African Horse Sickness
Summary

Introduction

This note provides a brief summary of an analysis undertaken by a DISCONTOOLS group of experts on African Horse Sickness (AHS). They reviewed the current knowledge on the disease, considered the existing disease control tools, identified current gaps in the availability and quality of the control tools and finally determined the research necessary to develop new or improved tools. Full details of the analysis can be downloaded from the web site at http://www.discontools.eu

Disease profile

There are nine different serotypes of AHSV (AHSV type 1-9) with marked genetic heterogeneity recognised within each serotype. The severity of clinical disease varies between each strain but it is likely that an outbreak of AHS involving any serotype could have a major impact on the equine population in a country which has never experienced the disease.

The disease is not directly contagious between horses. Transmission of AHSV occurs almost entirely through hematophagous arthropods, which act as biological vectors. AHSV is transmitted when an infected competent vector (*Culicoides* spp.) feeds on a susceptible host but only equids develop a viremia long and intense enough to complete the transmission cycle once bitten by a *Culicoides*. Horses, mules, donkeys and zebras play different epidemiological roles: the species that produce a prolonged viraemia and have low disease mortality, such as zebra and donkeys, act as reservoir hosts. As there is no carrier state, persistence of AHSV is believed to rely on sufficient density and renewal of susceptible reservoir hosts. Mortality due to AHS is related to the species of equidae affected and to the strain or serotype of the virus. In the most acute cases the mortality rate can be 95% with horses dying within a week. Mortality rate in horses is 70-95%, in mules it is around 50%, and in donkeys it is limited to 10%. Occasional hosts include elephants, onager, dogs and camels. Zebras and elephants may be infected without showing signs of disease.

The distribution of AHSV is dependent on the presence of the relevant insect vector. The disease is endemic in sub-Saharan Africa from where it occasionally spreads to other areas. A few outbreaks have occurred outside Africa, such as in the Near and Middle East (1959-63), in Spain (1966 serotype 9, 1987-90 serotype 4), in Portugal (1989-serotype 4), and Morocco (1989 – 1991 serotype 4). In 1987 AHSV was likely introduced to Spain by the importation of zebras from Namibia. The disease was successfully contained and eradicated by a combination of disease control measures (i.e. destruction, movement control and vaccination).

Risk

At least two field vectors are involved in the transmission of the virus: *Culicoides imicola* and *C. bolitinos*. The major vector of AHSV, *C. imicola*, occurs in southern Europe and northern spread is expected as global temperatures increase. As the distribution of *C. imicola* moves north, it may bring AHSV into the range of other *Culicoides* species that are potentially competent vectors and which are commonly found in northern Europe. Once infected via this 'baton effect', these species may be able to spread the virus over much of Europe.

Climate change may also increase the vector competence increasing the likelihood of viruses surviving from one year to the next.

The spread of disease is influenced by climatic conditions, which favour the spread of carrier insects (vectors) including warm, moist weather and high rainfall, as well as spread by wind dispersal. Epidemics of AHS tend to occur at cyclic intervals, and are often associated with drought followed by heavy rain presumably giving rise to large numbers of competent vectors.

Diagnostics

C-ELISA kits for AHS antibody detection are available worldwide with commercial diagnostic kits (ELISA and flow lateral assay) available in Europe. Molecular detection of AHSV is the best alternative for rapid diagnosis of AHS. The RT-PCR is a sensitive and rapid method for detecting AHSV nucleic acids during either the incubation period at the start of an AHS epizootic, or for epidemiological investigations in species where clinical signs may not be apparent. Once the disease has been confirmed, the virus needs further characterization, primarily the serotype

identification. To this aim, beside the virus neutralization test, several molecular tests have been published providing a rapid typing method for AHSV in biological samples. There is a need for validated and harmonized diagnostic assays. New developments in molecular and serological diagnostic methods will contribute to improvements in the diagnosis.

Vaccines

Currently, there are no commercially available inactivated or recombinant vaccines but there are some locally killed vaccines for use in some countries. Three types of vaccine for AHS can be considered: live attenuated, inactivated and recombinant. There are a number of live attenuated vaccines against AHS manufactured in the world. Most of them are mono- or polyvalent vaccines to be used in endemic situations. They provide a lifelong immunity after 2 or 3 doses.
Unfortunately, there is some concern about their safety and efficacy as lack of protection against the viral variants, the side effects induced by the vaccination or the interference with maternal immunity in foals. The inability to differentiate between infected and vaccinated animals is also a concern.
Furthermore, attenuated vaccines may not be safe in AHS-free countries due to the risk of transmission, reassortment (i.e. exchange of gene segments between vaccine and field strains) and reversion to virulence. No AHS vaccines are currently licensed in the EU.
It will be important to overcome the current problems with the live vaccines or, preferably, to develop new recombinant vaccines using various vectors or the reverse genetic technique.
Producing efficacious recombinant vaccines may be the way forward but would depend on a number of factors such as the better understanding of the molecular biology of the virus and the market demand for this type of AHS vaccine.
The development of cross-protective AHSV vaccines that have a long shelf life and that can provide rapid protection and be differentiated from natural infections during outbreaks is rightly seen as a major priority for research.

Pharmaceuticals

No pharmaceuticals at present apart from possible development of anti-virals or immune stimulants.

Knowledge

There are still significant areas of uncertainty in the understanding and knowledge about AHS. A better understanding of the pathogenesis is required along with increased knowledge on host immune responses to AHS. There is a need to identify the potential field vectors of AHSV beyond those already known (*C. imicola* and *C. bolitinos*). The potential mechanisms and likelihood of overwintering of AHSV in currently non-affected areas is not well understood and needs to be investigated. It is also important to carry out disease transmission modelling in currently non-affected areas along with modelling the use of vaccination as an effective control measure.

Conclusions

- There is the potential for AHS to spread into Europe and new areas due to either (i) the modification in the distribution of competent/potentially competent vectors or (ii) the modification in vector competence due to climate changes.
- The genome sequencing of AHSV circulating strains should be encouraged to assess the diagnostic capabilities of the molecular test in use and to investigate the potential for vaccine strains circulation and/or reassortment.
- There are no authorised and safe vaccines along with tests to differentiate vaccinated from infected horses. The development of cross-protective AHSV vaccines with a long shelf life and that can provide rapid protection and be differentiated from natural infections during outbreaks is a major priority for research.
- There is a need to acquire information on the pathogenesis and ecology of the virus to model the possible pathways of introduction and dissemination of the AHSV in naïve areas.