Ecology of African Horsesickness

P. Bourdin
1. E. M. V. T. Laboratoire de Recherches Vétérinaires, Dakar

I. Introduction

Ecologic study of animal disease is defined as the study of interactions between environment and the respective populations in a medical sense.

The so-defined ecology is related by Schwabe [53] to epidemiology, but he says ‘an epidemiology seen by an ecologist or a biologist who forgets his inborn anthropocentrism to sacrifice himself to holistic discipline’. The ecologist, be he physician or veterinarian, has to take an interest not only in the mode of disease transmission or restricted epidemiology, but also in comparative pathology, immunology, vertebrate and invertebrate zoology, climatology, agronomy, geography and even history.

Schwabe [53] has developed a 3-stage working method for ecologists:

First stage. The research worker registers observations, descriptive stage.

Second stage. He compares and analyzes his observations, analytic stage.

Third stage. He verifies hypotheses developed during the analytic stage by experimental work, experimental stage.

The ecology of African horsesickness (AHS) is related to arbovirus ecology, which has led to the classification of AHS virus within this group. Ollermann et al. [35] classify AHS virus together with Bluetongue virus in the subgroup of diplomaviruses. AHS ecology resembles arbovirus ecology and suggests the presence of a vector belonging to blood-sucking arthropods and the existence of a host. To date, experimental proof of vectorial transmission according to the World Health Organization criteria [5] has not been established and the existence of a natural host or reservoir...
is hypothetical. But if the identification of one or more reservoirs is of no consequence for endemically infected countries [27], it is of primary importance for endangered countries to prevent the introduction and distribution of the virus. We will enumerate in this article the most important points known and we will then deal with three as yet unanswered questions: appearance of an epizootic, existence of a vector and of a reservoir. These three questions are intimately linked, the ecology being the whole. In a future research program it will be indispensable to clarify first the problem of the reservoir; next that of the vector(s), Definition of the epizootiology of AHS depends directly on solution of these problems.

II. Analysis of Facts and Experimental Work Concerning Ecology

In this section discussion will be restricted to facts and the most useful experimental work relating to ecologic study. The reviews of CURASSON [12] and HENNING [20], as well as the monographs of RAFYI [48], HOWELL [21], MORNET and GILBERT [30], STELLMANN et al. [56, 57], give a much more detailed account.

A. Study of Facts

1. Geographic Distribution in the Past and Present

The cradle of the disease lies in Africa, more precisely in the countries on each side of the equator in the zones of dry tropical climate, i.e. Sahel-Sudanese climate between the isohytes from 250 to 1,000 mm. These zones may be extended by altitude [6] as is evident from figure 1. South of the equator the zones include Mozambique, Rhodesia, Zambia, Tanzania, Kenya, Angola, Transvaal, Natal and part of the Orange Free State as far as South Africa is concerned. North of the equator there is no marked boundary and the respective countries lie between the 10th and 12th parallel of latitude with a ventriflexion of the band from west to east and a dorsiflexion near the Ethiopian mountains. This band includes Senegal, the south of Mali and of Niger, Upper Volta, the south of Chad, Sudan up to Khartoum, part of Ethiopia and of Eritrea. This zone has for centuries primarily been agro-pastoral or sylvo-pastoral and the use of horses as draught or prestige animals is limited in the north by the desert and in the south by the tsetse fly [25].
From time to time AHS breaks out of its original cradle and extends into neighboring countries. This extension reaches the Cape region in South Africa, where the disease has been known since the 17th Century and recurs about every 20 years. North of the equator the spread of AHS is much less regular. In 1930-1931 AHS reached Egypt. In 1933-1934 it was again observed in Egypt, Palestine and the Lebanon. In 1959, Iran was reached and the disease spread east from there, extending into Afghanistan, Pakistan and India (1960); to the west it was recognized in Iraq, Jordan, Syria, Turkey, the Lebanon and Cyprus.

In 1965, AHS conquered the Saharian barrier and invaded the south of Morocco and Algeria. After being dormant during winter it wandered north into Tunisia and reached Spain in October 1966.

Since then the disease has apparently receded into its originating African zone to prepare for a new eruption.
2. Spread of AHS

AHS occurs endemically and periodically in the form of epidemics. The endemic form is habitually seen in the countries of western and central Africa with Sahelian climate favoring the breeding of horses. In this zone some kind of ecologic equilibrium seems to exist between vector, reservoir and the 1.5 million horses [4].

The horse was introduced into Africa south of the Sahara about 2,000 B.C.: ‘The inventory of Saharian rock dwellings shows that between this time and the start of our era the Sahara was crossed by numerous routes on two axes, one going from Lybia to Gao, the other from South Morocco to Mauritania and Tombuctu’ [25]. This stage was followed by trading with horses between the salt-producing countries of the Maghreb and the kingdoms of western Africa rich in gold mines. Commerce in horses was very active up to the Middle Ages.

It is probable that these animals have acquired progressively a natural resistance against AHS, a feature of the local breeds of today. That there must have been contact with the virus has been proved by serological surveillance studies carried out in nonvaccinated animals by MAURICE and PROVOST in Central Africa [26] and BOURDIN et al. in Senegal and Mali [8].

The ecologic equilibrium in these countries is upset by the importation of susceptible horses. Between 1882 and 1925, animais imported from France or Morocco by the military authorities were regularly decimated until a decision was taken to use only native horses in the mounted units. Since then AHS has become rare; it was observed occasionally when horses were imported for riding clubs or studs. MORNET [29] has seen it in Senegal, DUOTRE and LECLERC identified it in Chad [13] and BOURDIN [9] again in Senegal. GILBERT [19] has observed the disease in animals sired by imported stallions out of native mares.

The epidemic form seen within the last decade in the Middle East, Asia and the Maghreb has been known in South Africa since the 17th Century in the Cape region, when horses were first introduced there. In the more septentrional (northern) provinces of the South African Union (Natal, Transvaal) the horses, the majority being vaccinated, are very probably in close contact with the wild virus [17]. One can presume that there has been established in these regions some sort of artificial equilibrium.

The spread of AHS virus in new countries seems to be linked, according to HOWELL [21, 22], to the introduction of equidae or contaminated insect vectors, which find a zone with favorable ecologic conditions. He applies
this explanation to the appearance of AHS during the summer of 1959 in the coastal regions of Iran. After a winter pause the disease spread in 1960 along the rivers, the traditional nomadic routes and the major road axes.

For PILO-MORON et al. [45], AHS arrived in the south of Morocco and Algeria following the habitual routes (fig. 2). It is estimated that 40,000–50,000 nomads still live in south Algeria and that these people migrate between southern Morocco and Algeria and the Sahelian regions of Mali and Niger. These people migrate with herds consisting of dromedaries, donkeys, sheep, goats and a few dogs. The hypothesis of spread along these migratory routes is based on the presence of neutralizing and complement-fixing antibodies in donkeys living in the oases through which the routes lead. These animals could perhaps serve as markers or relays.

3. Climate and Topography

All authors stress the favorable influence of high humidity always following abundant rains and heat. Cold and drought are, on the contrary, unfavorable factors.

Fig. 2. Traditional migratory routes used by nomadic populations.
In the south of Africa, where the rainy season starts in December, the most dangerous months are February, March and sometimes April. In countries with a Sahelo-Sudanian climate the rain season lasts for 2-4 months with a maximum in August; it is followed by a rigorous dry season lasting 6-8 months. The vapor tension may attain 18-22 mm between the middle of June and the middle of November [6]. The months of October and November, in particular, favor the eruption of AHS.

Countries with a continental or Mediterranean climate show a distinct climatic incidence. Rafyi [48] noted in Iran that the disease spread in summer 1959, stopped after the first frosts and reappeared in the following year. In Algeria [54] the disease is widespread in October, regresses with the cold and is reactivated the following season. In Morocco, AHS reached alarming proportions at the end of the winter of 1965 to become extinguished in the summer of 1966 [24]. This writer mentioned that spring and summer, 1966, were relatively dry but were preceded by a very rainy autumn in 1965, which covered the valleys with an abundant flora.

Topographically, the zones favoring the spread of AHS are the low, swampy regions, the humid valleys with abundant flora [20], water courses, wells, puddles, mudholes and bore-holes [24, 48, 49, 54]. In mountainous regions, AHS is restricted to the lower zones. Theiler [59] observed AHS in South Africa usually below 500 m; under exceptional climatic conditions it was seen up to 1,200 m. In Ethiopia and Eritrea the disease does not occur at altitudes higher than 1,200–1,500 m [47]. In Kenya, closer to the equator, Pirani [46] found a focus at 2,400 m altitude.

4. Susceptibility of Animals

The horse is extremely susceptible, with a morbidity of more than 95% in epizootics and a mortality of 85% [48]. The mule is less susceptible with a 50-percent morbidity during epizootics.

The South African donkey (Equus asinus somalicus) is resistant [61]. Fatal cases have been seen in Egypt and Algeria in donkeys of the subspecies Equus asinus africanus [2, 54]. Orue [37] observed a very high mortality among the donkeys of the Cape Vert region. The zebra (Equus burchelli) may show a clinically evident form of disease in South Africa [22].

The dog is infected by ingestion [62]. Piercy [43] has described enzootics in animals of packs nourished with contaminated horse-meat.

The Angora goat, not susceptible under natural conditions, produces a febrile response to virus inoculation. The blood of such animals is virulent [62, 64].
5. Mode of Transmission

Direct transmission is exceptional and, if it occurs, it is by accident only. It is possible to keep healthy animals together with sick ones in an insect-free stable protected from blood-sucking arthropods. The massive use of insecticides during epizootics stopped the spread of virus in Spain in 1966. These two facts and the influence of climate support the theory of insect-borne transmission.

B. Analysis of Experimental Work

1. Resistance of AHS Virus to External Factors

The virus is stable outside the body. Putrefaction, dessication and temperature variation exert little effect. Only pH values below 6 are detrimental. The ideal pH for conservation varies from 6.5 to 8. Putrefaction, dessication and temperature variation exert little effect. Only pH values below 6 are detrimental. The ideal pH for conservation varies from 6.5 to 8 [1, 38].

Wild viscerotropic strains are stable at 4°C in OCG mixture [63]; neurotropic strains are stable in the presence of serum for 90 days at this temperature [1, 28]. At 37°C in the presence of calf serum the cell culture adapted neurotropic virus loses 6 logs within 40 days and only 2 logs at -20 to -25°C [000].

2. Persistence of Virus in Susceptible Animals

The horse experiences a short viremia; the virus can be found in the blood at the beginning of the febrile reaction and disappears fairly rapidly. After the ninth day, isolation from blood is only rarely achieved, except for the case described by THEILER [63] in which the disease was transmitted by massive inoculation of a healthy horse with blood from a horse that was sick 90 days previously. Viremia is difficult to detect in donkeys.

McINTOSH [27] could not isolate the virus from vaccinated horses responding to challenge. After intracardiac inoculation of ferrets, however, isolation was achieved from the blood of the ferret. The virus was pathogenic for suckling mice. The author thinks that the virus masked by antibodies in the horse was liberated in the ferret.

3. Study of Vectors: Virus in Blood-Sucking Insects and Transmission Experiments

In many articles on AHS a number of arthropods are cited as possible vectors. JOUBERT [23] compiled the following list:
But, according to this worker, many of these insects are with certainty ‘simply winged needles’ and, in fact, successful experimental transmissions can be counted on the fingers of one hand. Stomoxys calcitrans can mechanically transmit AHS virus [52]. Mosquitoes may exceptionally transmit the virus [34]. These authors found a predominance of A. caballi with some Culex and Anopheles among the mosquitoes living at Onderstepoort. They tried to transmit the disease to susceptible horses, either by inoculation with crushed mosquitoes that had fed on viremic horses 12 h to 55 days before, or by letting infected mosquitoes feed on susceptible horses. After 66 experiments with Aedes they obtained 4 positive results, all of them by inoculation. In one case the infective meal took place 12 h previously, in the three other cases 7 days before. Anopheles stephensi and C. pipiens infected horses when they were fed virus in vitro and put on horses 15-22 days after the meal [40, 41]. The horses died late, 25-38 days after having been stung. Ozawa et al. [42] continued their experiments using A. aegypti, fed in vitro with a suspension of wild type 9 virus adapted to cell cultures (titer $10^{6.2} \text{ID}_{50}/0.1 \text{ ml}$). The presence of virus in the mosquitoes was checked from 1 to 36 days; on the 36th day virus titers in certain lots of mosquitoes were still as high as $10^{2.3} \text{ ID}_{50}/0.1 \text{ ml}$. The authors explain the titer as being due to virus replication in the mosquito. AHS was also transmitted to a horse by A. aegypti which had been infected in vitro 19 days earlier. The horse died 18 days after contact with the mosquitoes.

Du Tor[14] captured Culicoides sp. in an AHS region and tried without success to transmit the disease to a horse by its inoculation with these crushed arthropods. In 1945, Culicoides were fed on a viremic horse and 12 days later were put on a susceptible horse, which died of AHS [15].

Wetz[67] fed A. aegypti and C. pipiens fatigans from the Onderstepoort collection and Culicoides sp. captured in light-traps on a virus suspension and on a viremic horse. All attempts to isolate virus from crushed arthropods in suckling mice failed 1-40 days after the infective meal; the same Culicoides were not able to infect susceptible horses.
4. Search for and Identification of the Reservoir

For the determination of a reservoir two complementary techniques can be used: intracerebral inoculation of suckling mice with material obtained from different vertebrates and a systemic serologic investigation of many species.

a) Serologic Investigations

Species suspected of harboring virus are selected and a large number of samples are tested. The complement fixation test indicates a contact within the last 6 months [22] and the reaction is group-specific. Serum neutralization detects more temporarily distant contacts and is type-specific. To our knowledge, no systematic large-scale investigations have been carried out.

PILO-MORON et al. [44] found neutralizing antibodies in 69% of 84 donkeys living in south Algerian oases. In dogs living in an AHS focus, 1 in 13 samples contained antibodies [27].

SHAH [55] was able to detect antibodies in samples from various species. 10 of 20 canine samples were positive, 8 of 42 donkeys and 1 of 42 cattle had antibodies in their serum. No antibodies were found in 34 human, 17 caprine, 21 avian and 2 ovine serum samples.

b) Inoculation of Suckling Mice or other Susceptible Species

Experimental inoculation with material from species other than equidae has only been done on a small scale. The results have always proven negative. THEILER [60] and BEVAN [7] were able to isolate virus from experimentally infected dogs. The same was possible in Angora goats [64] and in sheep [65], but THEILER'S attempts [61] to isolate a virus from wild animals, birds and amphibians were unsuccessful.

The difficulties to isolate AHS virus in nature from reservoirs or vectors are comparable to those encountered by WHO for west African arboviruses. Two centers of WHO, the Pasteur Institute in Dakar and the University of Ibadan, are systematically looking for arboviruses in man, domestic animals, wild animals and arthropods by intracerebral inoculation of 1 day-old suckling mice. In case of a positive result after 2-3 passages the sensitivity of the virus to ether and chloroform is checked and identification of the agent is pursued.

In Senegal, the material originates mainly from two centers, one 80 km from Dakar on the road to M’Bour in the forest of Bandia, the other near the Gambian border in Saboya. There are only a few horses near these centers.
because of the tsetse fly. The reports by Robin and Bres [50], Robin and Le Gonidec [51] for the virological study and by Taufflieb et al. [58] for the entomological investigations can be summarized in two tables. In these, the samples are classified in large groups according to their origin: man, wild fauna, mosquitoes, ticks, other arthropods. The group ‘wild animals’ includes mammalians (insectivores, chiroptera, primates, rodents, carnivores, artiodactyles) and ranges from the excessively numerous birds to reptiles. Liver, spleen and brain are routinely used to prepare inoculums. Mosquitoes and ticks are identified, classified and used for inoculation in lots. The group ‘other arthropods’ includes 162 lots of culicoides, 11 lots of tabanids and 24 lots of phlebotomes.

The total inoculations in 8 years amounted to 9,788 with 224 viruses isolated, only two of which were ether-resistant both being identified as Coxsackie virus (tables 1, II).

In Nigeria, the WHO arbovirologists work at Ibadan, a town situated in a humid tropical climate with very few horses. Investigations are also

Table Z. Number of inoculations with material from different species (extracted from WHO reports)

<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Man</td>
<td>113</td>
<td>552</td>
<td>284</td>
<td>19</td>
<td>54</td>
<td>332</td>
<td>162</td>
<td>1,516</td>
</tr>
<tr>
<td>Wild animals</td>
<td>171</td>
<td>297</td>
<td>529</td>
<td>291</td>
<td>448</td>
<td>241</td>
<td>1,230</td>
<td>3,207</td>
</tr>
<tr>
<td>Mosquitoes</td>
<td>5</td>
<td>113</td>
<td>145</td>
<td>488</td>
<td>327</td>
<td>483</td>
<td>1,171</td>
<td>2,732</td>
</tr>
<tr>
<td>Ticks</td>
<td>1</td>
<td>80</td>
<td>74</td>
<td>256</td>
<td>439</td>
<td>106</td>
<td>1,016</td>
<td>1,974</td>
</tr>
<tr>
<td>Other arthropods</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>154</td>
<td>205</td>
</tr>
</tbody>
</table>

1 Number of lots.
2 The group consists of the following lots: Culicoides, 297, tabanids, 14, phlebotomes, 48.

Table ZZ. Identified viruses (extracted from WHO reports)

<table>
<thead>
<tr>
<th>Origin</th>
<th>1968</th>
<th>1969</th>
<th>1970</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Man</td>
<td>39</td>
<td>16</td>
<td>2</td>
<td>57</td>
</tr>
<tr>
<td>Wild animals</td>
<td>46</td>
<td>-</td>
<td>10</td>
<td>56</td>
</tr>
<tr>
<td>Mosquitoes</td>
<td>81</td>
<td>-</td>
<td>10</td>
<td>91</td>
</tr>
<tr>
<td>Ticks</td>
<td>12</td>
<td>-</td>
<td>16</td>
<td>28</td>
</tr>
<tr>
<td>Other arthropods</td>
<td>2</td>
<td>0</td>
<td>0</td>
<td>2</td>
</tr>
</tbody>
</table>
carried out in north Nigeria, a zone of Sahelo-Sudanian climate. Other investigations take place in Chad, Dahomey, Togo and the north Cameroons. The yearly reports (1964-1969) published by the University of Ibadan in the framework of the Arbovirus Research Project can be summarized in two tables. Table III concerns the inoculations per year and the zoological groups. Table IV gives the number of viruses isolated from and identified in these groups. In this table each column contains two series of numbers: those to the left for arboviruses, those to the right for other viruses.

The samples from domestic animals are blood samples from animals from the central and northern provinces and slaughtered at Ibadan (bovine,

Table III. Yearly inoculations performed at Ibadan

<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Man</td>
<td>1,815</td>
<td>662</td>
<td>2,212</td>
<td>1,793</td>
<td>2,560</td>
<td>2,115</td>
<td>11,157</td>
</tr>
<tr>
<td>Domestic animals(^1)</td>
<td>220</td>
<td>1,568</td>
<td>1,295</td>
<td>679</td>
<td>330</td>
<td>1,048</td>
<td>5,140</td>
</tr>
<tr>
<td>Indicator animals(^2)</td>
<td>207</td>
<td>263</td>
<td>273</td>
<td>709</td>
<td>172</td>
<td>1,624</td>
<td></td>
</tr>
<tr>
<td>Wild fauna(^3)</td>
<td>438</td>
<td>1,279</td>
<td>3,429</td>
<td>2,794</td>
<td>2,723</td>
<td>10,663</td>
<td></td>
</tr>
<tr>
<td>Ticks</td>
<td>1,209</td>
<td>1,328</td>
<td>579</td>
<td>1,436</td>
<td>3,093</td>
<td>180</td>
<td>7,825</td>
</tr>
<tr>
<td>Mosquitoes</td>
<td>96</td>
<td>140</td>
<td></td>
<td>193</td>
<td>280</td>
<td>714</td>
<td>1,423</td>
</tr>
<tr>
<td>Culicoides</td>
<td>13</td>
<td></td>
<td>399</td>
<td>830</td>
<td>325</td>
<td>1,567</td>
<td></td>
</tr>
<tr>
<td>Other arthropods</td>
<td>13</td>
<td>83</td>
<td>63</td>
<td>68</td>
<td>10</td>
<td>297</td>
<td></td>
</tr>
</tbody>
</table>

1 Animals coming from central and northern Nigeria and slaughtered at Ibadan.
2 Indicator animals consist of sucking mice, chicken, rhesus monkeys and calves.
3 Mammalians, birds and reptiles captured in Nigeria, Chad and Dahomey.

Table IV. Virus isolated by near and species

<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>NA</td>
<td>A</td>
<td>NA</td>
<td>A</td>
<td>NA</td>
<td>A</td>
<td>A</td>
</tr>
<tr>
<td>Man</td>
<td>8</td>
<td>79</td>
<td>1</td>
<td>5</td>
<td>20</td>
<td>31</td>
<td>21</td>
</tr>
<tr>
<td>Domestic animals</td>
<td>14</td>
<td>-</td>
<td>160</td>
<td>2</td>
<td>45</td>
<td>13</td>
<td>13</td>
</tr>
<tr>
<td>Indicator animals</td>
<td>-</td>
<td>-</td>
<td>4</td>
<td>-</td>
<td>2</td>
<td>5</td>
<td>10</td>
</tr>
<tr>
<td>Wild fauna</td>
<td>-</td>
<td>-</td>
<td>1</td>
<td>7</td>
<td>3</td>
<td>16</td>
<td>1</td>
</tr>
<tr>
<td>Ticks</td>
<td>179</td>
<td>-</td>
<td>152</td>
<td>2</td>
<td>46</td>
<td>5</td>
<td>182</td>
</tr>
<tr>
<td>Mosquitoes</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>1</td>
<td>-</td>
<td>1</td>
<td>-</td>
</tr>
<tr>
<td>Culicoides sp.</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>44</td>
<td>3</td>
<td>7</td>
</tr>
<tr>
<td>Other arthropods</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>2</td>
<td>1</td>
<td>-</td>
</tr>
</tbody>
</table>

A = arbovirus, NA = nonarbovirus.
Ecology of African Horsesickness

ovine, caprine and porcine samples). The ‘sentinel animals’ or indicator animals (suckling mice, rhesus monkeys, chickens and calves) are exposed in the park of the University of Ibadan or on a nearby farm. The number of samples taken for inoculation amounts to 39,696 with 1,613 viruses isolated and identified in 6 years, 1,361 of these were arboviruses and 252 other viruses (herpes, myxovirus, picornavirus, poxvirus, rhabdovirus, reovirus). Among the ‘other viruses’, 9 bluetongue viruses were isolated, 8 from Culicoides sp. and one from Crocidura sp., both captured near Ibadan.

III. Discussion

AHS is an endemic, essentially African disease occurring on both sides of the equator in dry, tropical climates, such as the model Sahelo-Sudanian climate with its variants. The Sahara constitutes the northern limit of the endemic zone.

The disease appears in seasonal rhythm and is favored by heat and humidity. The horse seems to be an accidental host. In western Africa the horse has acquired a very solid natural immunity due to its very ancient cohabitation with the virus. Periodically, the disease breaks out of its habitual geographic area and reaches countries with a more temperate climate. This occurs generally during the hot season in those countries. It will disappear in winter and can sometimes recur during the following summer to become extinguished after the first frosts.

Arboviruses are transmitted in different cycles [10]. The same diversity of transmission must be expected to apply to AHS. Accordingly, we can distinguish a basic sylvatic cycle in the absence of the horse: infected vertebrate → arthropod → susceptible vertebrate.

In the proximity of such a cycle an equine population may exist and, depending on the frequency of contacts, the result will be a zone of weaker or stronger endemicity. A zone of strong endemicity is characterized by frequent contacts with susceptible populations, horses in our case, the contacts being old in adults, recent in young animals. The arbovirologists designate the form of evolution as ‘rural’, following, in the case of AHS, this scheme: infected vertebrate → arthropod → horse. Whether the horse is a dead-end is a question that remains unanswered [10].

The infected horse may be introduced into a zone with susceptible populations at a time when blood-sucking arthropods abound. The arthropods may be true biological vectors or ‘simple winged needles’; in any case,
ANS will become epidemic and the new cycle is as follows: infected horse →
arthropod → susceptible horse.

In this zone the ecologic conditions may differ from the zone where the
basic cycle takes place. If there is no reservoir, the greater variations of climatic conditions will interrupt the reproduction of vectors. In certain vectors, however, the phenomenon of the diapause may give rise to a reappearance of the virus as soon as the temperature becomes milder. Disappearance of the disease will then take place during the following winter.

This explanation seems to apply to the development of AHS in Iran and its spread to the east and the west. Howell [21] feels that the first focus was due to the introduction of infected horses from East Africa. The introduction of the virus into North Africa is more difficult to explain. In Algeria the first foci were observed in locations on the northern border of the Sahara (Bechar, Tadjmout, Laghouat) at the end of the routes used by nomads [45]. A serologic investigation of Saharian donkeys living close to the caravan routes reveals the presence of neutralizing and complement fixing antibodies at high titers. The trans-Saharan routes are used by nomadic populations amounting to about 50,000 people [66]. These nomads migrate, following the watering places of the northern Sahara toward the nearest Sahelian regions of Mali and Niger, where they find pastures for their herds consisting mainly of dromedaries, sheep, goats and donkeys. According to Pilomoron et al. [44], the donkeys (Equus asinus africanus) undergo an inapparent infection and serve as relays between Sahelian and Algerian horses. This hypothesis remains to be confirmed by virus isolation.

Ecologic circumstances point to the transmission of AHS by blood-sucking arthropods, but experimental evidence is contradictory. Two groups of vectors, mosquitoes and Culicoides, are mainly suspect. Some workers have obtained positive results with mosquitoes, negative with Culicoides; other have found the exact contrary. It is important to realize that the isolation of a virus from arthropods does not a priori imply a biological transmission. Two minimal conditions must be fulfilled, namely replication in the arthropod and transmission by the sting. Wetzel et al. [67] apply these requirements to AHS virus and plan to inoculate arthropods intrathoracically to verify if replication takes place. There is no profit in discussing the advantages and inconveniences of this artificial experiment; we shall consider only the ecological reasons and uncover the difficulties in identifying the vector.

The first difficulty is of a statistical nature. In 1944, Du Torr isolated AHS virus once from Culicoides sp. and achieved transmission in 1945 [14,
[15, 67]. In 1944, the same author also isolated bluetongue virus three times from Culicoides sp. and achieved transmission by C. pallidipennis. In the USA, FOSTER et al. [18] transmitted bluetongue with Culicoides variipennis. In 1971, NEVILL regularly isolated bluetongue from Culicoides sp. in South Africa during the warm season, provided large inoculums were given. Why this difference in spite of the close resemblance of the two viruses? Simply because the reservoir of bluetongue is represented by 12.8 million cattle and the host by 39 million sheep, whereas AHS concerns only 380,000 horses, the only vertebrate permitting isolation in South. Africa, as long as the reservoir is not identified.

The second difficulty is due to the Culicoides themselves; their identification is difficult and their biology incompletely defined. Some of them live in close contact with cattle, C. pallidipennis in particular, which lays eggs and reproduces in fresh cattle feces [32]. This Culicoides represents 97% of all those identified in Africa [11, 31, 32]. Moreover, Culicoides do not depend on a blood meal for their first oviposition. Within a lot of Culicoides captured in light-traps one can distinguish the insect having completed a gonotrophic cycle from the immature, but one cannot enumerate the cycles. The deviation of life and the frequency of contacts with the host are equally poorly defined.

The biology of mosquitoes is better known. In the case of AHS, unfortunately, the virus has never been isolated in the field, even though experimental transmission has been successful with certain species. Mosquitoes are, therefore, suspect but unconfirmed as vectors. The presence of virus in Iran and Algeria during two successive years may be explained by the persistence of the virus in certain species during hibernation. Proof remains to be supplied.

In conclusion, the criteria applied by arbovirologists have not been fulfilled for many viruses transmitted by arthropods, among them AHS virus. The easiest of these criteria is the isolation of wild virus in arthropods. The study of transmission is more difficult and the investigation of ecologic factors regulating the role of the vector can only be carried out with long-lasting and costly research work.

The last point, the reservoir, is hypothetical and, to use an expression of MORNET and GILBERT [30], it is a ‘fact of logic’. To prove its existence, however, is of utmost importance for the countries free of AHS [3].

There is no point in citing the possible reservoir species. We prefer to propose a research plan following the pattern of epidemiologic investigations of arboviruses. The starting point of this plan is a serological investigation.
tion in horses to determine the zone of strong endemicity with frequent contacts between horse and virus (rural form of the disease). These contacts imply that vector and reservoir are present. The serological investigation must, therefore, allow localization of the ecologic nidus where the virus is maintained in its basic cycle.

In Senegal, where AHS occurs habitually at the end of the hot season in October and November, Bourdin et al. [8] have examined 1,500 serum samples from different regions in December and January. The samples can be classified in two groups according to the zone of origin. The first group consists of samples of horses from agricultural regions, the second comes from agro-pastoral zones. The complement fixation test reveals the interesting feature that 20% of the ‘agricultural’ horses have antibodies compared with 66% in the ‘pastoral’ horses.

Before we comment on these results, it is necessary to summarize the mode of life of the horses from both regions. In the agricultural zone the draught horses live together with sheep in the yard of the house. They are the object of much attention and are fed and watered in the yard. In the bigger agricultural villages 50-100 horses, 300-400 sheep and a few donkeys may be found. Additionally there are cattle, usually kept in migratory herds, which come from sylvo-pastoral zones in the dry season and which live at the outskirts of the village.

In the sylvo-pastoral zone the horses live differently. This zone is situated in the northwestern part of Senegal and is essentially pastoral, breeding being extensive. Water supply has long been the limiting factor for breeding, as the only water was found in deep wells. Fortunately, the installation of drilled wells with large outputs 20 years ago has solved this problem. Around these wells 5,000–20,000 cattle and as many sheep and goats may live within a radius of 20-30 kilometers. The wells have initiated the founding of Peul’s breeder villages where some horses, used for pack work or prestige, and numerous donkeys, used for water transportation, are kept. The approach to such a well is characterized by a sandy zone barren of pasture but covered with excrement. Multitudes of birds breed close to the water and wild animals are more abundant than in the agricultural region. Bynighthesyenas and jackals are frequent visitors.

Drilled or traditional wells and their surroundings can constitute an ecological niche, in which the virus would be transmitted within its basic cycle. In the immediate surroundings of the well two elements favoring the multiplication of potential vectors are present: stagnating water, cisterns, puddles and fresh cattle feces. More distantly there lives, apart from dogs
and other domestic animals, a rather abundant wild fauna attracted by the water and here a reservoir may be found.

This first tentative investigation must be confirmed by other surveys on horses living close to other drilled wells and natural wells in the sylvopastoral zone. Later, it will become necessary to extend a serological investigation by neutralizing and complement fixing tests to other domestic and wild animals and to complete this study by capturing blood-sucking arthropods and wild animals, particularly at the end of the hot season to try to isolate the virus in suckling mice.

Acknowledgements

We would like to thank Dr. Y. ROBIN, Director of the Pasteur Institute of Dakar; Dr. M. CORNET, entomologist of ORSTOM, and Mrs. G. AME, whose precious advice has helped me to finish this work in time. We also thank Dr. DIALLO, Director of the Service de l'Elevage du Sénégal and his coworkers and our own direct coworkers: Mrs. A. LAURENT, Mr. G. BERNARD and Mr. A. M'BAYE for their efficient and competent help.

References

15 Du Toit, R. M.: Cited in Wetzel et al. [67].
17 Erasmus, B. J.: Personal communication (1971).


Author’s address: Dr. P. BOURDIN, I.E.M.V.T. Laboratoire de recherches vétérinaires, B.P. 2057, Dakar (Senegal)