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Ps Mellor

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Review article

African horse sickness: transmission and epidemiology

PS Mellor

Institute for Animal Health, Pirbright Laboratory, Ash Road, Pirbright, Woking, Surrey, UK

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Summary — African horse sickness (AHS) virus causes a non-contagious, infectious, arthropod-borne disease of equines and occasionally of dogs. The virus is widely distributed across sub-Saharan Africa where it is transmitted between susceptible vertebrate hosts by the vectors. These are usually considered to be species of *Culicoides* biting midges but mosquitoes and/or ticks may also be involved to a greater or lesser extent. Periodically the virus makes excursions beyond its sub-Saharan enzootic zones but until recently does not appear to have been able to maintain itself outside these areas for more than 2–3 consecutive years at most. This is probably due to a number of factors including the apparent absence of a long term vertebrate reservoir, the prevalence and seasonal incidence of the vectors and the efficiency of control measures (vaccination and vector abatement). The recent AHS epizootics in Iberia and N Africa spanning as they do, 5 or more yr, seem to have established a new pattern in AHS virus persistence. This is probably linked to the continuous presence of adult *C imicola* in the area. *Culicoides imicola* is basically an Afro-Asiatic insect and prefers warm climates. Therefore its continuous adult presence in parts of Iberia and N Africa may be due to some recent moderations of the climate in these areas.

African horse sickness / transmission / epidemiology

Résumé — **La peste équine : transmission et épidémiologie.** *Le virus de la peste équine provoque chez les équins et occasionnellement chez le chien, une maladie infectieuse non contagieuse, transmise par des arthropodes. Ce virus est largement réparti au Sud du Sahara où il est transmis aux hôtes vertébrés sensibles par des vecteurs. On a l'habitude de considérer que ceux-ci sont des moucheron piqueurs (Culicoides) mais des moustiques et/ou des tiques peuvent être également impliqués dans une plus ou moins grande mesure. Périodiquement, le virus fait des apparitions au-delà des zones enzootiques sub-sahariennes mais pendant longtemps il n'a pas réussi à se maintenir en dehors de ces zones pendant plus de 2–3 ans consécutifs. Ce fait est probablement dû à de nombreux facteurs mais en particulier à l'absence apparente d'un réservoir de vertébrés de longue durée chez les vertébrés, à la prévalence et à l'incidence saisonnière des vecteurs et à l'efficacité des mesures de contrôles (vaccination et réduction des vecteurs). Les épizooties récentes de peste équine dans la péninsule ibérique et en Afrique du Nord, qui se sont étendues sur 5 ans ou plus, semblent avoir établi un nouveau modèle de persistance du virus. Ceci est probablement dû à la présence permanente d'adultes de *C imicola* dans cette zone. *C imicola* est un insecte localisé essentiellement en Afrique et en Asie et qui préfère les climats chauds. La présence permanente d'adultes dans certaines parties de la péninsule ibérique et en Afrique du Nord pourrait s'expliquer par une modification récente du climat dans ces régions.*

peste équine / transmission / épidémiologie

INTRODUCTION

African horse sickness (AHS) virus is a double stranded RNA virus which causes a non-contagious, infectious, arthropod-borne disease of equines. The disease is characterised by clinical signs which develop as a result of the impaired function of the circulatory and respiratory systems giving rise to serous effusions and haemorrhage in various organs and tissues (Howell, 1963). The extent and severity of the clinical signs caused by AHS virus are frequently used to classify the disease into 4 distinct forms. In increasing order of severity these are horse sickness fever, the subacute or cardiac form, the cardiopulmonary or mixed form and the peracute or pulmonary form. Comprehensive accounts of the clinical signs, pathogenesis and pathology caused in equines by AHS virus have been published by Rafyi (1961), Howell (1963), Erasmus (1973), Mirchamisy and Hazrati (1973) and Lubroth (1988).

AHS virus exists as a number of distinct serotypes and to date 9 have been internationally recognised. Animals recovering from an infection with any one of these serotypes develop a solid immunity to it but may continue to be susceptible to heterologous serotypes (Anonymous, 1978).

Since under natural conditions AHS virus is transmitted between its vertebrate hosts virtually exclusively by the bites of various species of haematophagous arthropods its distribution is limited to those geographical areas where competent vectors are present and to those times of the year when conditions are favourable for vector activity.

HISTORY

Probably the first historical reference to AHS concerns an epizootic in the Yemen

which occurred in 1327 (Moulé, 1896). However despite this early record the virus group appears to have originated in Africa and was first recognised as a distinct disease entity there subsequent to the introduction of highly susceptible breeds of equine during the exploration of Central Africa. The earliest account comes from East Central Africa in 1569 (Theal, 1899). In southern Africa the disease has been recognised since the occupation of the Cape of Good Hope by the Dutch East India Company at the beginning of the 18th century when large numbers of deaths occurred in imported horses (Henning, 1956).

However, it was not until 1900 that M'Fadyean using samples of infected horse blood showed that the agent of horse sickness fever was able to pass through bacterial filters and concluded that it was an "ultravisible" virus. Theiler in a series of experiments covering several years (1908, 1915 and 1921) then recognised that AHS virus existed as a number of antigenically distinct strains but not until 1962 were the 2 most recent internationally accepted serotypes (8 and 9) identified and characterised (Howell, 1962). The isolation of an additional strain of AHS virus in Kenya (G-75), that apparently is not neutralised by antisera to any of the 9 recognised serotypes, suggests that a further serotype should now be added to the internationally recognised list (Davies and Lund, 1974; Davies, 1976).

GEOGRAPHICAL DISTRIBUTION AND OCCURRENCE

AHS virus is widely distributed across sub-Saharan Africa and is enzootic in a band stretching from Senegal and Gambia in the west to Ethiopia and Somalia in the east (Howell, 1963). It also occurs as far south as S Africa and may extend at times to

Egypt in the north. The Sahara desert, however, provides an effective geographical barrier which usually, though not invariably, prevents incursions into North and NW Africa from the infected areas further south.

Until relatively recently AHS virus was believed to be confined to Africa with the exception of occasional excursions across the Red Sea into SW Arabia (Rafyi, 1961; Mirchamsy and Hazrati, 1973). However, in the summer of 1959 the situation changed. Horse sickness fever appeared first in Saudi Arabia and the southern regions of Iran, and then spread northwards, eastwards and westwards to involve Afghanistan and Pakistan by the autumn of 1959. During the spring and summer of the following year the disease continued to spread, particularly along the courses of the great rivers which formed the major trade routes, its progress being facilitated by the movements of nomadic tribesmen and their animals (Howell, 1960). Syria, Lebanon, Jordan, Iraq, Turkey, Cyprus and extensive tracts of India were rapidly involved all within the next 6 months (Howell, 1960, 1965; Rafyi, 1961; Gorhe *et al*, 1965; Mirchamsy and Hazrati, 1973). However, by the end of 1961 in the face of a massive vaccination campaign and the deaths of over 300 000 equines the disease in Asia apparently came to a halt (Anwar and Qureshi, 1972). This was probably due to a combination of factors including adverse climatic conditions, vector abatement campaigns, vaccination and the virtual depopulation of susceptible equines over much of the area. The virus responsible for the epizootic was identified retrospectively, from isolations made in Pakistan, India and several Middle Eastern countries, as AHS virus serotype 9 (Howell, 1962; Shah, 1964; Rabah, 1966; Sers, 1967; Mornet *et al*, 1967).

In 1965 AHS again spread beyond its traditional enzootic zones in sub-Saharan

Africa and appeared in southern Morocco, rapidly extending into Algeria and Tunisia and eventually crossing the Straits of Gibraltar into southern Spain in October 1966 (Rabah, 1966; Diaz Montilla and Panos Marti, 1967, 1968; Mornet *et al*, 1967; Sers, 1967; Laaberki, 1969). The epizootic once more was caused by AHS virus serotype 9 (Diaz Montilla and Panos Marti, 1968). Pilo-Moron *et al* (1969) regarded the appearance of AHS in N Africa as being due to the movement of nomads and their animals, particularly donkeys across the Sahara from Central Africa where AHS virus serotype 9 was apparently enzootic (Maurice and Provost, 1967). The 1965–1966 epizootic succeeded in “overwintering” once in N Africa but the northern extension into southern Spain was eliminated within 3 wk apparently through a vigorous vaccination and slaughter policy (Diaz Montilla and Panos Marti, 1968).

Subsequent to 1966 AHS virus apparently remained quiescent in sub-Saharan Africa for over 20 yrs. However, in 1987 an outbreak of AHS due to serotype 4 was confirmed in the provinces of Madrid, Toledo and Avila in Central Spain (Lubroth, 1988). The origin of the outbreak is believed to have been the importation of 10 zebra from Namibia, 5 of which were taken to a Safari Park (El Rincon) 50 km SW of Madrid. This safari park subsequently became the site of the first 27 cases of AHS in Spain the first signs being detected on July 22nd and 23rd some 26 d after the arrival of the zebra (Lubroth, 1988; Mellor *et al*, 1990b). The epizootic continued for 3–4 months in central Spain, the last officially recorded death being in mid-October 1987 by which time 146 equines had died and over 38 000 had been vaccinated (Anonymous, 1987; Diaz Yubero, 1987). Unfortunately in October 1988 almost a year after the last reported death, a recrudescence of AHS due to serotype 4 was confirmed in the south of Spain at Sotogrande in Cadiz.

Later the same month the disease spread into the neighbouring Province of Malaga. Official records indicate that 156 equines died either directly or indirectly due to AHS, the last reported death being in early December (Rodriguez *et al*, 1989). Then at the end of July in 1989 AHS once more broke out at Sotogrande, the disease being confirmed by laboratory findings in early August (Anonymous, 1989a). In rapid succession cases also occurred in the provinces of Huelva, Cordoba and Seville in Andalucia, and Badajoz in Extramadura. This time the disease did not stop at international boundaries and during September spread into the southern 2 Portuguese provinces of Baixo Alentejo and the Al-gave, and then in October cases also occurred in Tetouan, Tanger and Larache in Morocco (Anonymous, 1989b, 1990a). It has been estimated that altogether some 2 000 equines died in these 3 countries during 1989 (Mellor, 1991). In September 1990 for the 4th successive year AHS due to serotype 4 was confirmed in Spain (Malaga) and caused the deaths of a further 66 equines, the last positive case being diagnosed in November (Anonymous, 1990b). In Morocco in 1990 AHS was detected in 6 Provinces with the loss of some 555 equines, however the virus did not apparently recrudescence in Portugal (Anonymous, 1992). In 1991 Portugal remained free from the disease and Spain also reported no cases but the virus continued its progression through Morocco involving some 20 provinces and extending for the first time south of the Atlas mountains (Anonymous, 1992). At the time of writing in early 1992 it remains to be seen whether further recrudescences of AHS will occur either in Morocco or in neighbouring Maghreb countries though in view of the widespread occurrence of the disease during 1991 this would seem to be not unlikely.

Separate from the incursion of AHS virus serotype 4 into the Mediterranean

theatre it is worthwhile recording that for the first time since 1959 AHS has also been reported in Saudi Arabia, where AHS virus serotype 9 was isolated from clinical cases in the SW, Abha Region in 1989 (Mellor *et al*, 1990a). Further information concerning the extent of this outbreak is apparently not available.

TRANSMISSION

AHS virus had long been thought to be transmitted by biting arthropods and suspicion has at one time or another fallen on a wide variety of species and genera. As early as 1903, Pitchford-Watkins and Theiler suggested that *Anopheles* mosquitoes could be involved. In 1912 Schuberg and Kuhn showed that *Stomoxys calcitrans* is capable of transmitting the virus mechanically (*ie* without replication of the virus in the vector). However, the importance of this method of transmission was later discounted because *S calcitrans* is a daytime feeder and it was found that confining horses in mosquito proof stables at night protected them against the disease (Theiler, 1915). Williams (1913) in the absence of other haematophagous insects during outbreak of AHS in the Sudan considered *Lyperosia minuta* to be a likely vector. Van Saceghem (1918) suspected ticks and Tabanids, and Carpano (1931) suggested *Anopheles*, *Aedes*, *Phlebotomus* and *Simulium* as possible carriers. However, Nieschulz *et al* (1934) and Nieschulz and Du Toit (1937) after a careful study of the mosquito fauna at Onderstepoort (S Africa) concluded that mosquitos are not vectors of horse sickness. These workers did record the survival of AHS virus in some mosquitoes for up to 27 d but despite numerous attempts they were never able to transmit the virus by mosquito bite. Then in 1944 came something of a breakthrough when Du Toit showed that

wild caught *Culicoides* species were infected with AHS virus and later in 1945 (quoted in Wetzell *et al*, 1970) he succeeded in transmitting the virus by *Culicoides* bite from an infected to a susceptible horse 12 d after the insects infecting blood meal. This was the first definitive demonstration of the biological transmission of AHS virus by any species of arthropod. Since that time AHS virus has been regarded by most workers primarily as a *Culicoides*-borne virus. However, the situation is far from being clear-cut and in more recent times a variety of reports have been published dealing with mosquitoes and ticks, in addition to *Culicoides*, as potential or actual vectors of AHS virus.

Mosquitoes

Ozawa and Nakata (1965) and Ozawa *et al* (1966a, b, 1970) recorded the successful transmission of AHS virus to horses *via* the bites of artificially infected *Anopheles stephensi*, *Culex pipiens* and *Aedes aegypti*. These authors found that virus replicated only in a limited number of mosquitoes allowed to engorge upon viraemic blood but transmission was demonstrated when refeeding these individuals at periods of between 15 and 22 d post infection (dpi). The maximum titres of virus recovered from infected mosquitoes were never significantly higher than the amounts ingested but an eclipse phase apparently occurred just subsequent to infection and virus was recovered for up to 35 dpi. Hussein (1982) and Abdallah (1983) also considered the mosquito, *C pipiens* to be a biological vector of AHS virus, and following on from their work El-Husseini *et al* (1986) reported that *C pipiens* was also able to induce "showering" of AHS virus from the spleens of latently infected dogs. Subsequent batches of mosquitoes feeding upon the same dogs thereby became

infected with the 'released virus' which they were subsequently able to transmit to susceptible hosts. 'Showering' is a phenomenon described by Luedke *et al* (1977) and Jones *et al* (1981) whereby the bites of a vector species of *Culicoides* (*C variipennis*) were purported to stimulate release of bluetongue (BT) virus from an unknown, extra-vascular depot into the blood stream of a bull 'latently' infected with the virus. Presumably it is envisaged that such a mechanism would be triggered by factor(s) in the saliva of the vectors, possibly in association with a degree of host stress. However, considerable doubt has recently been cast upon the validity of this earlier work and the whole BT virus showering phenomenon (Osburn, 1988; Walton, 1991). In the light of this it might be prudent to critically re-evaluate the apparent existence of a similar mechanism in dogs "latently"infected with AHS virus.

Ticks

Salama *et al* (1979, 1980) isolated AHS virus serotype 9 from 17% of 2 089 field samples of *Hyalomma dromadarii* collected in Egypt. Unfortunately it is not clear from their work whether the viruses isolated originated from the tissues of the ticks themselves, or whether they were present merely in the gut lumen, as a result of a recent viraemic blood meal. Nevertheless the same authors also reported the presence of AHS viruses in newly emerged (*ie* unfed) adult ticks and they succeeded in transmitting the virus *via* tick bite to camels and to horses. Infected ticks were reported to retain virus for up to 10 wk post infection and trans-stadial but not transovarial transmission was recorded (Dardiri and Brown, 1989).

Awad *et al* (1981) also succeeded in transmitting AHS virus, serotype 9, from infected to susceptible horses *via* the bites

of adult *H dromadarii*. Clean ticks then became persistently infected by feeding upon the viraemic horse. Trans-stadial transmission from larvae to nymphs and from nymphs to adults was demonstrated but once again transovarial transmission was not recorded.

Further studies in Egypt, by Salama *et al* (reported in Dardiri and Salama, 1988; Dardiri and Brown, 1989) showed that the brown dog tick *Rhipicephalus sanguineus sanguineus* is able to transmit AHS virus experimentally from infected dogs and horses to healthy dogs and horses. Trans-stadial transmission occurred from larvae to nymphs and from nymphs to adult but transovarial transmission was absent.

Culicoides

Subsequent to Du Toit's original work with S African *Culicoides* in 1944 and 1945 no studies involving AHS virus and *Culicoides* species seem to have been published until the reports of Kitaoka (1966) using *C puncticollis* and Wetzel *et al* (1970) using *C pallidipennis* (= *imicola*). Both of these studies failed to demonstrate either AHS virus replication in *Culicoides* or transmission by them. However, Boorman *et al* (1975) and Mellor *et al* (1975) using colonized *C variipennis* finally confirmed Du Toit's original work and demonstrated for the first time that AHS virus is able to replicate (by a factor of up to 10 000-fold) after oral ingestion by a species of *Culicoides*. These authors also showed that transmission could occur after 7–10 d incubation at 26 °C. Mellor *et al* (1975) further showed that the infection rates of *C variipennis* increased in a linear relationship with the titre of virus in the blood meal, to reach a peak at $\approx 10^{5.0} \log_{10}$ (mouse infective dose) $\text{MID}_{50}/0.02$ ml when $\approx 35\%$ of engorging midges were

persistently infected. Additional increases in virus titre failed to produce any change in infection rate indicating that at an infecting titre of $10^{5.0} \log_{10} \text{MID}_{50}/0.02$ ml the infection rate (IR) is equal to the susceptibility rate (SR) (Mellor, 1990). No *C variipennis* were persistently infected with AHS virus when the virus titre of the infecting blood meal was equal to or less than $10^{4.0} \log_{10} \text{MID}_{50}/0.02$ ml. It is likely that the failure of Kitaoka (1966) to record AHS virus replication in *C puncticollis* was due to his selection of a species that is probably unsusceptible to oral infection with the virus (Mellor *et al*, 1981). The negative results of Wetzel *et al* (1970) who were dealing with an apparently susceptible species of *Culicoides* may have been due to the low infecting titres of virus used in their experiments (*ie*, around $10^{2.8} \text{MID}_{50}/0.03$ ml) as compared to the minimum titres of $10^{4.0}$ – $10^{4.5} \text{MID}_{50}/0.02$ ml found necessary by Mellor *et al* (1975).

In 1985, Erasmus (personal communication; cited in Venter *et al*, 1991) reported the isolation of AHS virus serotypes 2, 4 and 7 from field collected non-blood-engorged *C imicola* in S Africa and Blackburn *et al* (1985) also isolated AHS virus serotype 4 from non-blood-engorged *C imicola* in Zimbabwe. Apparently over the years numerous isolations of AHS virus have been made from wild-caught *C imicola* in S Africa but none have been made from other species of *Culicoides* or from non-blood-engorged mosquitoes (Erasmus, personal communication, 1988). However, recently Mellor *et al* (1990b) have reported several isolations of AHS virus serotype 4 from non-blood-engorged, wild-caught *Culicoides* during the 1988 epizootic in Spain. While 4 of these isolations were from *C imicola* 2 were from mixed pools of *Culicoides* consisting mainly of *C pulicaris* and *C obsoletus* but excluding *C imicola*. The 2 isolations from 'other' species of *Culicoides* originated from insect

catches in which *C imicola* were also present, but on testing these proved to be negative for AHS virus. There is therefore clearly no possibility of AHS virus from infected *C imicola* 'contaminating' specimens of other *Culicoides* species in the same insect catch and consequently these 2 isolations must be considered as being valid. The final AHS virus isolation from insects reported by Mellor *et al* (1990b) came from a pool of blood-engorged mosquitoes. Its significance is therefore difficult to interpret since the presence of virus may merely reflect a recent viraemic blood meal. However, the fact that mosquitoes are clearly able to acquire AHS virus from viraemic hosts in this way means that they are potentially capable of transmitting it, either biologically as Ozawa *et al* (1966a, b, 1970) suggested or possibly even mechanically.

Further studies dealing with detailed aspects of the virogenesis of AHS virus in *Culicoides* are seemingly not yet available. However, it is of relevance to note that those species of *Culicoides* which are either proven or suspect vectors of AHS virus (*C imicola*, *C variipennis*, *C obsoletus*, *C pulicaris*) have also been implicated in the transmission of the related BT viruses (Mellor *et al*, 1990b). In other words these 2 virus groups have several vector species of *Culicoides* in common. Therefore it is logical to suppose that many of the principles expounded in the recent review by Mellor (1990) which deals with the replication of BT virus in *Culicoides* will find equal application to the replication of AHS virus in *Culicoides*. While it would be inappropriate to reiterate the detailed conclusions of the BT paper here, the general cycle of events that any arbovirus, including AHS virus, must follow through a competent insect vector may be summarised as follows. Virus is ingested as part of a blood meal from a viraemic host and is deposited in the lumen of the hind part of the vector's

mid-gut. The virus attaches to the luminal surface of the mid-gut cells, infects these cells and replicates in them. Progeny virus is then released through the basement lamina into the haemocoel from where the secondary target cells or organs, including the salivary glands, are infected. Subsequent to virus replication in the salivary glands transmission may take place. Individual vectors once persistently infected usually remain so for life. However, it should be remembered that not all female insects within a vector species are necessarily susceptible to infection with a particular arbovirus, or if infected are competent to transmit that virus. A series of barriers or constraints exists within certain individuals of a vector species which either prevent virus infection or else restrict it in such a way as to prevent transmission. These refractory and susceptible traits within a vector species are under genetic control but the mechanisms by which they are expressed are as yet poorly understood (Mellor, 1990).

EPIDEMIOLOGICAL CONSIDERATIONS

Apart from the ingestion of virus contaminated meat by dogs (Van Rensburg *et al*, 1981; Hess, 1988) AHS virus is transmitted in nature between its vertebrate hosts virtually exclusively by the bites of certain species of haematophagous arthropods. Its geographical distribution and seasonal incidence is therefore limited not only by the requirement for susceptible vertebrate hosts but also by the necessity for competent arthropod vectors. In most circumstances it is the arthropod vector which is the controlling factor. This is evidenced by the observations of most workers in the field, who have shown that temperature and moisture are the main factors determining the incidence of AHS (and the prevalence of the vectors) and that the disease

disappears abruptly after the first frosts, despite the continuing presence of large numbers of susceptible vertebrate hosts (Hess, 1988). Therefore since throughout recorded history AHS has made periodic excursions beyond its enzootic zones in sub-Saharan Africa it follows that competent anthropod vectors must also be present at least at certain times or seasons in these epizootic areas. Indeed, Sellers *et al* (1977) and Sellers (1980) have amassed a considerable amount of convincing circumstantial evidence to suggest that the emergence of AHS from its enzootic zones may frequently be due to long range dispersal flights by infected vectors travelling with the prevailing winds. These authors infer flight ranges of up to 700 km and consider that wind borne infected vectors were the most likely cause of the spread of AHS from Morocco to Spain in 1966, from Turkey to Cyprus in 1969, and from Senegal to the Cape Verde Islands in 1943. However, until the recent series of epizootics in Spain, Portugal and Morocco (1987–1991) AHS virus has apparently been unable to persist for more than 2–3 consecutive years at most, beyond its traditional enzootic zones (Rafiyi, 1961; Gorhe *et al*, 1965; Bourdin, 1973; Mirchamsy and Hazrati, 1973). At first sight the reasons for this are difficult to determine, particularly in view of the fact that the related BT virus group, which utilises similar vectors, has succeeded admirably in establishing itself apparently permanently, across Asia, Australia and the Americas (Mellor, 1990). However BT virus has 2 major advantages. It infects ruminants which are generally far more abundant than are equines and the viraemia it causes in some ruminant species, particularly cattle, can be as long as 70–100 d (Nevill, 1971; Osburn *et al*, 1983). In the absence of a true carrier animal or of transovarial transmission in the vector this extended period of viraemia could consti-

tute an overwintering mechanism for BT virus, particularly in those parts of its range where adverse climatic conditions curtail vector activity for up to 2–3 months of the year.

AHS virus conversely causes a much briefer viraemia in its vertebrate hosts. This ranges from a maximum of 27 d in zebra down to 18 d in the horse, although in both species it is usually much less (Erasmus *et al*, 1978; personal communication, 1988). The duration of viraemia in donkeys and mules has apparently not been determined with any degree of certainty but it seems to be somewhat longer than in horses, though less than in zebra (Erasmus, personal communication, 1988) while in dogs it is usually considered to be transitory (Anonymous, 1978). Therefore in the absence of a long-term vertebrate reservoir and of transovarial transmission through the vector, AHS virus will presumably only be able to survive *via* continuous and uninterrupted cycles of transmission between its vertebrate and invertebrate hosts, with no 'vector-free' period being greater than the maximum duration of viraemia. In much of sub-Saharan Africa the climatic conditions are suitable for continuous vector activity throughout the year. Under such conditions cycling of virus between vertebrate and invertebrate hosts will proceed without interruption and also without the need for an "unidentified vertebrate reservoir". Further north and south, climatic conditions are likely to be less conducive to continuous vector activity and an annual 'vector-free' period will occur. This period will tend to occupy an increasing portion of the year as distance from the equator increases. When the length of the vector-free period exceeds the duration of viraemia in the local susceptible vertebrate population AHS virus will be unable to persist and should it occur at all, will only do so in epizootic form. Such epizootics are likely to be a regular or annual event when

the 'vector-free' period is relatively short and when AHS virus enzootic zones are adjacent, but will be sporadic or occasional if the 'vector-free' period is lengthy and should AHS virus enzootic zones be remote. This latter is the situation which until recently seemed to apply to southern Europe and N Africa. However, the 1987–1991 epizootics in the area patently do not fit into this pattern and the persistence of AHS virus in the western Mediterranean region for 5 consecutive yr (at least) is an unprecedented situation (Mellor, 1991). The question is therefore, how has the virus managed to establish itself in this area and in the face of a concerted vaccination and vector abatement campaign (Diaz Yubero, 1987; Anonymous, 1987, 1992; Lubroth, 1988).

It is clear that the transmission of AHS virus in Iberia (and Morocco) is closely linked with the presence of *C imicola*, the only confirmed field vector (Erasmus, 1987, personal communication). *Culicoides imicola* is an insect which is basically an Afro-Asian species and it is present throughout Africa (Walker and Davis, 1971; Wirth and Dyce, 1985) and as far east as Laos (Howarth, 1985). It was only as recently as 1982 that *C imicola* was first recorded in Europe, from Cordoba in southern Spain (Mellor *et al*, 1983) and not until 1984 that it was first identified in Portugal (Mellor *et al*, 1985). Since that time we have learned that it is widely distributed in Iberia ranging as far north at least as Madrid and Toledo in Spain (Mellor and Boned 1988, unpublished observations) and Vila Real (41°30'N) in northern Portugal (Capela and Caeiro, 1991). It is likely that in common with most other species of *Culicoides* the distribution of *C imicola* is dependent upon a series of environmental factors including topography, temperature, availability of vertebrate hosts and availability of suitable breeding sites. Furthermore, its range will vary with the prevailing

weather conditions, moving north in favourable years and retreating south again during adverse conditions. At the moment we have very little idea of the amplitude and frequency of these variations or of the precise environmental conditions that control them (Mellor, 1987). However, in the context of AHS virus persistence it is now known that there are areas of southern Spain and southern Portugal where *C imicola* is present in the adult phase throughout the year (Mellor and Boned, 1989; unpublished data; Capela, 1992; personal communication). Bearing in mind the apparent absence of a long-term vertebrate reservoir (at least in Europe), it is this 'all the year round' presence of adult *C imicola* due possibly to some recent moderation of the climate, which has facilitated the overwintering of AHS virus in southern Spain and/or Morocco from 1987–1991. Similarly the inability of the virus to overwinter in the Madrid–Toledo area of Spain, following its original introduction in 1987, was due to the fact that adult *C imicola* disappear off the wing around the end of November in this area and do not reappear until the following April (Mellor and Boned, 1989; unpublished data). Consequently in central Spain *C imicola* is either not present or else is only present as larvae for 3–4 months of the year, far too long for AHS virus to persist in vertebrates alone.

The last officially reported equine death in central Spain during 1987 occurred in mid-October (Diaz Yubero, 1987) while the first clinical cases in southern Spain in 1988 were not detected until early October, almost 12 months later (Mellor *et al*, 1990b). The long 'apparent', inter-epizootic silence plus the considerable distance of > 500 km between the 1987 and 1988 outbreak areas gave rise to a number of theories regarding their separate origin. These have included the transport of infected equines and the flight of infected vectors both from N Africa during 1988, and vac-

cine reversion. None of these theories are likely. Unlike 1966 when there was an epizootic of AHS virus serotype 9 centred in N Africa, which may well have given rise to the Spanish outbreak of the same serotype in that year (Sellers *et al*, 1977), in 1987 and 1988 there was no evidence at all of AHS anywhere across N Africa. Vaccine reversion is equally unlikely in view of the time period of > 9 months between the end of vaccination in 1987 and the outbreak in 1988. Also the S African vaccines used in Spain have apparently never been known to revert to virulence (Erasmus, personal communication, 1988).

All of the recent series of AHS epizootics in Spain, Portugal and Morocco (1987–1991) have been due to AHS virus serotype 4 (Lubroth, 1988; Mellor *et al*, 1990b; Hooghuis, personal communication, 1991; Anonymous, 1992). This is serotype which has never previously been recorded outside sub-Saharan Africa and at no time during the course of these epizootics has there been any other evidence to suggest that AHS virus type 4 is spreading out of its enzootic zones (Mellor, 1991). It therefore seems reasonably certain that there was only one introduction of AHS virus type 4 into the western Mediterranean area and that was into central Spain in 1987. Since that time the virus has persisted, overwintering 4 times in the process.

The long periods of apparent quiescence between the annual disease episodes that were seen in Spain from 1987 to 1990, are a feature that is reasonably common to several *Culicoides*-transmitted Orbiviruses in the Northern Hemisphere. Epizootic haemorrhagic disease of deer virus and BT virus may both cause annual bouts of disease in the late summer and autumn (Inaba, 1975; Yonguc *et al*, 1982; Osburn *et al*, 1983; Anonymous, 1991). This is related to the population densities of the vector species of *Culicoides* which

tend to peak at that time of the year, a situation which also applies in Spain and Portugal (Mellor and Boned, 1988; unpublished data; Capela, 1991; personal communication). This being so the most likely explanation for the observed incidence of AHS in southern Spain is that even during the inter-epizootic periods, the virus continued to circulate between susceptible equines and insect vectors but at a very low level due to reduced vector population densities and activity rates. In such a situation any infections at all in horses would possibly still have been detected by the authorities but the infection of a small number of donkeys or mules, which both tend to present a less dramatic clinical picture, might have escaped notice. The increased life span of the adult vectors during the cooler times of the year, coupled with a longer period of viraemia in donkeys and mules, would also tend to reduce the number of transmissions necessary to maintain the virus.

Although *C imicola* has long been the only confirmed (*Culicoides*) field vector of AHS virus, isolations of this virus have now been made in Spain from mixed pools of non-engorged *Culicoides* consisting almost entirely of *C obsoletus* and *C pulicaris* but excluding *C imicola* (Mellor *et al*, 1990). The range of *C obsoletus* and *C pulicaris* extends much further north than that of *C imicola* and these species are probably among the commonest *Culicoides* in northern Europe. Intuitively one feels that if either or both of them have been involved in AHS virus transmission in Spain then they are likely to have been of much less importance than *C imicola*. However, this assessment is based upon the absence of any other records linking *C obsoletus* and *C pulicaris* with AHS. Since AHS only rarely extends as far north as Spain (and Portugal), it may be that the paucity of evidence linking these 2 species with this virus have more to do with a lack of oppor-

tunity than with vector incompetence. Furthermore, it is well documented that different populations of a vector species of *Culicoides* can vary widely in their ability to transmit a particular virus (Jones and Foster, 1978; Jennings and Mellor, 1987), therefore some European populations of *C. obsoletus* and *C. pulicaris* may prove to be more efficient AHS virus vectors than populations of the same species further south. It is also the case that species of *Culicoides* which are competent to transmit AHS virus are, as has been mentioned earlier in this paper, also able to transmit the closely related BT viruses. If these 2 virus groups do usually share common vectors then it may be of importance to note that both *C. pulicaris* and *C. obsoletus* have been implicated as potential vectors of BT virus (Mellor and Pitzolis, 1979; Jennings and Mellor, 1988). It is possible that the northerly extension in the range of AHS virus to include SW Europe may have brought the virus into contact with new, previously unsuspected vectors which, without the vigorous control programmes implemented by the Spanish and Portuguese Veterinary Authorities, could have precipitated even further northwards spread of the disease. It is clearly of importance that the competence of all potential vector species of *Culicoides* in areas considered to be vulnerable to AHS virus incursion should be elucidated, so that risk can be assessed, and if necessary effective control measures devised and implemented in advance of possible disease outbreaks.

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