerlies – India	*C.* spp	Sellers *et al.* (1977)
Northerlies – Australia	*C.* spp	Murray (1970)
Southerlies – America	*Circulifer tenellus*	Douglass & Cook (1954)
	Empoasca fabae	Johnson (1969)
	Macrosteles fascifrons	Chiykowski & Chapman (1965)
	Spodoptera frugiperda	Brown *et al.* (1969)

Winds associated with the movement of insects

Carriage on the wind of insects such as locusts, armyworms, aphids and leafhoppers has long been known. In investigations on the desert locust (*Schistocerca gregaria*) Rainey (1951, 1973, 1974) demonstrated the movement of locusts in association with the ITCZ throughout East Africa, Red Sea, other parts of Africa, Middle East and India.

A list of winds between and outside the ITCZ and the species of insects carried by them is shown in Table 1 (see also Fig. 1). It will be noted that a number of species of insects are carried by the same wind system.

Dynamics of the spread of insect-borne viruses by the movement of hosts and the wind-carriage of vectors

Factors involving viruses, movement of hosts, wind-carriage of insects, weather and climate can be linked together to provide the following synthesis.

(1) Vertebrate hosts (man, animals, birds) migrate or move to and fro from one area to another in organized or random ways to take advantage of food or breeding conditions in the area to which they are moving.

Table 2. *Host and vectors in bird, pig–mosquito cycles*

	Hosts		Vectors	
Virus	Primary	Secondary	Migratory	Static
Japanese encephalitis	Birds	Man	*Culex tritaeniorhynchus*	*Culex gelidus*
	Pigs	Horses	*C. vishnui*	*C. annulus*
				C. fuscocephalus
Murray Valley encephalitis	Birds	Man	*Aedes vigilax*	*C. annulirostris*
Western equine encephalitis	Birds	Horses	*Culex tarsalis*	*C. inornata*
		Man		*Culiseta melanura*
Eastern equine encephalitis	Birds	Horses	*Aedes vexans*	*Culiseta melanura*
		Man	*A. sollicitans*	*Mansonia perturbans*
St Louis encephalitis	Birds	Man	*Culex tarsalis*	*C. pipiens*
				C. nigripalpus
West Nile	Birds	Man	*C. univittatus*	*C. pipiens*
			C. vishnui	

(2) Blood-sucking insects migrate or move to and fro from one habitat to another and thus exploit conditions for breeding, reinforce insect populations already present and take advantage of food sources in the area or reaching these as a result of (1) above.

(3) Because of their size, the direction and extent of movement of migratory blood-sucking insects is determined by winds. Warm winds are needed and these winds occur between the northern and southern limits of the Intertropical Convergence Zone or outside these limits result from the northerly or southerly movement of warm tropical air.

(4) The virus is taken by the migrating host or vector to areas where the host is found or to which it migrates. When the vector is responsible, the virus will be carried by infected insects borne on the wind. As a result, virus will be introduced to new areas or to areas in which the virus has disappeared. In addition, the virus will reinforce virus already present in an area as a result of a cycle in local hosts and vectors, 'overwintering' in the host, transovarial transmission, survival of infected adult insects or other means.

(5) At the start of the movement the virus may have come into the migratory host or vector from a local host–vector cycle. At the end of the migratory movement the virus may enter a local host-vector cycle.

(6) Thus there is a dynamic system involving host, vector and virus. This system is maintained between different areas by local cycles and by cycles involving migratory movement of vertebrates and of insects over many kilometres, dictated by weather conditions.

EXAMPLES OF SPREAD OF INSECT-BORNE VIRUSES

The viruses will be discussed in two groups:

(1) Main host–vector cycle in birds and mosquitoes – Japanese encephalitis, Murray Valley encephalitis, Western, Eastern and St Louis encephalitis viruses (Table 2).

Table 3. *Hosts and vectors in mammal–midge cycles*

Virus	Hosts	Vectors
African horse sickness	Horses, mules, donkeys, zebras	*Culicoides* spp.
Bluetongue	Cattle, sheep, goats, wild ruminants	*Culicoides* spp.
Ibaraki	Cattle	*Culicoides* spp.
Epizootic haemorrhagic disease of deer	Deer	*Culicoides* spp.
Bovine ephemeral fever	Cattle, buffaloes	*Culicoides* spp. (?)
Akabane	Cattle, sheep, goats	*Culicoides* spp.

Table 4. *Hosts and vectors in mammal–mosquito cycles*

Venezuelan equine encephalitis		
Static	Rodents	*Culex* (*melanoconion*)
		C. aikenii
Migratory	Horses, donkeys, man	*Aedes taeniorhynchus*
		A. sollicitans
		A. vexans
Vesicular stomatitis		
Static	? Forest mammals, man	*Phlebotomus*
Migratory	Horses, cattle, sheep, pigs	? Biting flies
Yellow fever		
Static		
Urban	Man	*Aedes aegypti*
Sylvan	Monkeys	*A. africanus*
		A. simpsoni
		Haemagogus spp.
		Sabethes spp.
Migratory		
Savannah	Monkeys	*Aedes luteocephalus*
	Man	*A. metallicus*
		A. vittatus
		A. furcifer-taylori
Rift Valley fever	Sheep, cattle, man	*Culex pipiens*
		C. theileri
		C. univittatus
		Eretmapodites chrysogaster
		Mansonia spp.
		Aedes spp.

Fig. 1. Map of the world showing the average January and July position of the Intertropical Convergence Zone (ITCZ), the direction of warm winds between and north and south of the ITCZ positions (arrows) and the Rift Valley Convergence Zone (RVCZ). In the United States of America the arrows represent the three routes taken by insects carrying vesicular stomatitis virus. The viruses and locations discussed in the text are indicated as follows: JE = Japanese encephalitis; MVE, Murray Valley encephalitis; WEE, Western equine encephalitis; EEE, Eastern equine encephalitis; WN, West Nile encephalitis; AHS, African horse sickness; BT, Bluetongue; IB, Ibaraki; BEF, bovine ephemeral fever; VEE, Venezuelan equine encephalitis; YF, yellow fever; RVF, Rift Valley fever. O, Onderstepoort, South Africa; I, Ibadan, Nigeria; V, Vom, Nigeria; SLE, Saint Louis encephalitis.

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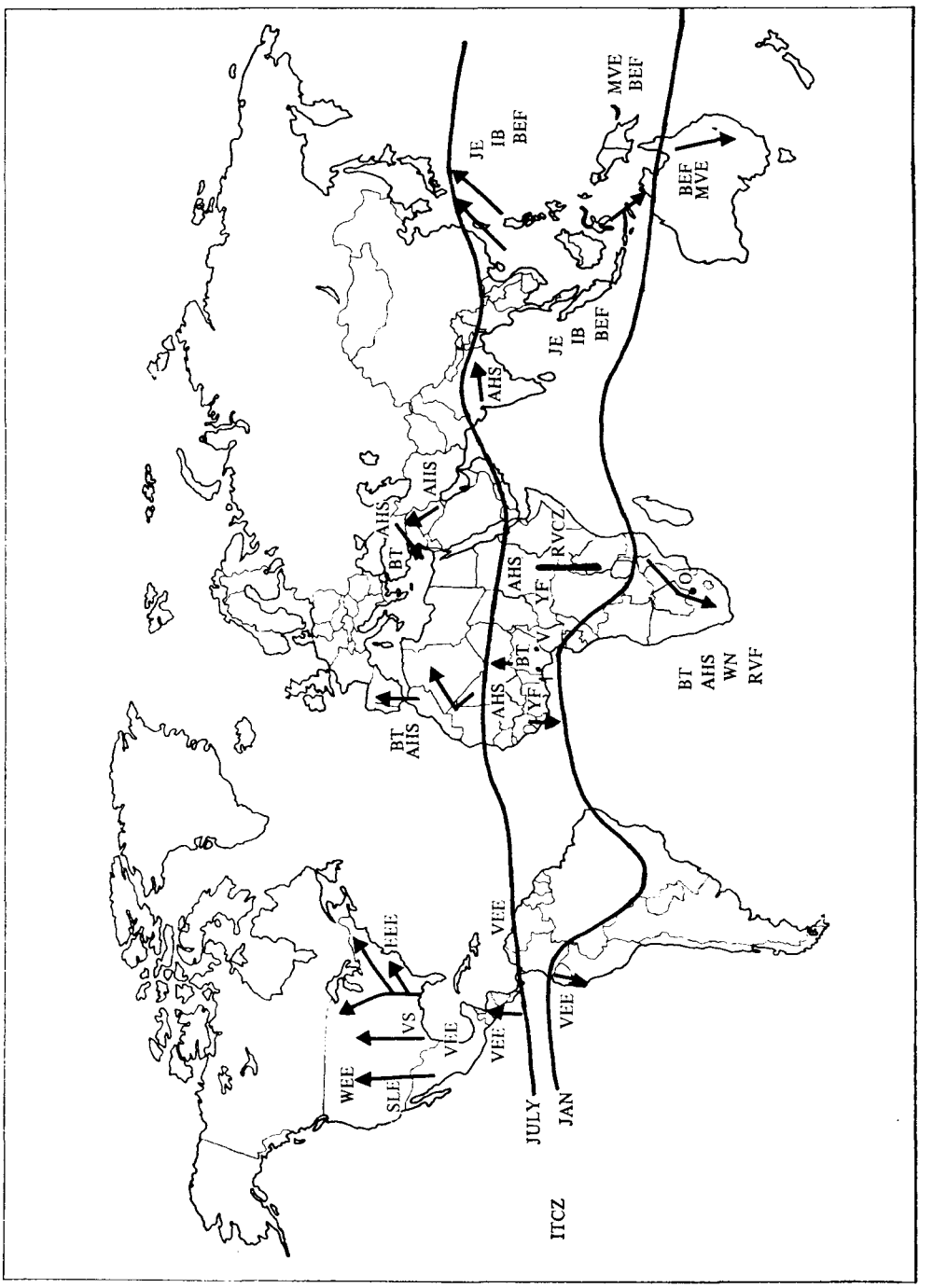

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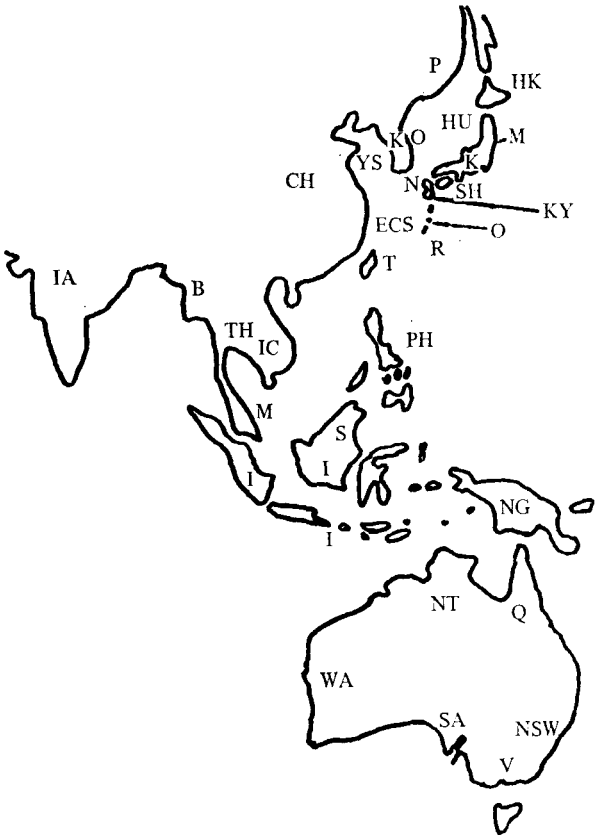

Fig. 2. Location map of Eastern Asia and Australia showing places discussed in the text. P, Primorsk region of Siberia, USSR; HK, Hokkaido, Japan; HU, Honshu, Japan; M, Miyagi, Japan; K, Kyoto, Japan; SH, Shikoku, Japan; N, Nagasaki, Japan; KY, Kyushu, Japan; O, Okinawa, Ryukyu Islands; R, Ryukyu Islands; KO, Korea; CH, China; YS, Yellow Sea; ECS, East China Sea; T, Taiwan; PH, Philippines; IA, India; B, Burma; TH, Thailand; IC, Indo-China; M, Malaysia; S, Sarawak; I, Indonesia; NG, New Guinea; NT, Northern Territories, Australia; Q, Queensland; WA, Western Australia; SA, Southern Australia; NSW, New South Wales, Australia; V, Victoria, Australia.

(2) Main host–vector cycle in mammals and midges or mosquitoes – African horse sickness, bluetongue, Ibaraki, bovine ephemeral fever and Venezuelan equine encephalitis viruses and in addition vesicular stomatitis virus (Tables 3, 4).

In some instances detailed epidemiological evidence of the timing, location and spread of a virus is lacking, but analogies can be made with the behaviour of other viruses, their hosts and vectors present in a particular area. Viruses often share a common location or method of spread and appear at similar times of the year; species of insects act as vectors of more than one virus, and individuals from different species of insects are carried on the same or similar winds.

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Table 5. *Date of first detection of Japanese encephalitis virus or antibody in mosquitoes or pigs in Ryukyu Islands and Japan*

	Site of sample			
Year	Okinawa 26° 30′ N, 128° E	Nagasaki 33° N, 130° E	Kyoto 35° N, 136° E	Miyagi 38° 30′ N, 141° E
1965	—	30 May	23 July (54)*	27 August (35)
1966	11 April	21 June (71)	20 July (29)	1 September (43)
1967	2 May	6 June (35)	10 July (34)	15 August (36)
1968	15 May	18 July (64)	23 July (5)	10 September (49)
1969	16 April	9 July (84)	17 July (8)	15 September (60)
1970	2 June	15 July (43)	4 August (20)	10 September (37)
1971	4 May	12 July (69)	6 August (25)	—
1972	27 June	16 August (50)	31 August (–16)	10 September (42)
1973	6 June	9 July (33)	15 July (6)	15 September (62)

Dates from Hayashi *et al.* (1978*a*), Ura (1976), Ishida *et al.* (1976) and Maeda *et al.* (1978).
* Figures in parentheses indicate the time in days after the detection date in the preceding column.

BIRD–MOSQUITO CYCLE

(i) *Japanese encephalitis*

Japanese encephalitis virus circulates in pigs and birds as hosts and in mosquitoes as vectors, man and horses being indicator hosts of the disease (Table 2).

Evidence of the disease or virus (isolation or presence of antibodies) has been found in Japan, Korea, the Primorsk region of Siberian Russia, China, Taiwan, Indo-China, Thailand, Burma, India, Malaysia, Indonesia and the Philippines (see Figs. 1, 2). Most work on the disease and virus has been done in Japan, Taiwan, Korea, Sarawak and Indonesia.

At the most northern part of the affected area disease or evidence of infection is not found every year. In some years there are epidemics. In Hokkaido, North Japan, a greater degree of infection was found when the mean mid-day temperature was 20 °C or higher in summer for a longer period than usual (Yamada, Hashimoto & Kanamitsu, 1972). In other parts of Japan the virus is found nearly every year. The earliest date of detecting virus by isolation from mosquitoes or by detecting a rise of antibody in pigs varies from year to year. In addition this date varies with the site sampled and is earliest in the southwest of Japan and latest in the northern parts of Honshu (Buescher & Scherer, 1959; Fukumi *et al.* 1975; Hayashi, Mifune & Shichijo, 1965; Hayashi *et al.* 1978*a*, Ishida *et al.* 1976; Maeda *et al.* 1978; Wada *et al.* 1975). Table 5 shows the dates for various parts of Japan as well as for the island of Okinawa which lies south-west of Japan in the Ryukyu group. The latest date for the disappearance of virus also varies from one part of Japan to another, and there is an interval from October to May each year, when no virus can be detected. Many suggestions have been put forward for the method of overwintering. It has recently been demonstrated that transovarial transmission of Japanese encephalitis virus can take place (Rosen *et al.* 1978). Other suggestions are that virus in mosquitoes emerging from hibernation starts to multiply in them once these mosquitoes have taken a blood meal

in the spring. It is also suggested that migratory birds re-introduce virus each year. However, it has been shown that the appearance of overwintered females in each year bears no relation to the first appearance of virus. In Nagasaki overwintered females appeared in the last two weeks of March or the first of April, whereas the first appearance of antibody in pigs or isolation of virus from mosquitoes occurred from 30 May to 16 August (see Table 5 for the dates) (Wada *et al.* 1975). Migrant birds arrive in March but no virus is detected until the end of May or early June (Hayashi *et al.* 1965). Overwintering in local hosts has been suggested but this does not account for the interval between the presence of mosquitoes and hosts and the detection of the virus.

Despite the arrival of birds and the emergence of female vectors, Japanese encephalitis does not occur until after the onset of south-west winds, suggesting that virus must be freshly introduced each year. The south-west winds result from the north-easterly movement of warm air at the start of the Bai-U season, normally around 15 June (Arakawa & Taga, 1969). After the onset of the season the south-westerly winds regularly occur and it is these winds that are most likely responsible for bringing mosquitoes infected with Japanese encephalitis virus to south-west Japan. For example, in 1966, evidence of infection first detected between 21 and 24 June was preceded by south-west winds on the 15 and 16 June. In 1964, however, infected mosquitoes were found in Aino, Nagasaki prefecture, on 19 May (Takahashi *et al.* 1965). Between 7 May and 10 May 1964, a front moved north-east and lay at 33° N, 131° E; this was followed by a further front on 17 and 18 May. South-west winds associated with these fronts most likely brought infected mosquitoes to south-west Japan. There are also instances of the date of first infection in south-west Japan later in the year. In 1970, 1972 and 1974 evidence of infection was not found until the second half of July or early August, however, south-west winds had occurred in those years from about 11 June.

These findings suggest that infected *C. tritaeniorhynchus* are introduced each year and that infection in south-west Japan comes from mainland China and the Ryukyu Islands. Late detection of infection in Japan probably means that in China and the Ryukyu Islands infection with Japanese encephalitis to a sufficient level occurred later in a year. Kisimoto (1971) demonstrated that catches of planthoppers in the East China Sea were associated with south-west winds in the Bai-U season. Since 1964 female *C. tritaeniorhynchus* mosquitoes have been caught on ships around Japan in the East China Sea, Yellow Sea and West Pacific Ocean (Asahina, 1970; Hayashi *et al.* 1978*a*, Hayashi, Suzuki & Asahina, 1978*b*).

Virus infection in most years coincides with the presence of nestling birds and young pigs which have lost their maternal antibody. The nestlings and pigs are thus readily available for virus multiplication.

This suggests that the epidemiology of Japanese encephalitis in Japan is as follows. Virus is introduced each year by infected *C. tritaeniorhynchus* females migrating on the warm south-west winds from further south. They infect young pigs and/or nestling birds and initiate a cycle of infection. If the local numbers of mosquitoes are sufficient, the virus will spread into man or horses, causing disease. At the same time the virus is carried further north on winds to other

parts of Japan. At the end of the season migratory mosquitoes, some of which have been caught on north-east winds in the Pacific at 29° N, 135° E (Asahina, 1970), or migratory birds, coming from or passing through areas where the virus is circulating, will carry the virus further south. The time of the year the virus is introduced depends on the warm winds and on the extent of infection further south.

Similar conditions to those in Japan may exist in Korea where disease starts in different places each year (Kono & Kim, 1969).

In Amami and in Okinawa and in parts of Taiwan, evidence of virus infection is found mainly during the late spring and summer, but virus is present for a longer period and has been isolated during the winter (Hayashi *et al.* 1978*a*).

Further south in other countries such as Burma, Thailand and India (Arora & Singh, 1974) first evidence of virus infection is found at the beginning and end of the rainy season, i.e. May or August or September, coinciding with the advance and retreat of the south-west monsoon. In Saigon there are two periods – one from June to August and another from January to April – coinciding with south-west and north-east monsoons respectively.

In the endemic areas of Indonesia and Sarawak the virus is maintained in a mosquito–pig and mosquito–bird cycle (Simpson *et al.* 1974; Simpson *et al.* 1976; Van Peenen *et al.* 1975*a*, *b*). The species of mosquitoes involved are *C. gelidus*, which appears to be non-migratory and maintains its population during the year, and *C. tritaeniorhynchus*, which has peaks of population in October to January and April to July.

During these two periods virus is isolated from mosquitoes and antibody conversion occurs in pigs. If it is accepted on the evidence for introduction of virus to Japan that *C. tritaeniorhynchus* mosquitoes are migratory and are carried on the wind, then it is likely that these mosquitoes are carried on the wind in the more southerly areas such as Indonesia, Malaysia and other parts of Southeast Asia. Thus it is probable that in Indonesia and Malaysia mosquitoes are carried NE on the south-west winds of the ITCZ during May to August and SW on the north-east winds of the ITCZ during November to February, thereby introducing infection from one area to another during these movements.

Given, therefore, a static mosquito–pig/bird cycle with *C. gelidus*, *C. annulus* (Taiwan) or *C. fuscocephalus* (Tawain) as vectors and a migratory mosquito–pig/bird cycle with *C. tritaeniorhynchus* or *C. vishnui* (India) as vectors, a number of zones can be identified in the areas where Japanese encephalitis is found.

Zone A. This is a zone where a permanent cycle of virus infection is maintained by a mosquito–pig cycle (*C. gelidus*). *C. tritaeniorhynchus* also breed during the year, their peaks coinciding with the north-east and south-west monsoons. *C. tritaeniorhynchus* are shallow pool breeders (occupiers of temporary habitat) and migrate with the movement of the ITCZ to the south and north of the zone and outside the zone. When infected with virus, the *C. tritaeniorhynchus* mosquitoes convey the virus within the zone and beyond it. This zone includes Indonesia and Malaysia.

Zone B. This is a zone where there is a wet and dry season. Infection is brought

into the zone by migratory *C. tritaeniorhynchus* from zone A, but the virus can be maintained during the dry season since there are sufficient numbers of vectors and hosts to maintain a cycle at a low level. Examples of this zone would be parts of Thailand, Burma, Indo-China and South China.

Zone C. In this zone there is a warm summer but a short cooler period during the winter during which the numbers of vectors are greatly reduced. Infection is introduced by *C. tritaeniorhynchus* each year. Young pigs and nestling birds are infected and the virus passes from them into other host–vector cycles. Sometimes the weight of infection is so great that survival of virus may occur during the cooler period in surviving adult mosquitoes, by transovarial transmission or in certain hosts. Examples are Taiwan, parts of China and the Ryukyu Islands.

Zone D. This zone has a warmer summer but a cold winter, too long for the virus to overwinter in hibernating adult vectors or in local hosts.

Infection is introduced by *C. tritaeniorhynchus* each year. At the time of introduction there is an abundance of hosts (young pigs and nestling birds of migratory or local species.) The time of introduction depends on the warm south-west winds (Bai-U rains) and on the presence of virus in zones B and C. Examples of this zone are Japan and Korea.

Zone E. This zone has a cool summer and a cold winter, an unsuitable habitat for the vectors of Japanese encephalitis; introduction of infection is rare and depends on the incursion further north than usual of warm winds bringing infected mosquitoes. Infection is present for a short period and may not occur again for a number of years. This zone lies further north than zone D.

Zone F. This zone has a cool summer and a very cold winter. No Japanese encephalitis occurs in this zone, which is unsuitable for vectors and is too far north for vectors to be introduced on warm winds. Pigs or migratory birds infected with virus or having antibody may enter the zone, but even in summer no transmission of virus takes place. This zone includes northeastern Siberia.

In zones B, C and D at the end of the season, infection is carried south by infected mosquitoes on the north-east monsoon or by migratory birds.

Thus there is a dynamic system with movement of migratory mosquitoes on the south-west and north-east monsoons and on warm winds. The migratory mosquitoes join mosquitoes of their own species which have overwintered in a particular zone and thus the local species population is maintained. There is a renewal each year of susceptible hosts in the form of young pigs or nestlings of local or migratory birds. The virus multiplies in the vectors and in the hosts and is carried by migratory mosquitoes and migratory birds from one area to another; thereby its survival is ensured. In some areas a cycle is maintained all the year; in some, the carriage of virus in infected hosts or vectors reinforces virus that has survived by overwintering in vectors or by transovarial transmission; in others the virus is introduced to areas from which it has disappeared or which it has not started to colonize. The behaviour of the virus is thus adapted to an optimum method of survival.

(ii) *Murray Valley encephalitis*

The presence of Murray Valley encephalitis virus has been demonstrated in Australia and New Guinea (see Figs. 1 and 2). Like Japanese encephalitis, the hosts are birds and the vectors mosquitoes, mainly *Culex annulirostris* and *Aedes vigilax* (Table 2). Man is affected on occasion and suffers disease. Australian X disease is believed to be Murray Valley encephalitis (Anderson, 1954; French, 1973; Doherty, 1974). Antibodies to Murray Valley encephalitis virus have been found in Indonesia (Kanamitsu *et al.* 1979).

Epidemics of these diseases, under one or other of these synonyms, occurred in Queensland and New South Wales in 1916–17, 1917–18, 1922 and 1925, in the Murray Valley of New South Wales, Victoria and South Australia in 1950–1, and in Victoria, New South Wales, Queensland, Northern Territory and northeast Western Australia in 1974. Abnormally heavy rainfall over central Queensland and western New South Wales in October–November of 1916, 1917, 1924, 1950 and 1974 has been correlated with outbreaks the following summer; in 1953 and 1961, however, when there was also heavy rainfall, epidemics did not result. Evidence of virus has also been found in the south of Australia in 1956, 1957 and 1971.

Similarities can be found with Japanese encephalitis, and Murray Valley encephalitis essentially occupies the same habitats in the southern hemisphere as Japanese encephalitis in the northern. There are bird–mosquito cycles occurring in eastern Indonesia and in New Guinea. Migratory mosquitoes carry the virus south to northern Australia with the north-west monsoon. From there in certain years, northerly winds such as were responsible for the spread of bovine ephemeral fever (Murray, 1970) carry the infected mosquitoes south to the Murray Valley, where, with the increase in number of mosquitoes due to moist conditions already present, the virus multiplies in local birds and is transferred to man. At the end of the season the virus is carried north again in infected migratory mosquitoes or migratory birds.

Zone A (as described for Japanese encephalitis) would be in Indonesia and New Guinea; the other zones would extend from north Australia, south to Victoria, South Australia and New South Wales. Movement of virus out of zone A or from zone B further south may not occur every year but depends on the state of the virus cycle in these two zones.

(iii) *Western equine, Eastern equine and Saint Louis encephalitis*

Western equine (WEE), Eastern equine (EEE) and St Louis (SLE) encephalitis viruses circulate in mosquitoes and birds and spread on occasions to man (all three viruses) and horses (WEE and EEE). The vectors are listed in Table 2.

Evidence of virus (isolation or presence of antibody) for all these diseases has been found in North, Central and South America. Most work on the viruses has been done in the United States, Canada and the Caribbean, but little apart from work at Belem has been done in the countries bounded by the limits of the Intertropical Convergence Zone.

Fig. 3. Location map of North, Central and South America showing places discussed in the text. The arrows shown in the United States of America (USA) indicate four of the routes taken by insects infected with St Louis encephalitis virus. Can, Canada; C, California, USA; F, Florida, USA; NO, New Orleans, USA; B, Brownsville, USA; MEX, Mexico; LT, Las Tablas, Mexico; G, Guatemala; S, El Salvador; H, Honduras; N, Nicaragua; CR, Costa Rica; COL, Colombia; VEN, Venezuela; EC, Ecuador; GU, Guayas, Ecuador; PE, Peru; BRA, Brazil; GJ, Guajira (Venezuela & Colombia).

These viruses occupy the same habitats in North, Central and South America as Japanese encephalitis and Murray Valley encephalitis viruses do in Asia and Australia. However, work on Japanese encephalitis was done in an area dominated by islands whereas work on WEE, EEE and SLE was done on a land mass represented by USA and Canada. Analysis of the various factors affecting the spread of disease is thus more difficult.

(*a*) *Western equine encephalitis*

Western equine encephalitis is distributed widely in North America (see Figs. 1, 3). Epidemics of the disease in horses have occurred in Saskatchewan, Alberta and Manitoba in Canada, in western and central United States, but rarely in eastern United States (Hanson, 1972; Hayes & Wallis, 1977). Epidemics have also occurred in man, for example, in 1941 in northern United States and western Canada and again in 1975 in Manitoba (Lillie, Wong & Drysdale, 1976; Waters, 1976). Essentially the virus circulates in mosquitoes (*Culex tarsalis*) and in birds, especially the house sparrow (Table 2).

Antibody development in sentinel chickens has been investigated at various places in North America. In 1965 antibody to WEE virus was first detected in

Texas, then Colorado and finally in Saskatchewan (Hess & Hayes, 1967). The delay in appearance of antibody could be due to the time taken for the virus to move north or represent later emergence of mosquitoes and virus after overwintering.

Investigations in Texas show that the virus circulates between *Culex tarsalis* mosquitoes and nestling birds. In studies in Hale County, Texas, from 1965 to 1969 (Holden *et al.* 1973) WEE virus was first detected between 12 June and 2 July; before then there was no evidence of infection in mosquitoes or nestling birds.

The time relationship of emergence of adult mosquitoes, the presence of nestlings and the detection of virus has been followed in a swamp in Maryland. In 1969 the sequence began with the emergence of adult mosquitoes during the spring together with the arrival of migrant adult birds. By May and June migrant and resident birds were nesting. Virus activity was not detected until 15 July, about 3 weeks after the first nestlings were found (Dalrymple *et al.* 1972). Similar findings were made in 1966 and 1967 (Muul, Johnson & Harrison, 1975).

In both Texas and Maryland, the interval between the appearance of mosquitoes or migratory birds and the detection of virus would appear to be too long for overwintering to have occurred. The introduction of virus by migratory mosquitoes on warm winds would coincide with the timing.

In other areas (California, Colorado and Florida) virus isolations have been made from *C. tarsalis* or from *Culiseta melanura* mosquitoes collected during the winter (Hess & Hayes, 1967; Wellings, Lewis & Pierce, 1972; Hayes & Wallis, 1977).

(*b*) *Eastern equine encephalitis*

Eastern equine encephalitis virus is found mainly in the eastern half of North America (see Figs. 1, 3), but virus has been isolated in Texas, Michigan, Wisconsin and Alberta (Hanson, 1972). Epidemics in horses have occurred in the Atlantic coast states. The main cycle is in birds and in *Culiseta melanura* mosquitoes, but *Aedes sollicitans* and *Aedes vexans* mosquitoes are also involved (Table 2). Work done in a swamp in Maryland indicated that the appearance of virus occurred after the emergence of adult mosquitoes and the arrival of migrant birds but coincided with the time of the emergence of nestlings (Saugstad, Dalrymple & Eldridge, 1972; Muul *et al.* 1975). As mentioned before, WEE was also found at this time and again the period of lack of virus activity is too long for overwintering of virus in mosquitoes or local birds. *In Florida, however, EEE virus has been isolated from mosquitoes (Culiseta melanura and Culex spp) during the winter* months and cases of disease in horses have been found in all months of the year (Wellings *et al.* 1972; Bigler *et al.* 1976).

(*c*) *St Louis encephalitis*

Cases of St Louis encephalitis in man have been reported from most states of the United States (Luby, Sulkin & Sanford, 1969; Creech, 1977; McGowan, Bryan & Gregg, 1973) (see Figs. 1, 3). Epidemic years were noted from time to time, the most recent being 1974–5. In 1975 St Louis encephalitis was found in

southern Ontario in Canada in man for the first time (Spence *et al.* 1977). The virus circulates in a rural cycle in *Culex tarsalis* mosquitoes and in birds, in a town cycle in mosquitoes of the *Culex pipiens* complex and in birds and man and, in Florida, in *Culex nigripalpus*–bird cycles (Table 2). In the 1975 epidemic several states were infected about the same time but in general the disease appeared to move northwards along the Mississippi and Ohio river basins.

Isolations of the SLE virus are not so widespread as those of WEE and EEE. For example, in Florida SLE was isolated in the epidemic years in 1962 and 1977 and on three occasions in October and November 1969 (Wellings *et al.* 1972). In California it has been isolated in many years (Luby *et al.* 1969) and more recently has been demonstrated between April and December 1972 in Arizona, New Mexico, Colorado and Texas (Hayes *et al.* 1976). In California, where WEE virus was isolated from mosquitoes collected in all months except December, SLE virus was limited to collections from June to September with one isolation in March.

In 1975 studies were carried out in Memphis, Tennessee, before and during the epidemic in that area (Levy *et al.* 1978). The first signs of SLE infection were the development of antibody in juvenile sparrows in early May (around the 13th) and the isolation of virus from *Culex restuans* mosquitoes between 18 and 31 May. Thus there was an interval between the appearance of adult mosquitoes and migratory birds in April and virus infection in May. The situation may be similar to that with WEE and EEE in that virus or antibody is not detected until susceptible nestlings are available, and virus may be brought to the area by infected mosquitoes on warm winds.

In 1975 a widespread epidemic of SLE occurred, with over 2000 reported cases of human disease, mainly throughout the eastern two-thirds of the USA. The earliest cases in man were at the end of June and the latest in October. In May and June high temperatures associated with warm winds from the south were found in the eastern parts of the United States and temperatures higher than normal occurred in August and September. The pattern of spread suggests that warm winds were probably responsible for dispersing infected mosquitoes through the states in the Mississippi and Ohio river valleys and into Canada.

As with vesicular stomatitis (see later) outbreaks of SLE are found in certain areas and appear to follow recognisable routes as they progress northwards. These areas or routes for SLE are (i) California, (ii) Arizona, New Mexico, Colorado and further north, (iii) states of the Mid West from west Texas northwards, (iv) the Mississippi and Ohio River valleys, (v) the north-eastern states from Georgia north-eastwards and (vi) Florida (Fig. 3). In 1972 SLE was in the west, in 1974–5 mainly in the central states (route iv) and 1977 in Florida. This suggests that, as with vesicular stomatitis (see later), the origin of the SLE virus may be further south in Mexico, Central America and the Caribbean.

Culex tarsalis is a vector common to both SLE and WEE. *Culex tarsalis* breeds especially in irrigated areas, in other words temporary habitats, and is known to be carried on the wind. WEE has been found further north and more widespread than SLE. However, the routes followed by SLE may also be followed by WEE. It is therefore suggested that *Culex tarsalis* mosquitoes infected with WEE or

SLE would be carried on warm winds northwards to North America from further south.

The three viruses, WEE, EEE and SLE have been found in Mexico, Central America, the Caribbean and northern South America. The isolations were made between May and November, but cases of disease or isolations of virus have occurred in December and February. Winds responsible for carriage of infected mosquitoes may have been similar to those responsible for the spread of Venezuelan equine encephalitis (see later).

Many of the features discussed for WEE, EEE and SLE (arrival of mosquitoes infected with virus coinciding with warm southerly winds and presence of susceptible nestlings) are similar to those for Japanese encephalitis and thus zones based on climate, local cycles and migratory cycles involving mosquitoes and birds can be put forward.

Zone A probably lies in the equatorial region in northern South America and in parts of Central America – here the virus is maintained in a local mosquito–bird cycle. The migratory phase is represented by *C. tarsalis* for WEE and SLE and *Aedes vexans* and *Aedes sollicitans* for EEE.

Zone B lies in the Caribbean and Central America. Virus can survive through the dry season in a local cycle, but mosquitoes migrating to and fro reinforce the virus in the area and migratory birds bring the virus from further north. For SLE this probably represents zone C as well.

Zone C is probably represented for WEE and EEE by California, and by Florida and some other Gulf States. Virus is introduced each year but virus can maintain itself in local cycles during adverse conditions; continuity is, however, ensured by reintroduction.

Zone D is represented by the central latitudes of the United States. In the case of WEE and EEE, virus is introduced each year carried by migratory mosquitoes borne on warm winds. The arrival of the virus occurs about the time that nestling birds are hatched and these provide a source of growth of virus, which later in the season spills over into the horse or man–mosquito cycle. At the end of the season the virus may overwinter in some years but it is also carried south again in migratory mosquitoes and in migratory birds travelling south.

Zone E is represented probably by parts of Canada or parts of the United States with a short summer. The virus is introduced at times, stays for a short period, but does not come again for a number of years, unless warm winds carry the virus further north. Zone F is represented by other parts of Canada.

As with Japanese encephalitis there is thus, in the northern hemisphere, a constant movement north of migratory mosquitoes carrying virus coinciding with warm winds, and movement south again at the end of the season by mosquitoes and birds (Lord & Calisher, 1970). In certain parts in zone B and zone C virus can persist in local mosquitoes, birds, bats or other animals, but reintroduction of virus also takes place. The viruses have been isolated in Brazil and Argentina and other parts of South America, but details are lacking for similar zones to be demonstrated.

MAMMAL–MIDGE/MOSQUITO CYCLE

(i) *African horse sickness and bluetongue*

(*a*) *African horse sickness*

In African horse sickness horses, mules or donkeys act as hosts and *Culicoides* midges as vectors (Table 3). Disease is seen mainly in horses especially in those horses imported into countries where the virus circulates each year and in horses infected as a result of introduction of virus to new areas.

Evidence of infection with African horse sickness virus has been found in most of Africa south of the Sahara and in countries bordering the Red Sea. Epidemics outside these areas have occurred from time to time – in Egypt in 1928, 1943–4, 1953 and 1971, Palestine 1944, Cape Verde Islands 1944, Middle East and Indian sub-continent 1959–61, North Africa and southern Spain 1965–6 (Sellers *et al.* 1977 (see Fig. 1)). The disease has also occurred frequently in South Africa. Epidemics in the Cape were seen in 1780, 1811, 1819, 1839 and 1854/5 as well as in 1913/14 and 1918 (Theiler, 1921).

Evidence of wind-carriage of infected midges was found for the spread of African horse sickness from Morocco to Spain (1966), Turkey to Cyprus (1960), from Senegal to Cape Verde Islands (1943), through the Middle East (March to June 1960), from the Sahara to North Africa (1965) and from Pakistan to India (1960). The winds were warm (temperatures 15–25 °C by night or 20–40 °C by day), and similar winds had been shown in past years to have carried locusts or moths in these areas. The winds described were apart from those across the Sahara outside the boundaries of the ITCZ. Within the boundary of the ITCZ disease also occurs especially in horses introduced from abroad. In West Africa disease has been found during September–November after the end of the rainy season (Best, Abegunde & Taylor, 1975). In Sudan two peaks have been noted in June and in September–November, again associated with the beginning and end of the rainy season (A. W. Chalmers, personal communication, 1975; Eisa, 1974). In Sudan Ceratopogonidae have been captured in winds associated with the movement of the ITCZ (Bowden & Gibbs, 1973).

No analysis has been done on the spread of African horse sickness in South Africa, but appearance of the disease further south is associated with the heavy rains affecting the high veld. This suggests a similarity to the appearance of Rift Valley fever, West Nile and bluetongue viruses in these areas.

(*b*) *Bluetongue*

With bluetongue, virus is transmitted by *Culicoides* midges as vectors with sheep, cattle, goats and wild ruminants as hosts (Table 3).

The distribution of the virus is based on the habitats of the vectors, *Culicoides variipennis*, *C. imicola*, *C. brevitarsis* and other species and includes North, Central and South America, Africa, Middle East, Indian subcontinent, South-east Asia and parts of Australia.

Clinical signs of disease are uncommon in cattle and goats. In sheep disease is found in exotic breeds introduced to tropical areas and, in local breeds, on the

edge or fringe of an area where bluetongue is usually present. Such edges or fringes are found at higher altitudes (e.g. Kenya) or at higher latitudes (e.g. Cyprus, Turkey, Portugal and Spain) in the northern hemisphere and Cape Province, South Africa, in the southern.

The origin of virus in some of these fringe areas has been analysed. In Portugal it was found that southerly winds, unusual for that time of year, would have carried infected midges from Morocco to Portugal in June 1956 (Sellers *et al.* 1978). In Cyprus in 1965 north-easterly winds in September were probably responsible for bringing infected midges from Turkey and Syria to the island, and in August 1977 north-easterly winds carried infected midges to Cyprus (Sellers *et al.* 1979).

In Cyprus the population of midges starts to increase in mid-April and reaches a peak in mid-May. A further peak is reached in September and October (J. Boorman quoted by Sellers, 1975). However, despite the previous presence of midges, first outbreaks of bluetongue have been noted in September and October in 18 of the 24 years in which bluetongue occurred since 1924. Thus, in these years as well as in 1965 and 1977, it is likely that virus was introduced to the island by infected *Culicoides* midges on the wind. In the three years when an outbreak was noted in May, it was possible that the virus had overwintered in sheep or goats or in adult *Culicoides*.

From 1963 to 1967 Nevill (1971) studied the variation in numbers of *Culicoides* and the date of first appearance of virus in *Culicoides* and in cattle at Onderstepoort, South Africa. During the winter months, from May to August, the numbers of *Culicoides* caught were low, but during the 5 years of trapping, midges were caught on 1–80 nights during these months. Despite an increase in midge population in August, presence of virus in cattle or in midges was not detected until 15 December (1964), 18 November (1965) and 28 September (1967) at the earliest. Thus there was a gap between the emergence of adult *Culicoides* and the isolation of bluetongue virus, as was shown with mosquitoes and the viruses of Japanese, Western equine, Eastern equine and St Louis encephalitis. Nevill (1971) believed that the virus could overwinter in infected adult midges and cattle and that failure to isolate the virus earlier was the result of insufficient sampling; however, in 1974 there was an extension of bluetongue into the Cape associated with other insect-transmitted diseases – Rift Valley fever, West Nile and African horse sickness. This was preceded by north-easterly winds, which were probably responsible for carrying midges infected with bluetongue virus.

In Africa and Western Asia, African horse sickness and bluetongue viruses are transmitted by the same species of midge, *Culicoides imicola*. Wind-carriage of infected midges has been shown to bring African horse sickness and bluetongue to the Iberian peninsula and to Cyprus. African horse-sickness virus has also been shown to be carried by infected midges in south-easterly winds in the Euphrates–Tigris valley, on the ITCZ in Algeria and by westerly disturbances in India and Pakistan. It would be likely that in some years similar winds would have carried midges infected with bluetongue virus.

Less work has been done on the analysis of the spread of bluetongue in areas

between the northern and southern limits of the ITCZ. However, Bowden & Gibbs (1973) caught *Culicoides* at heights up to 80 m during the passage of the ITCZ in October in the Sudan. In Nigeria, studies on *Culicoides* and on infection with bluetongue have been carried out in Ibadan and at Vom. At Ibadan, Dipeolu (1977) captured *Culicoides* throughout the year and found two peaks in numbers in April and in October. At Vom, *Culicoides* were caught throughout the year. Numbers of *C. imicola* (a vector of bluetongue) were at a peak in August and September (J. Boorman, personal communication, 1977).

Development of antibodies to bluetongue in calves and heifers at Vom was followed throughout the year. Rise in antibodies to bluetongue (seroconversion) was found in all months of the year except March and July. Peaks of seroconversion occurred in May and June and in September, October and November (Taylor & McCausland, 1976; W. P. Taylor & K. A. J. Herniman, personal communication, 1978). Seroconversion to some types occurred only once a year; in animals that developed antibodies to more than one type, the interval between seroconversion to the different types varied from 1–7 months. Thus it is likely that new types of bluetongue virus are introduced into a herd and that this occurs most often during the passage of the ITCZ northwards in April and May and southwards in August and September.

Analogies can be made between the behaviour of bluetongue and African horse sickness and that of Japanese encephalitis. In Cyprus, Portugal, Spain and South Africa, disease results from the carriage of infected midges on warm winds; in Cyprus and South Africa virus infection occurs some time after the emergence of overwintering adult midges; in the tropics between the limits of ITCZ there is carriage of midges on the wind and the peaks of infection occur at the time of passage of the ITCZ. Zones similar to those of Japanese encephalitis can be delineated. However, in zone A (rainfall greater than 50 mm all the year) the density of the horse and ruminant population is less than in zone B. The zones can be defined as follows:

Zone A. In this zone the climate is warm and humid most of the year with rainfall over 50 mm every month. Thus numbers of *Culicoides* midges are present all the year and a cycle of infection can be maintained in *Culicoides* and in local animals. The new crop of young animals become infected after the loss of maternal antibody but the virus is adapted to the host and local animals do not show disease. Cycles of infection move through the areas, occasioned by movement of hosts and vectors. Horses or exotic breeds of sheep introduced to these areas show disease. This zone includes Malaysia and Indonesia, the Congo region of Africa, the Amazon region of South America and tropical forests of Central America.

Zone B. In this zone there are wet and dry seasons. Infected *Culicoides* move to and fro with the movement of the ITCZ and other winds; movements of nomadic herdsmen and their animals in order to take advantage of fresh grazing help in the maintenance and spread of virus. In addition, in static herds and flocks, young ruminants or foals are born throughout the year, and provide hosts for the midges and for the viruses. The virus circulates in the host–vector cycle and, under adverse conditions, may survive either in adult *Culicoides* (hiber-

nating or aestivating) or in blood of ruminants or equidae. Peaks of infection occur at the beginning and at the end of the rainy season when migrations of *Culicoides* occur on the movements of the Intertropical Convergence Zone. As in zone A, the local animals do not show clinical disease but breeds of sheep and horses introduced show signs of disease. Zone B probably includes most of the countries between the northern and southern limits of the ITCZ, e.g. West Africa, Southern Sudan, parts of Kenya, Central Africa, northern parts of Southern Africa, India, Pakistan, parts of S.E. Asia, northern Australia, Central America, Mexico and parts of Texas. Each year on suitable winds, virus would be carried between zones A, B and C.

Zone C. In this zone there is a cooler winter. The summer may be dry or may be wet. Bluetongue or African horse sickness is introduced every year on warm winds or animals from zone B. At the same time the climate is such that in most years the virus can overwinter in adult *Culicoides* or in the blood of mammalian hosts, or in some cases by transplacental transmission. Virus is carried back again at the end of the season in migrating animals or migrating *Culicoides*. Examples of this zone for bluetongue are the Middle East, California and parts of South Africa.

Zone D. Zone D is similar to zone C except that the winter may be cooler. Virus is introduced to the zone by midges carried on the wind or by movement of animals; it may persist through one or two winters, disappear and be reintroduced again. Disease is usually seen in the sheep. Examples of this zone are Cyprus, Western Turkey, the high altitudes of Kenya, parts of the United States, Cape region of South Africa and possibly parts of Australia.

Zone E. In zone E virus is introduced rarely, e.g. once every few years by infected midges carried on the wind or by movement of animals. Disease occurs in the sheep. If there is sufficient weight of infection, the virus may persist, but as the numbers of vectors diminish as a result of the long winter, the disease dies out; the opportunity for reintroduction may not occur for a number of years. The best example of this is Spain and Portugal but this may also have occurred in Canada.

Zone F. This is a zone where there is a long winter, and conditions during the summer are not suitable for the species of midge vector to breed. If infected animals are introduced, virus cannot be transmitted and therefore does not persist.

As with Japanese encephalitis, there is a dynamic system with virus moving with vector and host from one zone to another and back. The boundaries of the zone vary from year to year or differ with the virus and the cycle. Movement over longer distances is more likely to be through migrating midges than animals. Ruminants or horses move or are transported over shorter distances.

(ii) *Ibaraki and bovine ephemeral fever*

(*a*) *Ibaraki*

Ibaraki virus affects cattle, causing a disease very similar to bluetongue. In laboratory experiments virus multiplied in midges from a *C. variipennis* colony

(J. Boorman & M. Vorley, personal communication, 1977) and it is likely that it is transmitted by midges in the field (Table 3). It was first recognized in cattle in Japan in August 1959 and remained in that country until December 1959 (see Figs. 1, 2). It was probably seen previously in Japan from August to December in 1950 and 1951. In 1959 the first cases of the disease were in Miyazaki (228 cases) and Kagoshima (85 cases) prefectures in Kyushu district and in Kochi prefecture (4 cases) in Shikoku district (Omori *et al.* 1969), i.e. the south-west of Japan. No precise date in August was given but the pattern of the subsequent outbreaks suggests the latter half of August. The incubation period in calves is between 4 and 12 days with a mean of 8; if a 7- to 10-day incubation period in the midge is assumed, the disease would have come to Japan by infected midges between 8 and 18 days before the first outbreak (minimum 4 days, maximum 22 days). In the surface synoptic charts of the Japan Meteorological Office for August 1959, the path of the tropical storm Ellen can be tracked from 4 to 7 August. On 4 August at 12.00 GMT the eye of Ellen was at 24·5° N, 129° E. On 5 and 6 August it moved north and on 7 August the eye was at 31° N, 129° E in the East China Sea between the east coast of China and the south-west of Japan. This means that winds circulating anticlockwise around the centre of the storm would have brought infected midges from China to south-west Japan. The dates of the first outbreaks would have been between 15 and 25 August (8–18 days after 7 August).

In 1950 Ibaraki disease was also present in Japan from August onwards and bovine ephemeral fever was present at the same time (Omori, 1961; Inaba *et al.* 1968). On 27 July the centre of a tropical storm was at 29° N and 133° E and on 29 July it was at 31° N, 128° E; thus infected insects would have been carried on the wind from Taiwan or China to Japan. Later, on 19 August, a tropical depression lay at 31° N, 133° E and south-westerly and westerly winds would have taken infected midges further east in Japan.

Antibodies to Ibaraki virus have also been found in Indonesia (Inaba, 1975) and the virus is probably widely distributed in tropical and temperate zones in S.E. Asia.

(*b*) *Bovine ephemeral fever*

Bovine ephemeral fever virus circulates in cattle and buffaloes as hosts and in *Culicoides* (*Culicoides marksi*, *Culicoides brevitarsis*) and in *Culex annulirostris* as vectors (Davies & Walker, 1974; St George *et al.* 1977) (Table 3).

Evidence of disease has been found in Australia as far south as New South Wales, the northern part of Victoria and the north-eastern part of South Australia (St George *et al.* 1977). It has been seen in Papua New Guinea, Indonesia and Japan and is common throughout Africa and India (see Figs. 1, 2).

Murray (1970) and Newton & Wheatley (1970) correlated the spread of ephemeral fever in 1967–8 with warm winds. Initially, the north-west monsoon possibly blew infected insects from Indonesia; then low-pressure areas over inland Queensland caused warm winds to blow clockwise from the north and finally a high-pressure system over the Tasman Sea led to warm northerly winds

bringing infection to Victoria. In 1970–1 there was a further epidemic; in late 1970 and early 1971 the disease spread rapidly from northern New South Wales in December to reach Victoria in January. Similar outbreaks occurred in 1972–3 and 1973–4, with Queensland and New South Wales being involved. Between these periods of epidemics, cases occurred in Australia most months of the year and virus persisted, e.g. in Hunter Valley, New South Wales in 1972–3 (St George *et al.* 1977). No wind analysis has been done of spread since the 1967–8 epidemic.

Bovine ephemeral fever was also found in Japan at the same time as Ibaraki disease from August to December in the western and central parts of Japan (Inaba *et al.* 1968). Outbreaks were recorded in 1949, 1950, 1951, 1955, 1956 and 1958. In the 1950 outbreak the virus would have been brought to Japan by infected insects carried on the same winds as those responsible for bringing insects infected with Ibaraki virus, i.e. the tropical storm of 29 July at 29° N, 133° E. In the 1949 outbreak virus would have come as a result of tropical storm Faye, which travelled from 15 to 17 July from 27° N, 129° E to 31° N, 130° E; the infective insects would have come from China. Another tropical storm, Judith, travelled from 23° N, 128° E on 13 August to 30° N, 123° E on 15 August.

Long-distance carriage of infected insects on the wind most likely has been responsible for spread of bovine ephemeral fever in other countries. Bevan (1907) described the spread of bovine ephemeral fever in Rhodesia between January and March 1907, when the disease was found to spread a distance of 160 miles or more within 2 weeks.

In south-east Asia and Australia the distribution and movement of Ibaraki and bovine ephemeral fever viruses are similar to those of Japanese encephalitis and Murray Valley encephalitis viruses. Thus zones similar to those for these two viruses and for bluetongue virus can be identified to take account of the movement of infected midges on the ITCZ and on warm winds, movement of cattle and local cycles of persistence of virus in cattle, buffaloes and adult midges.

Zone A would be in Indonesia and Malaysia. Zones B, C, D, E and F would be the same as for Japanese encephalitis north of the equator and for Murray Valley encephalitis south of the equator.

(iii) *Venezuelan equine encephalitis*

The Venezuelan equine encephalitis (VEE) complex consists of four groups (see Johnson & Martin, 1974): the epidemic and endemic strains, Florida strain, Mucambo and Pixuna strain. In these groups the cycle is mosquito–mammal.

The endemic strains have been isolated in northern South America, Central America and Mexico. The epidemic strains have been found in Ecuador, Peru, Colombia, Venezuela, Trinidad, Central America, Mexico and United States (see Figs. 1, 3).

With the endemic strains the virus circulates in a small rodent–Culex mosquito cycle in the forest and the edge of the forest in regions of high or moderate rainfall well distributed throughout the year (Table 4). Virus of the epidemic strains circulates in a horse/man–migrating mosquito cycle, the commonest mosquitoes being *Aedes taeniorhynchus* and *Aedes sollicitans* (Table 4). The disease is found

in the dry areas of savannah, dry tropical forest, tropical thorn forest or in dry river valleys usually during the rainy season. The mosquitoes that act as vectors occupy temporary habitats and are carried long distances on the wind between these habitats. These mosquitoes probably carry infection over many kilometres.

Epidemics of VEE have occurred from time to time. In 1941 an epidemic occurred in Colombia; this was followed by another in the Guajira, Venezuela in 1942; during that year and in the following the disease spread eastwards across Venezuela to Trinidad. Movement of infected *Aedes taeniorhynchus* on the wind was thought to bring VEE to Trinidad in 1943 (Gilyard, 1945).

In 1969 VEE appeared on the Guatemala–El Salvador border and subsequently spread southwards to Honduras (1969), Nicaragua (1969, 1970) and Costa Rica (1970) and north-eastwards through Mexico (1969, 1970 and 1971) to Texas (1971). The first cases in Texas were seen at Live Oak on 23 June 1971 and at Brownsville on 1 July 1971 (Aphis, 1973). Subsequent cases in Texas were seen (i) along the Gulf of Mexico to Harris County (north and north-easterly direction) and (ii) up the Rio Grande valley to Val Verde County in a north-westerly direction. If one assumes a 4-day incubation period in the horse the horses affected would have been bitten on 19 June and 27 June respectively and the mosquitoes responsible would have bitten an infected horse 4–6 days previously, i.e. earlier than 15 June and 23 June. During mid-June VEE was reported in Mexico 30–50 miles south of Brownsville. The Venezuelan Daily Weather Reports (DWR) for that period show that from 1 June 1971 an area of high pressure lay north and east of New Orleans causing south-east and south winds to blow on the Mexican coast. On 10, 13, 14, 15, 17, 22, 23, 24 and 28 June and 1, 2, 4, 5, 12 and 13 July a high-pressure area lay south of New Orleans located in Gulf of Mexico between the Yucatan peninsula and New Orleans. This means that the winds followed the curve of the Gulf of Mexico, i.e. on the Texas Coast they were from the south-west and west. These winds would have helped the carriage of infected mosquitoes and been responsible for the outbreaks along the Gulf until 18 July. On other days in June and early July the winds were east, south-east and south and these would have been responsible for blowing infected mosquitoes along the Rio Grande valley.

During the epidemic in Mexico there were many instances of disease spreading for long distances. In one instance VEE suddenly appeared in June 1971 in Las Tablas, San Luis Potosi State, 110 km from the coast where disease was occurring (Vilchis, 1972). Disease in horses had preceded that in man. It was suggested that infected people or infected mosquitoes may have been carried to the area on trains, but no cases at intermediate stages occurred. High-pressure areas lay north and east of New Orleans in late May and in early June; easterly winds blowing from the coast would have carried the infected mosquitoes to Las Tablas.

The examples of VEE spread examined so far occurred outside the northerly limits of the Intertropical Convergence Zone.

In January 1969 an epidemic of VEE broke out in the Guayas region of Ecuador (Gutierrez *et al.* 1975) and it was difficult to account for its appearance in that area. In the Venezuelan DWR between December 1968 and January 1969 the ITCZ

lay on most days between 1° N and 7° N off the Pacific coast of South America. On 9 December 1968 the western portion lay on the equator and the central portion on 3° S. Guayas lies about 2° 30′ S. It is suggested that the movement south of the ITCZ was responsible for bringing infected mosquitoes to Guayas province on 9 December 1968. The source was probably Colombia (810 km distance).

In 1969 VEE appeared at the end of May on the Guatemala–El Salvador border (Hinman, McGowan & Henderson, 1971). It is uncertain how it arrived there as there was no record of infected horses or people coming to that area. During May the ITCZ is moving north, but the final northerly limit is not reached until July. Examination of the Venezuelan DWR for 15 May 1969 shows that the northern boundary of the ITCZ lay along the Pacific coast from 16° N, 95° W to 7° N, 78° W and at the southern edge of the mountain chain in Costa Rica and Panama from 10° N, 85° W to 8° N, 80° W. On 16 May a high-pressure area had its centre at 12° N, 86° W and winds blowing clockwise would have carried infected mosquitoes from the Costa Rica–Panama coast at 8° N, 83° W to land at the Guatemala–El Salvador border at 13·5° N, 90° W. This is a distance of 1200–1300 km. It is suggested that the source of infective mosquitoes was in Colombia. The disease would not be noticed in areas lying on the route because the virus of the endemic type is present in Colombia and in Panama.

The outbreaks of epidemic VEE are found on the edge of the southern limit of the ITCZ in Ecuador and Peru and on the edge of the northern limit in Colombia and Venezuela. On one occasion the virus was carried further north by the ITCZ to the Guatemala–El Salvador border and subsequently spread to other countries of Central America and Mexico and southern United States. The virus has not been found in some of the geographical areas covered by the zones described for Western, Eastern and St Louis encephalitis viruses, although climatic conditions are suitable for vectors of the virus. Zones can, however, be delineated as follows:

Zone A is represented by the endemic VEE–the rodent-*Culex* (*melanoconion*) cycle in the forests of Colombia and Central America.

The epidemic cycle is found in zones B and C, the virus being carried there by the movement of migrating mosquitoes, *Aedes taeniorhynchus* and *Aedes sollicitans*. The virus persists during the dry season and reappears the following year, but may eventually die out. These zones are represented by Colombia, Venezuela, Ecuador, Peru and parts of Central America and Mexico.

Zones D and E include those areas where the virus enters, fails to persist or persists 1 year; it eventually disappears and may not be found again for a number of years. These zones are represented by parts of Mexico, the U.S.A. and Trinidad.

The mechanism of change from the forest (endemic) cycle (zone A) to savannah (epidemic–migratory) cycle (zone B) is not known.

The wind systems responsible for the carriage of mosquitoes infected with VEE virus are also probably responsible for the carriage of insects infected with WEE, EEE, SLE and other insect-borne viruses.

(iv) *Vesicular stomatitis*

Although all the vectors have not been identified, it is becoming accepted that one of the main methods of transmission of vesicular stomatitis is by insects. Virus has been identified in North, Central and South America, causing disease in horses, cattle and pigs (see Figs. 1, 3). In Panama *Phlebotomus* has been identified as a vector. In other areas biting flies are thought to be vectors (Table 4).

In the accounts of the epidemics of vesicular stomatitis in the United States and Canada, three major routes of spread are listed: (i) south-eastern states and east of the Mississippi valley: Georgia, Alabama, Florida, N. and S. Carolina, Virginia, Maryland, Louisiana (east), Mississippi (east), Arkansas (east), Missouri, Tennessee, W. Virginia and New Jersey; (ii) east of the Rocky Mountains: Texas (east), Louisiana, Oklahoma, Arkansas, Missouri, Kansas, Colorado (east), Nebraska, Iowa, Minnesota, Wisconsin, N. and S. Dakota and Manitoba; and (iii) western: Texas (west), New Mexico, Arizona, Colorado (west), Utah, Wyoming, Idaho and Montana (see Fig. 1). In 1937 an extensive epidemic was noted and vesicular stomatitis was seen in Wisconsin, Minnesota, E. Dakota and Manitoba (route ii), in Montana (route iii) and in W. Virginia (route i). In 1949 vesicular stomatitis occurred in (i) Alabama, Mississippi, Georgia, Tennessee and Florida, (ii) Minnesota, Wisconsin and Manitoba and (iii) Colorado, Utah, Wyoming and Montana (Hanson, 1952), the disease occurring from June until October.

An account of vesicular stomatitis in the United States 1963–7 was given by Jenney (1968) and the pattern of spread in relation to the three major routes can be seen with outbreaks of the two types of vesicular stomatitis (New Jersey and Indiana) during these years. In 1963 the outbreaks (New Jersey type) were in Alabama, Florida, Georgia and South Carolina (route i). In 1964 New Jersey type was found in the eastern states (route i) but Indiana type occurred in Texas, Oklahoma, Missouri, Arkansas and Colorado (route ii). In 1965 Indiana type was in Arizona, New Mexico, Utah and Colorado (route iii). In 1969 New Jersey type was found in Arkansas, Oklahoma and Texas (route ii) and both New Jersey and Indiana types were in Colorado, New Mexico and Utah (route iii). While some of the outbreaks in different areas have been attributed to movement of horses and cattle, on many occasions there was no record of movement. Hanson (1952), in putting forward his hypothesis of an arthropod vector, noted that much of the spread occurred along corridors on pastures and along valley bottoms and dry river valleys. Hanson (1968) also pointed out that aphids, leafhoppers and lepidoptera migrate on warm southern winds from the Gulf Coast into Canada each spring. Lauerman (1967) commented that some outbreaks of vesicular stomatitis in Georgia were preceded by the passage of anticyclones. The possible movements on route (ii) are paralleled by the long-distance migration routes of the spread of leafhoppers from southern United States through the central United States to Canada (Chiykowski & Chapman, 1965). Route (iii) has a parallel with the spring migration of the beet leafhoppers (Douglass & Cook, 1954). The migratory areas of the aphid green bug have much in common with those of route (i) (Johnson, 1969). This would suggest that a suitable warm wind would carry infected insects

for varying distances over a number of days and, in addition, that the origin of the disease in U.S.A. may be further south in Mexico and Central America. This is supported by the apparent movement westwards of types Indiana from 1964 to 1966 and New Jersey from 1963 to 1966.

If the southern edge of the route is extended southwards, it would reach Mexico and Central America. Together with the evidence on wind carriage of insects infected with virus, this would suggest that the origins of the outbreaks of vesicular stomatitis each year are further south, and that the apparent jumps in the United States are due to lateral movements of the virus in Central America.

The following zones can be derived:

Zone A. This is an endemic cycle in Central America and northern South America represented by a mammal-*phlebotomus* cycle.

Zone B. This would be in Central America and there the virus is maintained in a biting-fly–mammal cycle (horses, ruminants).

Zone C. In this zone the virus may overwinter for one or two seasons and is represented by parts of Georgia and Florida.

Zones D and E. The virus is carried to these areas by biting flies on warm southerly winds and epidemics occur. In zone D the epidemics may occur every 1 or 2 years. In zone E the epidemics are rare. These zones are represented by the United States and Canada. There are insufficient data to determine the zones for South America.

The routes taken by the spread of vesicular stomatitis are similar to those taken by the spread of SLE and, as with VEE further south, the wind systems responsible for the carriage of insects infected with vesicular stomatitis are probably responsible for the carriage of insects infected with WEE, EEE and SLE.

DISCUSSION

Spread of insect-borne viruses over distances results from movement of migratory hosts and vectors from a host–vector cycle in one area to a host–vector cycle in another. The same or different hosts and vectors may be involved in the local cycle and the migratory cycle.

In the past the part played by the migration of blood-sucking insects in the transmission of animal viruses over distances has been underestimated although the spread of plant virus diseases by aphids, leafhoppers and planthoppers and the migration of insect pests is well recorded (Bowden, 1973, 1976). It was thought that as animals, birds and bats could move or be taken long distances and as mosquitoes and midges were supposed to be weak fliers, spread of disease and virus was due to movement of the host or of the vector carried on ship, vehicle or aircraft. The present analysis has produced evidence that virus is being carried on the wind by infected insects in addition to being disseminated by the movement of the host.

With Japanese encephalitis, Murray Valley encephalitis, Western, Eastern and St Louis encephalitis, it can be seen that the spread of virus can be correlated with the movement of mosquitoes on warm winds between and outside the

ITCZ and the availability of hosts such as the nestlings of local or migratory birds or piglets.

Among the group (mosquito–bird or pig cycle) Japanese encephalitis provides the best evidence. Times of appearance of mosquitoes, isolation of virus and development of antibody have been investigated, meteorological data are available and the capture of mosquitoes at sea has been demonstrated. With Murray Valley encephalitis, Eastern and Western equine encephalitis and St Louis encephalitis viruses, less information had been collected, but their behaviour can be compared with that of Japanese encephalitis, and the factors responsible for spread can be correlated with those involved in the spread of insect pests or of other animal and plant viruses. The mosquito–bird viruses so far discussed, are found in the American continent or in Eastern Asia and Australia. In Africa and Western Asia their role may be filled by West Nile virus (Table 2). In 1974 there was an epidemic in South Africa in which West Nile virus was involved (McIntosh *et al.* 1976). The time of outbreak suggests that virus was brought to the area by infected mosquitoes carried on northerly or north-easterly winds, and outbreaks of Rift Valley fever (Barnard & Botha, 1977), bluetongue and African horse sickness at the same time were also possibly introduced by infected insects carried on similar winds.

Zones A, B, C, D, E and F were derived for Japanese encephalitis virus in order to distinguish the different behaviour of virus, host and vector according to climate and environment. Zones A, B and C are equivalent to endemic zones and zones D and E to epidemic zones. The differentiation of zones is easier for Japanese encephalitis than for the others because of the information collected and because the disease extends over an area broken up with islands. Differentiation of zones for Western, Eastern and St Louis encephalitis viruses is more difficult because of movement of disease over a continental mass. It may be possible to analyse the behaviour of West Nile virus by zones but at the moment there is insufficient information on its occurrence in endemic areas in Africa.

With African horse sickness, bluetongue, Ibaraki disease, bovine ephemeral fever, VEE and vesicular stomatitis, spread of disease or virus can be correlated with the movements of the ITCZ, with the carriage of insects on the wind outside the limits of the ITCZ and with the availability and movement of hosts. In some instances good epidemiological data were available since the viruses responsible cause overt signs of disease in valuable domestic animals. Spread of viruses has taken place over areas where winds occur which have been responsible for carriage of insect pests and of insect vectors of plant viruses. Zones such as had been identified for Japanese encephalitis were delineated for these viruses.

Infected midges carried on the wind were probably responsible for long-distance spread of epizootic haemorrhagic disease of deer (Table 3) in North America and for the conveyance of Akabane virus (Table 3) to south-west Japan on winds similar to those responsible for Japanese encephalitis and bovine ephemeral fever virus.

Mosquitoes involved in a savannah cycle of yellow fever may have carried yellow fever virus (Table 4) for varying distances. In investigations of the epi-

demiology of yellow fever in Uganda and Kenya, Haddow *et al.* (1951) and Lumsden & Buxton (1951) suggested that infected mosquitoes carried on the wind for 32 km were responsible for the introduction of yellow fever to islands in Lake Victoria. Outbreaks in Ghana (1969), Togo (1969), Mali and Upper Volta (1969) and Nigeria (1969) occurred in September in regions inland. These regions are some distance away from the coastal area where a jungle mosquito–monkey cycle exists. Savannah mosquitoes infected with virus could have been carried to inland areas in September as a result of the movement of the ITCZ. In May 1940 there were outbreaks of yellow fever in Nuba mountains in Sudan (Kirk, 1941) and in August–September 1959 at Kurmak on the Sudan-Ethiopian border (Berdonneau *et al.* 1961; Satti & Haseeb, 1966), both places being situated at some distance from possible sources of yellow fever at the time (southern Sudan 1939–40, north-east Congo 1958); infected savannah mosquitoes could have carried virus to these areas on southerly winds associated with the ITCZ.

In virus diseases transmitted by ticks and maintained in a tick–mammal cycle the concept of nidality in foci has been put forward. This may also apply to the maintenance of mosquito or midge-host cycles in some areas; but the analyses here suggest that insect-borne virus can be maintained in a continuous cycle of migratory hosts and migratory vectors moving over wide areas in zones A and B where temperatures are suitable all the year. The movement of a cycle from east to west or west to east can be discerned in these zones. Examples include yellow fever in West Africa, Venezuelan equine encephalitis in northern South America as well as St Louis encephalitis and vesicular stomatitis in Central America and African horse sickness in West Africa. From time to time movement of infected insects on the wind takes the virus further north, and the area where it lands depends on the origin on the east–west axis and the direction of the winds conveying the infected insects.

Since the 1930s, it has been known that under suitable temperature and wind conditions a large population of insects of various species and sizes, including Ceratopogonidae and other blood-sucking insects, could be captured at heights up to 1·5 km over land and sea (Hardy & Milne, 1938; Glick, 1939). Movement of many species of insects over long distances has also been recorded (Johnson, 1969). The evidence of the introduction of virus by blood-sucking insects carried on the winds, the timing of the introduction of the virus and its appearance and disappearance in various areas suggests that in the air there is a constant movement to and fro of members of the insect population. Thus new areas are colonized by the insects, old sites reoccupied or local populations of the same species reinforced. The viruses as parasites of the insects, which in turn are parasites of the host, are adapted to such movements.

Flight over land probably takes place during the night, since in the Middle East during 1960, spread of African horse sickness by infected midges did not take place when midday temperatures were less than 30 °C (Sellers *et al.* 1977), i.e. temperatures at night were considerably less. Over the sea, however, flight may take place by day or night. A possible explanation is the need for the insect to avoid desiccation. Thus, over land areas such as the United States or Middle

East, spread of virus by infected insects would not be expected until night temperatures had reached a minimum. Other insects that fly during the morning or afternoon may be seen to move earlier in the season. With survival rates of 0·8% (Walker, 1977) it is more likely that flight is carried out by young adults; a blood meal must be taken before flight and the insect must then survive at least 7 days to ensure multiplication and transmission of the virus.

Reintroduction of virus by migratory insects or vertebrates is not incompatible with overwintering of the virus in the area by other means. It has been suggested that failure to detect virus in the various hosts or vectors or in the eggs was the result of sampling of insufficient numbers or lack of suitable techniques for virus isolation. Techniques have improved and transovarial transmission has been demonstrated for a number of viruses such as California encephalitis (Watts *et al.* 1973), Japanese encephalitis (Rosen *et al.* 1978), yellow fever (Aitken *et al.* 1979) and vesicular stomatitis (Tesh, Chaniotis & Johnson, 1972). However, in zone E, where infection occurs only once every 5, 6 or 7 years, virus is unlikely to have persisted for that period but is more likely to be introduced in infected insects carried to the area by warm winds at a time when suitable conditions for virus, host, vector and climate coincide. If winds are carrying insects to these extreme zones, then it is likely that wind movement of infected insects is occurring in the other zones.

In some areas the epidemiology of a virus can be explained if it is assumed that virus is being maintained both as a result of introduction by infected insects on suitable winds and by persistence during adverse conditions. For example, in eastern and south-eastern Australia it appears as though bovine ephemeral fever survives either through reintroduction by warm winds in summer or by persistence in cattle and buffalo hosts; with Murray Valley encephalitis in the same area, the virus is introduced every few years, but may not persist beyond one or two years after introduction. Similarly, in southern Africa, Rift Valley fever could be brought in, persist or overwinter for one or two seasons in localized areas, but disappear again when insufficient susceptible hosts remain or survival of vector or virus through adverse conditions is not possible. With Western and Eastern encephalitis, the limit of survival during adverse conditions is further north than with St Louis encephalitis, possibly because of the greater variety of hosts. At present little information is available on the minimum number of hosts, the minimum numbers of infected vectors and the minimum length of the overwintering period necessary for a virus to survive during adverse conditions. Measles, for example, disappears from time to time in populations in cities and in islands of about 300000 or less (Bartlett, 1960; Black, 1966) and there may be critical levels of susceptible hosts, numbers of vectors and amounts of virus, above which the virus can survive and below which it dies out. However, in some instances, survival of virus may be enhanced by the adoption of transmission of virus by other means; for example, transplacental passage of virus in bluetongue, infection by the respiratory route in Venezuelan equine encephalitis and Rift Valley fever or infection by contact in vesicular stomatitis.

The movement of infected vectors to and fro with the introduction and reintroduction of virus into various areas makes control difficult. In the face of an

outbreak, knowledge of wind movements of insects and trade movements of animals is important in determining the areas where control would have the greatest effect.

Attempts to eliminate migratory insects or their stages might have a temporary effect on numbers, but unless the environment is changed, the site would be recolonized sooner or later as is happening with *Simulium* in onchocerciasis (river blindness) in West Africa. In the case of man and domestic animals, it would be better to protect the host by vaccination or by spraying, dipping or dusting with repellent insecticides. In zone E vaccination would present a problem. Would it be advisable to vaccinate each year if disease is only coming in one year in five or to vaccinate in the year of an outbreak? When the disease is seen every year, vaccination would be feasible in the hosts at risk, since as the situation is dynamic and boundaries between zones arbitrary, the extent of infection or its persistence in a zone could vary considerably from year to year. However, at any one time, it is important to determine in which zone an area lies, since the system is dynamic, borders between zones are not clear cut and the extent of infection or persistence may vary from year to year.

I am grateful to J. Boorman, E. P. J. Gibbs, P. S. Mellor, W. P. Taylor, J. N. Wilson and other members of the Department of General Virology for their helpful interest and criticisms. I am also grateful to D. E. Pedgley and M. R. Tucker of the Centre of Overseas Pest Research for discussions on the meteorological aspects. I also profited greatly from discussions with J. Bowden, Rothamsted, A. L. Dyce and M. D. Murray of CSIRO, Sydney, and K. C. Sellers lately of Ibadan.

REFERENCES

Aitken, T. H. G., Tesh, R. B., Beaty, B. J. & Rosen, L. (1979). Transovarial transmission of yellow fever virus by mosquitoes (*Aedes aegypti*). *American Journal of Tropical Medicine and Hygiene* **28**, 119–21.

Anderson, S. G. (1954). Murray Valley encephalitis and Australian X disease. *Journal of Hygiene* **52**, 447–68.

Aphis (1973). *The Origin and Spread of Venezuelan Equine Encephalomyelitis*, pp. 25–9. U.S. Department of Agriculture, Hyattsville.

Arakawa, H. & Taga, S. (1969). Climate of Japan. Chapter 2 (pp. 119–58) in *World Survey of Climatology*, vol. 8. Amsterdam: Elsevier.

Arora, R. R. & Singh, N. N. (1974). Epidemiological study of an epidemic of Japanese encephalitis in Bankura district of West Bengal during 1973. *Journal of Communicable Diseases* **6**, 310–19.

Asahina, S. (1970). Transoceanic flight of mosquitoes on the northwest Pacific. *Japanese Journal of Medical Science and Biology* **23**, 255–8.

Barnard, B. J. H. & Botha, M. J. (1977). An inactivated Rift Valley fever vaccine. *Journal of the South African Veterinary Association* **48**, 45–8.

Bartlett, M. S. (1960). The critical community size for measles in the United States. *Journal of the Royal Statistical Society* A **123**, 37–44.

Berdonneau, R., Sérié, C., Panthier, R., Hannoun, C., Papaioannou, S. C. & Georgieff, P. (1961). Sur l'épidémie de fievre jaune de l'année 1959 en Ethiopie. *Bulletin de la Société de Pathologie Exotique* **54**, 276–83.

Best, J. R., Abegunde, A. & Taylor, W. P. (1975). An outbreak of African horse sickness in Nigeria. *Veterinary Record* **97**, 394.

BEVAN, L. E. W. (1907). Preliminary report on the so-called 'stiff sickness' or 'three-day-sickness' of cattle in Rhodesia. *Journal of Comparative Pathology and Therapeutics* **20**, 104–13.

BIGLER, W. J., LASSING, E. B., BUFF, E. E., PRATHER, E. C., BECK, E. C. & HOFF, G. L. (1976). Endemic eastern encephalomyelitis in Florida: a twenty year analysis, 1955–1974. *American Journal of Tropical Medicine and Hygiene* **25**, 884–90.

BLACK, F. L. (1966). Measles endemicity in insular populations. Critical community size and its evolutionary implication. *Journal of Theoretical Biology* **11**, 207–11.

BOUCHER, K. (1975). *Global Climate*. London. English Universities Press.

BOWDEN, J. (1973). Migration of pests in the tropics. *Mededelingen-Fakulteit Landbouwwetenschappen, Gent* **38**, 785–96.

BOWDEN, J. (1976). Weather and phenology of some African Tabanidae. *Journal of the Entomological Society of South Africa* **39**, 207–45.

BOWDEN, J. & GIBBS, D. G. (1973). Light-trap and suction-trap catches of insects in the northern Gezira, Sudan, in the season of southward movement of the Inter-Tropical Front. *Bulletin of Entomological Research* **62**, 577–96.

BROWN, E. S., BETTS, E. & RAINEY, R. C. (1969). Seasonal changes in distribution of the African Armyworm *Spodoptera exempta* (Wlk) (Lep. Noctuidae), with special reference to Eastern Africa. *Bulletin of Entomological Research* **58**, 661–728.

BUESCHER, E. L. & SCHERER, W. F. (1959). Ecologic studies of Japanese encephalitis virus in Japan. IX. Epidemiologic correlations and conclusions. *American Journal of Tropical Medicine and Hygiene* **8**, 719–22.

CHIYKOWSKI, L. N. & CHAPMAN, R. K. (1965). Migration of the six-spotted leafhopper *Macrosteles fascifrons* (Stol). 2. Migration of the six-spotted leafhopper in central North America. *Research Bulletin of the Agricultural Experimental Station. University of Wisconsin*, no. 261, pp. 21–45.

CREECH, W. B. (1977). From the Center for Disease Control. St Louis encephalitis in the United States, 1975. *Journal of Infectious Diseases* **135**, 1014–6.

DALRYMPLE, J. M., YOUNG, O. P., ELDRIDGE, B. F. & RUSSELL, P. K. (1972). Ecology of arboviruses in a Maryland freshwater swamp. III. Vertebrate hosts. *American Journal of Epidemiology* **96**, 129–40.

DAVIES, F. G. & WALKER, A. R. (1974). The isolation of ephemeral fever virus from cattle and *Culicoides* midges in Kenya. *Veterinary Record* **95**, 63–4.

DIPEOLU, O. O. (1977). Potential vectors of bluetongue in Nigeria. *Bulletin of Animal Health and Production in Africa* **25**, 17–23.

DOHERTY, R. L. (1974). Arthropod-borne viruses in Australia and their relation to infection and disease. *Progress in Medical Virology* **17**, 136–92.

DOUGLASS, J. R. & COOK, W. C. (1954). The beet leafhopper. *Circular United States Department of Agriculture*, no. 942, 21 pp.

DUVIARD, D. (1977). Migrations of Dysdercus spp. (*Hemiptera: Pyrrhocoridae*) related to movements of the Inter-Tropical Convergence Zone in West Africa. *Bulletin of Entomological Research* **67**, 185–204.

EISA, M. (1974). The isolation and identification of type 9 African horse sickness virus in the Sudan. *British Veterinary Journal* **130**, 606–9.

FRENCH, E. L. (1973). A review of arthropod-borne virus infections affecting man and animals in Australia. *Australian Journal of Experimental Biology and Medical Science* **51**, 131–58.

FUKUMI, H., HAYASHI, K., MIFUNE, K., SHICHIJO, A., MATSUO, S., OMORI, N., WADA, Y., ODA, T., MOGI, M. & MORI, A. (1975). Ecology of Japanese encephalitis in Japan. I. Mosquito and pig infection with the virus in relation to human incidences. *Tropical Medicine* **17**, 97–110.

GAYRAL, P. H. & CAVIER, R. (1971). Données entomologiques et écologiques actuelles sur les vecteurs de la fièvre jaune en Afrique de l'Ouest. *Bulletin de la Société de Pathologie Exotique* **64**, 701–8.

GILYARD, R. T. (1945). A clinical study of Venezuelan virus equine encephalomyelitis in Trinidad, B.W.I. *Journal of the American Veterinary Medical Association* **106**, 267–77.

GLICK, P. A. (1939). The distribution of insects, spiders and mites in the air. *Technical Bulletin U.S. Department of Agriculture*, no. 673.

GUTIERREZ, E., MONATH, T. P., ALAVA, A., URIGUEN, D., ARZUBE, M. & CHAMBERLAIN, R. W. (1975). Epidemiologic investigations of the 1969 epidemic of Venezuelan encephalitis in Ecuador. *American Journal of Epidemiology* **102**, 400–413.

HADDOW, A. J., DICK, G. W. A., LUMSDEN, W. H. R. & SMITHBURN, K. C. (1951). Monkeys in relation to the epidemiology of yellow fever in Uganda. *Transactions of the Royal Society of Tropical Medicine and Hygiene* **45**, 189–224.

HAGGIS, M. J. (1971). Light trap captures of *Spodoptera exempta* (Walk.) in relation to wind direction. *East African Agricultural and Forestry Journal* **37**, 100–108.

HANSON, R. P. (1952). The natural history of vesicular stomatitis. *Bacteriological Reviews* **16**, 179–204.

HANSON, R. P. (1968). Discussion of the natural history of vesicular stomatitis. *American Journal of Epidemiology* **87**, 264–5.

HANSON, R. P. (1972). Virology and epidemiology of eastern and western arboviral encephalomyelitis of horses. *Proc. 3rd International Conference of Equine Infectious Diseases, Paris.* pp. 110–14. (Basel: Karger, 1973.)

HARDY, A. C. & MILNE, P. S. (1938). Aerial drift of insects. *Nature, London* **141**, 602.

HAYASHI, K., MIFUNE, K. & SHICHIJO, A. (1965). Problems of overwintering of Japanese encephalitis virus in Japan. *Endemic Diseases Bulletin, Nagasaki University* **7**, 99–106.

HAYASHI, K., MIFUNE, K., MATSUO, S., SHICHIJO, A., SUZUKI, H., URA, M., MAKINO, Y., WADA, Y., ODA, T., MOGI, M. & MORI, A. (1978*a*). Ecology of Japanese encephalitis virus in Japan, particularly the results of surveys in every interepidemic season from 1964 to 1976. *Tropical Medicine* **20**, 81–96.

HAYASHI, K., SUZUKI, H. & ASAHINA, S. (1978*b*). Notes on the transoceanic insects captured on East China sea in 1977. *Tropical Medicine* **20**, 131–42.

HAYES, C. G. & WALLIS, R. C. (1977). Ecology of western equine encephalomyelitis viruses in the eastern United States. *Advances in Virus Research* **21**, 37–83.

HAYES, R. O., FRANCY, D. B., LAZUICK, J. S., SMITH, G. C. & JONES, R. H. (1976). Arbovirus surveillance in six states during 1972. *American Journal of Tropical Medicine and Hygiene* **25**, 463–76.

HESS, A. D. & HAYES, R. O. (1967). Seasonal dynamics of western encephalitis virus. *American Journal of Medical Sciences* **253**, 333–48.

HINMAN, A. R., MCGOWAN, J. E. JR. & HENDERSON, B. E. (1971). Venezuelan equine encephalomyelitis: surveys of human illness during an epizootic in Guatemala and El Salvador. *American Journal of Epidemiology* **93**, 130–36.

HOLDEN, P., HAYES, R. O., MITCHELL, C. J., FRANCY, D. B., LAZUICK, J. S. & HUGHES, T. B. (1973). House sparrows, *Passer domesticus* (L.), as hosts of arboviruses in Hale County, Texas. I. Field studies 1965–1969. *American Journal of Tropical Medicine and Hygiene* **22**, 244–53.

INABA, Y. (1975). Ibaraki disease and its relationship to bluetongue. *Australian Veterinary Journal* **51**, 178–85.

INABA, Y., TANAKA, Y., SATO, K., ITO, H., OMORI, T. & MATUMOTO, M. (1968). Bovine epizootic fever. I. Propagation of the virus in suckling hamster, mouse and rat, and hamster kidney BHK21-W12 cells. *Japanese Journal of Microbiology* **12**, 457–69.

ISHIDA, N., YAMAMOTO, H., ENDO, K., KONNO, J. & KAROJI, Y. (1976). Epidemiology of Japanese encephalitis: factor analysis of obvious decline of Japanese encephalitis infection in recent years. *Japanese Journal of Bacteriology* **31**, 375–86.

JENNEY, E. W. (1968). Vesicular stomatitis in the United States during the last five years (1963–1967). *Proceedings of the United States Livestock Sanitary Association* **71**, 371–85.

JOHNSON, C. G. (1969). *Migration and Dispersal of Insects by Flight.* London: Methuen.

JOHNSON, K. M. & MARTIN, D. H. (1974). Venezuelan equine encephalitis. *Advances in Veterinary Science and Comparative Medicine* **18**, 79–116.

KANAMITSU, M., TANIGUCHI, K., URUSAWA, S., OGATA, T., WADA, Y., WADA, Y. & SAROSO, J. S. (1979). Geographic distribution of arbovirus antibodies in indigenous human populations in the Indo-Australian Archipelago. *American Journal of Tropical Medicine and Hygiene* **28**, 351–63.

KIRK, R. (1941). An epidemic of yellow fever in the Nuba Mountains, Anglo-Egyptian Sudan. *Annals of Tropical Medicine and Parasitology* **33**, 225–31.

KISIMOTO, R. (1971). Long distance migration of plant hoppers *Sogatella furcifera* and *Nilaparvata lugens*. *Proceedings of Symposium on Rice Insects, Toyko* 1971, 201–16.

KONO, R. & KIM, K. H. (1969). Comparative epidemiological features of Japanese encephalitis in the Republic of Korea, China (Taiwan) and Japan. *Bulletin of the World Health Organization* **40**, 263–77.

LAUERMAN, L. (1967). Vesicular stomatitis in temperate and tropical America. Ph.D. thesis, University of Wisconsin, Madison.

LEVY, J. S., CARVER, H. D., MOSELEY, I. K., CALISHER, C. H., FRANCY, D. B. & MONATH, T. P. (1978). St Louis encephalitis in Memphis – Shelby County, Tennessee, 1975: epidemiologic aspects of human cases. *Southern Medical Journal* **71**, 633–7.

LI, KUANG-PO, WONG, HONG-HSIANG, & WOO, WAN SEI. (1964). Route of the seasonal migration of the Oriental Armyworm Moth in the eastern part of China as indicated by a three-year result of releasing and recapturing marked moths. *Acta phytophylacica Sinica* **3**, 101–10.

LILLIE, L. E., WONG, F. C. & DRYSDALE, R. A. (1976). Equine epizootic of western encephalomyelitis in Manitoba – 1975. *Canadian Journal of Public Health* **67**, Suppl. 1, 21–7.

LORD, R. D. & CALISHER, C. H. (1970). Further evidence of southward transport of arboviruses by migratory birds. *American Journal of Epidemiology* **92**, 73–8.

LUBY, J. P., SULKIN, S. E. & SANFORD, J. P. (1969). The epidemiology of St Louis encephalitis: a review. *Annual Review of Medicine* **20**, 329–50.

LUMSDEN, W. H. R. & BUXTON, A. P. (1951). A study of the epidemiology of yellow fever in West Nile district, Uganda. *Transactions of the Royal Society of Tropical Medicine and Hygiene* **45**, 53–78.

MCGOWAN, J. E. JR., BRYAN, J. A. & GREGG, M. B. (1973). Surveillance of arboviral encephalitis in the United States, 1955–1971. *American Journal of Epidemiology* **97**, 199–207.

MCINTOSH, B. M., JUPP, P. G., DOS SANTOS, I. & MEENEHAN, G. M. (1976). Epidemics of West Nile and Sindbis viruses in South Africa with *Culex* (*culex*) *univittatus* Theobald as vector. *South African Journal of Science* **72**, 295–300.

MAEDA, O. J., KARAKI, T., KURODA, A., KAROGI, Y., SASAKI, O. & TAKENOKUMA, K. (1978). Epidemiological studies on Japanese encephalitis in Kyoto City area, Japan. II. Annual patterns of virus dissemination on virus recoveries from unfed *Culex tritaeniorhynchus summorosus*. *Japanese Journal of Medical Science and Biology* **31**, 39–51.

MURRAY, M. D. (1970). The spread of ephemeral fever of cattle during the 1967–68 epizootic in Australia. *Australian Veterinary Journal* **46**, 77–82.

MUUL, I., JOHNSON, B. K. & HARRISON, B. A. (1975). Ecological studies of *Culiseta melanura* (Diptera: Culicidae) in relation to eastern and western equine encephalomyelitis viruses on the eastern shore of Maryland. *Journal of Medical Entomology* **11**, 739–48.

NEVILL, E. M. (1971). Cattle and *Culicoides* biting midges as possible overwintering hosts of bluetongue virus. *Onderstepoort Journal of Veterinary Research* **38**, 65–72.

NEWTON, L. G. & WHEATLEY, C. H. (1970). The occurrence and spread of ephemeral fever of cattle in Queensland. *Australian Veterinary Journal* **46**, 561–8.

OMORI, T. (1961). Bluetongue-like disease in Japan. *Bulletin de l'Office International des Épizooties* **55**, 1109–17.

OMORI, T., INABA, Y., MORIMOTO, T., TANAKA, Y., ISHITANI, R., KUROGI, H., MUNUKATA, K., MATSUDA, K. & MATUMOTO, M. (1969). Ibaraki virus, an agent of epizootic disease of cattle resembling bluetongue. I. Epidemiologic, clinical and pathologic observations and experimental transmission to calves. *Japanese Journal of Microbiology* **13**, 139–57.

Oxford World Atlas (1973). London: Oxford University Press.

RAINEY, R. C. (1951). Weather and the movements of locust swarms: a new hypothesis. *Nature, London* **168**, 1057–60.

RAINEY, R. C. (1973). Airborne pests and the atmospheric environment. *Weather, London* **28**, 224–39.

RAINEY, R. C. (1974). Biometeorology and insect flight: some aspects of energy exchange. *Annual Review of Entomology* **19**, 407–39.

RATCLIFFE, F. M., MYERS, K., FENNESSY, B. V. & CALABY, J. H. (1952). Myxomatosis in Australia. A step towards biological control of the rabbit. *Nature, London* **170**, 7–11.

REEVES, W. C. (1974). Overwintering of arboviruses. *Progress in Medical Virology* **17**, 193–220.

ROSEN, L., TESH, R. B., LIEN, J. C. & CROSS, J. H. (1978). Transovarial transmission of Japanese encephalitis by mosquitoes. *Science* **199**, 909–11.

ST GEORGE, T. D., STANDFAST, H. A., CHRISTIE, D. G., KNOTT, S. G. & MORGAN, I. R. (1977). The epizootiology of bovine ephemeral fever in Australia and Papua-New Guinea *Australian Veterinary Journal* **53**, 17–28.

SATTI, M. H. & HASEEB, M. A. (1966). An outbreak of yellow fever in the southern Fung and Upper Nile Province, Republic of Sudan. *Journal of Tropical Medicine and Hygiene* **69**, 34–44.

SAUGSTAD, E. S., DALRYMPLE, J. M. & ELDRIDGE, B. F. (1972). Ecology of arboviruses in a Maryland freshwater swamp. I. Population dynamics and habitat distribution of potential mosquito vectors. *American Journal of Epidemiology* **96**, 114–22.

SEDDON, H. R. (1938). The spread of ephemeral fever (three-day sickness) in Australia in 1936–37. *Australian Veterinary Journal* **14**, 90–101.

SELLERS, R. F. (1975). Bluetongue in Cyprus. *Australian Veterinary Journal* **51**, 198–203.

SELLERS, R. F., PEDGLEY, D. E. & TUCKER, M. R. (1977). Possible spread of African horse sickness on the wind. *Journal of Hygiene* **79**, 279–98.

SELLERS, R. F., PEDGLEY, D. E. & TUCKER, M. R. (1978). Possible windborne spread of bluetongue to Portugal, June–July 1956. *Journal of Hygiene* **81**, 189–96.

SELLERS, R. F., GIBBS, E. P. J., HERNIMAN, K. A. J., PEDGLEY, D. E. & TUCKER, M. R. (1979). Possible origin of the bluetongue epidemic in Cyprus, August 1977. *Journal of Hygiene* **83**, 547–55.

SIMPSON, D. I. H., BOWEN, E. T. W., WAY, H. J., PLATT, G. S., HILL, M. N., KAMATH, S., WAH, L. T., BENDELL, P. J. E. & HEATHCOTE, O. H. U. (1974). Arbovirus infection in Sarawak, October 1968 – February 1970: Japanese encephalitis virus isolations from mosquitoes. *Annals of Tropical Medicine and Parasitology* **68**, 393–404.

SIMPSON, D. I. H., SMITH, C. E. G., MARSHALL, T. F. de C., PLATT, G. S., WAY, H. J., BOWEN, E. T. W., BRIGHT, W. F., DAY, J., MCMAHON, D. A., HILL, M. N., BENDELL, P. J. E. & HEATHCOTE, O. H. U. (1976). Arbovirus infections in Sarawak: the role of the domestic pig. *Transactions of the Royal Society of Tropical Medicine and Hygiene* **70**, 66–72.

SPENCE, L., ARTSOB, H., GRANT, L. & TH'NG, C. (1977). St Louis encephalitis in southern Ontario: laboratory studies for arboviruses. *Canadian Medical Association Journal* **116**, 35–7.

TAKAHASHI, K., MATSUO, R., KUMA, M. & NOGUCHI, H. (1965). Studies on mosquito infection with Japanese encephalitis virus in 1964 in Nagasaki prefecture. *Endemic Diseases Bulletin, Nagasaki University* **7**, 165–77.

TAYLOR, W. P. & MCCAUSLAND, A. (1976). Studies with bluetongue virus in Nigeria. *Tropical Animal Health and Production* **8**, 169–73.

TESH, R. B., CHANIOTOIS, B. N. & JOHNSON, K. M. (1972). Vesicular stomatitis virus (Indiana serotype); transovarial transmission by phlebotomine sandflies. *Science* **175**, 1477–9.

THEILER, A. (1921). African horse sickness (Pestis equorum). *South Africa, Department of Agriculture Science Bulletin*, no. 19.

URA, M. (1976). Ecology of Japanese encephalitis virus in Okinawa, Japan. I. The investigation on pig and mosquito infection of the virus in Okinawa island from 1966 to 1976. *Tropical Medicine* **18**, 151–63.

VAN PEENEN, P. F. D., JOSEPH, P. L., ATMOSOEDJONO, S., IRSIANA, R. & SAROSO, J. S. (1975*a*). Japanese encephalitis virus from pigs and mosquitoes in Jakarta, Indonesia. *Transactions of the Royal Society of Tropical Medicine and Hygiene* **69**, 477–9.

VAN PEENEN, P. F. D., JOSEPH, P. L., ATMOSOEDJONO, S., IRSIANA, R. & SAROSO, J. S. (1975*b*). Isolation of Japanese encephalitis virus from mosquitoes near Bogor, West Java, Indonesia. *Journal of Medical Entomology* **12**, 573–4.

VILCHIS, J. (1972). Human disease: Mexico. In *Proceedings of the Workshop Symposium on Venzuelan Encephalitis Virus, Pan American Health Organisation, Washington*, pp. 215–217.

WADA, Y., KAWAI, S., ODA, T., MIYAGI, I., SVENAGA, O., NISHIGAKI, J., OMORI, N., TAKAHASHI, K., MATSUO, T., ITOH, T. & TAKATSUKI, Y. (1969). Dispersal experiment of *Culex tritaeniorhynchus* in Nagasaki area (preliminary report) *Tropical Medicine* **11**, 37–44.

WADA, Y., ODA, T., MOGI, M., MORI, A., OMORI, N., FUKUMI, H., HAYASHI, K., MIFUNE, K., SHICHIJO, A. & MATSUO, S. (1975). Ecology of Japanese encephalitis in Japan. II. The population of vector mosquitoes and the epidemic of Japanese encephalitis. *Tropical Medicine* **17**, 111–27.

WALKER, A. R. (1977). Adult lifespan and reproductive status of *Culicoides* species (Diptera, Ceratopogonidae) in Kenya, with reference to virus transmission. *Bulletin of Entomological Research* **67**, 205–15.

WATERS, J. R. (1976). An epidemic of Western encephalomyelitis in humans – Manitoba, 1975. *Canadian Journal of Public Health* **67**, Suppl. 1, 28–32.

WATTS, D. M., PANTUWATANA, S., DE FOLIART, G. R., YUILL, T. M. & THOMPSON, W. H. (1973). Transovarial transmission of La Crosse virus (California encephalitis group) in the mosquito, *Aedes triseriatus*. *Science* **182**, 1140–41.

WELLINGS, F. M., LEWIS, A. L. & PIERCE, L. V. (1972). Agents encountered during arboviral ecological studies: Tampa Bay area, Florida, 1963 to 1970. *American Journal of Tropical Medicine and Hygiene* **21**, 201–13.

YAMADA, K., HASHIMOTO, N. & KANAMITSU, M. (1972). Epidemiological study of Japanese encephalitis in Hokkaido. 4. Serological and climatological investigations of Japanese encephalitis virus infection in domestic animals. *Virus* **22**, 38–47.