NOTES TAKEN BY ANNE LAMBOURN AT THE

EMERGING EQUINE DISEASES CONFERENCE,

NEWMARKET, 23 JUNE 2008

Summary
Brigadier Paul Jepson
Dr Richard Newton
Dr Chris Oura
Dr Philip Mellor
Prof. Alan Guthrie (AHS)
Dr Jules Minke
Prof. Traub-Dargatz
Prof. Alan Guthrie (WNV)
Matthew Hartley
Kirsten Rausing

15 July, 2008

SUMMARY OF NEWMARKET CONFERENCE, 23 JUNE 2008

EMERGING EQUINE DISEASES

The Seminar was hosted by both The Horse Trust and the Thoroughbred Breeders' Association, and supported by Merial and Fort Dodge Animal Health. The purpose of the Seminar was to raise awareness of African Horse Sickness and West Nile Virus by providing an assessment of the current situation and details of how the UK can best manage the risk and prepare for the future. With the increase in global travel, the unpredictable effects of climate change, and the arrival and rapid spread of Bluetongue in 2007 these diseases are no longer considered distant threats. "These diseases, and in particular African Horse Sickness, have the potential to devastate the UK's $4 billion equine industry and bring all equestrian activity to a halt for some considerable time".

The emphasis was very much on cooperation and communication between the equine industry, veterinary and other experts, pharmaceutical companies, government, insurance companies and other interested parties, with conference participants being urged to spread the word and to play their part in ensuring that an "effective strategy to manage an outbreak" is put in place. The current EU legislation Council Directive 92/35 was viewed as outdated and in urgent need of renewal. The overarching plan is to deliver an alternative disease control strategy based on preventive vaccination not slaughter, using state of the art diagnostics and vaccines, and incorporating the wisdom of those with first hand experience of dealing with these diseases in the field. There was a clear warning by Professor Guthrie of the dangers of a slaughter on suspicion approach to disease control. The emphasis was on being prepared before the disease arrived, so that we did not have a panic knee-jerk response, with all the unpleasant, far reaching and unforeseen consequences of that type of approach. It was not a question of if the diseases arrive, but when.

The morning session was chaired by Joe Grimwade, Chairman of the TBA Veterinary Committee, and included presentations by Dr Richard Newton (Animal Health Trust, Newmarket), Professor Philip Mellor (IAH), Professor Alan Guthrie (University of Pretoria, S. Africa), and Dr Josie Traub-Dargatz (Colorado State University).

The afternoon session was chaired by Brigadier Paul Jepson (Chief Executive of The Horse Trust), and included presentations by Professor Alan Guthrie, Dr Chris Oura (IAH), Dr Jules Minke (Merial, France), and Matthew Hartley (Defra). The seminar was brought to a close by Kirsten Rausing, Chairman of the Thoroughbred Breeders' Association.

Dr Richard Newton gave an overview of the threat to the UK of emerging equine diseases, Professor Mellor gave details on AHS and Bluetongue with reference to epidemiology, vectors and climate change, Professor Guthrie gave presentations on AHS and West Nile Virus in South Africa, and Dr Josie Traub-Dargatz gave a presentation on the WNV outbreaks in the USA. Dr Oura gave details of the role of IAH as an AHS OIE reference laboratory, with details of diagnostics, Dr Minke gave details on the new vaccine technologies for AHS and WNV and the imminent trials of recombinant AHS vaccines in S Africa, and Matthew Hartley spoke about prevention, detection and control of AHS and WNV in the UK, and "Defra working with industry in partnership". Brigadier Jepson spoke about the Working Group set up by The Horse Trust to ensure preparedness for disease, and both he and Kirsten Rausing stressed the need for action at European level to update Council Directive 92/35.

Brigadier Paul Jepson, Chief Executive The Horse Trust

Chair, Afternoon Session

Brigadier Jepson gave details of the African Horse Sickness Working Group.
"The AHSWG, established in 2007 and chaired by The Horse Trust, has brought together representatives of DEFRA, the IAH, British Horse Racing Authority, British Horse Society, Animal Health Trust, The Donkey Sanctuary, the insurance industry, British Equine Veterinary Association, Cambridge University Veterinary School, veterinary pathologists and other key interest groups from the British equestrian industries".

It is concerned with ensuring that the UK is as best prepared as possible to prevent and control AHS. During the first year it concentrated on collecting evidence from scientists and entomologists, and now the consultation process has been widened to included insurance industries and other. It has 3 aims:
i) to develop an acceptable control strategy
ii) to push for the production of effective vaccines
iii) to raise awareness of AHS, not only amongst the horse public, but also amongst politicians and at EU level.
The Group is thus working with two scenarios:
i) how best to cope with AHS under the existing control strategy
ii) and acceptable future control strategy

The message is "**Invest now in order to prevent problems later".**

Brigadier Jepson stressed the need to lobby politicians both in the UK and Europe. He also emphasised the need to campaign for better border controls in view of the very large number of animal movements (some probably illegal) into the UK and through Europe.

NB He drew attention to a forthcoming Conference in February 2009 at Adlington Equestrian Centre (Macclesfield) on climate change and disease, when all parts of the equine industry will be represented.

Dr Richard Newton, Head of Equine Epidemiology and Disease Surveillance at the Animal Health Trust, Newmarket.

EMERGING EQUINE DISEASES: OVERVIEW OF THE THREAT TO THE UK.

Dr Newton's main message was that prevention is the key. We must learn lessons from the information available now, as there is a real threat to the UK posed by such diseases as AHS, WNV, Equine Infectious Anaemia . We must act to prevent the diseases arriving and becoming endemic. He cited the arrival of WNV in the USA in 1999, BTV in Northern Europe in 2006, and EIA in Ireland in 2006. He discussed the spread of vectors and climate change, but also highlighted the role of global movements/trade ("allowing" the vector or host to be imported - jet setting mosquitoes - which could result in the parachuting in of disease), and of biological factors e.g. the EIA outbreak in Ireland.

Regarding WNV, he spoke of the 1999 outbreak in the USA which resulted in the disease spreading rapidly and becoming endemic. He referred to the success of the vaccination campaign which was started in 2001. WNV vaccine is now a core vaccine for horses in the USA. Dr Josie Traub-Dargatz of Colorado State University elaborated on the vaccination campaign in Nebraska and Colorado in her presentation.

Regarding the UK, the risk of WNV is currently low for horses resident in the UK. There is apparently no clinical WNV in UK birds (although Ernie Gould, Oxford, disputes this). Birds act as the reservoir host with mosquitoes as the vectors. Although horses (and humans) can get bitten and contract the disease, they are regarded as dead end hosts i.e. they are not a reservoir of infection (this is in contrast to AHS). The disease is not infectious from horse to horse. However, Dr Newton emphasised that the following points should be borne in mind:
i) role of migrating birds in introducing/spreading disease
ii) the effect of climate change on mosquito vector distribution.
iii) presence of susceptible native birds in the UK
iv) "jet setting" mosquitoes – both malaria and WNV have been spread in this way.
v) there are no equine vaccines for AHS and WNV registered for use at present in the EU.
vi) there is currently no equine surveillance in the UK.

His conclusion was that there is a great need for better knowledge in the UK on emerging diseases:
i) we must be better able to recognise and diagnose the disease i.e. "raise our game regarding diagnosis".
ii) we must be better prepared and use expertise of other countries e.g. S Africa and USA.
iii) we need to improve our basic knowledge about where the threats are, about transmission, biological agents, endemicity, methods of control and eradication.
iv) we have to identify what key factor is missing in the scenario (e.g. if climate and host present, but no vector), analyse the risk, and take any steps possible to prevent it coming into the country in the first place or gaining a foothold (presumably preventive vaccination, spraying aircraft for mosquitoes, strict monitoring of animal imports and stopovers and quarantine, surveillance and testing?)

NB The A. H. Trust is not offering surveillance at the present time for WNV.
The VLA has capability for diagnosis of WNV.

Dr Chris Oura, Head of Non-Vesicular Reference laboratories, IAH.

WHEN OR IF AHS STRIKES: AHS OIE REFERENCE LABORATORY ACTIVITIES AND RESPONSIBILITIES

OIE Reference Laboratories for AHS are located in the UK at IAH, Pirbright, in Spain, and in South Africa. Dr Oura gave details as follows:

Principal role of OIE laboratories *(now known as World Organisation for Animal Health):*
i) to function as a centre of expertise and standardisation of diagnostic techniques.
ii) to store and distribute biological reference products and any other reagents used in the diagnosis and control of AHS.
iii) to develop new procedures for diagnosis and control of AHS
iv) to develop new procedures for diagnosis and control of AHS
v) to gather, process, analyse and disseminate epizootiological (stet) data relevant to AHS
vi) to place expert consultants at the disposal of the OIE.

The labs are also concerned with:
i) provision of scientific and technical training for Member Countries of OIE
ii) provision of diagnostic testing facilities to Member Countries
iii) organisation of scientific meetings on behalf of the Office
iv) coordination of scientific and technical studies in collaboration with other labs or organisations.
v) publication and dissemination of information

The IAH tests all UK cases of exotic disease, it screens imported animals for antibodies (ELISA), it tests samples from round the world, it monitors movement of disease strains around the world, and it works on the development of new diagnostic tests – very rapid development in this field.

Types of tests re AHS:
1. Group specific tests used to confirm which disease:
Competitive ELISA
Gel based PCR (the sample is stained using a polyacrylamide gel. The RNA of the virus is amplified by x 100,000)
Real time RT-PCR
These confirm whether AHS. Based on blood from live animal.
2. Type specific tests used to confirm which serotype:
Virus isolation
Serotype specific PCR
Serum neutralisation SNT
Virus neutralisation VNT Use spleen and lymph samples if animal dead.

New generation diagnostic tests:
Dr Oura then described the work taking place at IAH by Peter Mertens on developing a serotype specific PCR which amplifies the particular RNA of the serotype, giving a serotype result within 24 hours. The test also does a fingerprint/sequence analysis which indicates where the virus/serotype originated. Thus these new generation tests will use real time RT-PCR tests (instead of gel staining PCR), to identify serogroup and serotype **all in a single assay** Rt(TaqManR)PCR **i.e. high throughput multiplex rt RT-PCR for AHS.** Results shown on screen. (See Contingency planning table regarding diagnostic testing).

Dr Philip Mellor, Head, Dept of Arbovirology,IAH, Pirbright.

AFRICAN HORSE SICKNESS AND BLUETONGUE: EPIDEMIOLOGY, VECTORS AND CLIMATE CHANGE.

Dr Mellor gave a detailed and authoritative presentation on the background to bluetongue and African Horse Sickness and their similarities: both viruses are arboviruses, they both have vertebrate hosts, and the disease is transmitted between the hosts by species of Culicoides midge. The virus obviously can only survive if the insect vector and host are present, but temperature, for example, is also critical: temperatures need to be sufficiently high for the virus to multiply inside the vector. Increasing temperatures also increase the efficiency of transmission by the insect vectors in Europe.

Climate change
The distribution of the insect vectors which can transmit both of these diseases has been changing with the rise in temperatures. He attributed the sudden and massive incursion of blue tongue northwards to climate change, explaining that the period 1976 to 2000 was a major warming period, and the 1990s were the warmest decade on record. Also winter temperatures showed a significant rise, meaning that some areas no longer had a vector free period. He referred to the movement north of the "Culicoides Imicola line" by 700 km since 1998, from Africa to the Mediterranean Basin (consequently C Imicola now overlaps with C Obsoletus and C pulicaris enabling the virus to be transferred to novel vectors) and to the series of BTV incursions into Europe which have had such disastrous consequences, particularly for the sheep industry with over 1.5 million deaths. He referred to the BTV8 virus "jumping" to northern Europe from Africa in 2006. Apparently the July temperatures in Maastricht in 2006 were 6 degrees higher than had ever been experienced before, and these higher temperatures meant more efficient spread of disease once it had arrived.

Research
Dr Mellor also pointed to areas where research was urgently needed e.g. overwintering, and also the breeding sites of Culicoides, as these are poorly defined . The midges need moisture to breed, but the breeding sites are varied e.g. leaf litter and dung for C. Obsoletus in the UK, wet meadows for C. Pulicaris, cattle dung for C. Dewulfi, organic enriched but not waterlogged ground for C. Imicola. He referred to investigations into cases where the cause of a bluetongue outbreak was not immediately obvious e.g. the source of one case in Cyprus was eventually traced to C. Imicola found to be breeding in a leaking irrigation pipe in a sheep pen.

African Horse Sickness
In 1966 there was an outbreak of AHS Type 1 in Spain. The disease was eliminated and did not reappear for 20 years. However, in 1987 AHS Type 4 appeared. The source was traced to zebras which had been brought into Spain from Namibia for a safari park. Zebras provide a reservoir for AHS but do not display clinical signs. The zebras were put out to graze in meadows where C. Imicola were breeding, and AHS broke out and spread. In 1988 the disease reappeared in Malaga and spread into Portugal, with the outbreak lasting until 1991. Controlled and eradicated by vaccination. (C. Imicola apparently occurs in most of Spain and Portugal now).

Disease spread
NB:
i) high mobility of vector – 10 hour flight over water, and 100km travelled. Overland spread thought to be 2 km per day. Thus disease can spread very quickly, and over a wide area, and thus be very difficult to control.
ii) role of wind (Saharan dust).
iii) big protection zone needed because of facility to travel.
iv) Culicoides can transmit a range of viruses – appearance of novel vectors.

BTV is a wake up call. "As AHSV is transmitted by the same species of biting midge as is BTV and under similar environmental conditions, the recent devastating experiences with TV suggest that Europe could also be vulnerable to future incursions form AHSV."

Professor Alan Guthrie, Director of Equine Research Centre, Faculty of Veterinary Science, University of Pretoria, South Africa, and served on OIE working groups for AHS, EI and WNV. Also AHSWG adviser.

LIVING WITH AHS: CLINICAL SIGNS, TREATMENT AND CONTROL IN SOUTH AFRICA.

Summary
Professor Guthrie gave an extremely stimulating and informative presentation which was supplemented by information from papers/articles by him which were distributed at the Conference. He supported his talk with graphic pictures of the effects of the disease. The main thrust of his message was:
i) it is **not if but when** AHS arrives.
ii) the disease has extremely serious consequences for the equine industry with high mortality and the potential for devastating the industry.
iii) with changes in climate and the potential for all Culicoides to be vectors of AHS the threat is very real.
iv) although the UK is not the number one risk area, with blue tongue being currently of much more significance, that does not diminish the need to act now.
v) there is always a risk the disease could be parachuted in e.g. Spain, due to international trade/movements of horses, and being spread by novel vectors. Competent vectors exist in most parts of the world so one has to take care not to introduce the virus in the first place.
vi) we should vaccinate even though there are perceived drawbacks to current vaccines. Vaccination with live attenuated vaccines has been successful in South Africa.
vii) there is a very urgent need to develop the new recombinant vaccines acceptable to the EU. Trials of canarypox platform vaccines (Merial) are going ahead within the next 2 months in South Africa – based on cooperation/collaboration between Jules Minke (Merial), Professor Guthrie, and Prof. James McClachlan of UCDavis
viii) **slaughter on suspicion is counterproductive** as it discourages reporting of suspected disease and encourages illegal movement, both of which have very serious consequences in terms of disease spread.
ix) strict movement controls, vaccination, stabling of horses dawn to dusk, vector control, surveillance all implemented as soon as disease suspected.

Below I have made a summary of some of the main issues covered in the presentation. Further detail can be found in the above-mentioned papers.

Occurrence
AHS is endemic in eastern, central Africa and much of southern Africa. Progress north blocked by Sahara. It is not regarded as endemic in most of South Africa, except for the north eastern area. Here it appears each year, usually in January. It spreads south, optimum conditions being early heavy rains followed by warm dry weather. Height of infection usually March and April. Dies off with first frosts at end of April/May. NB Regionalisation of South Africa for purposes of trade in 1997 – Cape Town province allowed to export horses to EU.

Vector and host
i) Culicoides bolitinos and imicola principally in S Africa, but **potential for transmission of AHS by all Culicoides**.
ii) The primary cycle is between Culicoides and the zebra/African donkey. The secondary cycle is between Culicoides and horses. "In view of the high mortality in horses, this species is regarded as an accidental or indicator host".
iii) In South Africa, zebras (and probably African donkeys as well), if in large enough numbers, can act as a reservoir of disease e.g. Kruger National Park where there is a continuous transmission cycle between midge and zebra.
iv) Zebras and African donkeys largely asymptomatic or very mild fever. Horses have severe symptoms with high fatalities, usually 70-95% depending on form of disease (see below). Mules less susceptible with fatalities of 50-70%
v) Horses are **not** carriers of the disease –viraemia for only 28 days in horses.
vi) Biting flies, as opposed to midges, may play a small role in transmission – this is not thought to be significant for a variety of factors.
vii) Dogs can contract the highly fatal form of AHS if they have fed on carcase of animal that died of AHS. However, Culicoides do not readily feed on dogs, so dogs are not associated with spread or maintenance of AHSV.

Serotypes
There are 9 serotypes in tropical Africa, with 3, 4 and 9 occurring outside Africa.
All can be found in South Africa.

Diagnosis
NB Differential diagnosis as clinical signs of cardiac form of AHS similar to equine encephalosis.
OIE listed disease so suspected cases must have laboratory confirmation.
Use blood samples during febrile stage, or specimens of lungs, spleen and lymph nodes at necropsy.

Forms of disease
Dunkop or pulmonary form: peracute form with 95% mortality, incubation period of 3 to 4 days, followed by high fever and very rapid progressive respiratory failure due to severe oedema of the lungs and hydrothorax. Death occurs usually within 30 minutes to a few hours of first signs of respiratory problems.
Dikkop or cardiac form: 50%+ mortality, incubation of 5 to 7 days, followed by fever for 3 to 4 days. Death within 4 to 8 days of onset of fever. Oedamatous swellings of the head (swelling in supraorbital fossae characteristic) and neck, and tongue sometimes swollen and cyanotic. Severe lesions in the heart. "Severe hydropericardium is almost invariably present". Lesions in digestive tract are more severe than in Dunkop form.
Mixed form: most common form of AHS but rarely diagnosed clinically – found on pm. Two forms: i) mild pulmonary distress followed by oedematous swellings, then cardiac failure.
ii) initial "subclinical cardiac form suddenly followed by marked respiratory distress and other signs typical of the pulmonary form. Death usually occurs 3 to 6 days after the onset of the febrile reaction".
Horsesickness fever: usually just a mild fever. Other clinical signs rare. Usually in donkeys and zebra or partially immune horses that succumb to infection.

Treatment
No specific treatment, just supportive treatment, nursing and rest. Slightest exertion may cause death. Light work can only be resumed at least 4 weeks after recovery.

Control
Prophylactic vaccination in South Africa.
Three courses advocated:
i) as weanlings at 6 to 12 months and then
ii) as yearlings at 12 to 18 months and then
iii) annual revaccination.
Usually vaccinate in late winter or early summer (September to November) well before the peak season for the disease (March/April).
Live polyvalent attenuated vaccine supplied by Onderstepoort Biological Products.
Two components given three weeks apart: Bottle 1 - serotypes 1,3,4
Bottle 2 – serotypes 2,6, 7, 8
Cross protection is afforded by 6 against 9, and by 8 against 5.

Control of outbreaks
Define Infected Area and apply strict controls swiftly: no movement in or out of horses, stabling of horses dusk to dawn, vector control, rectal temperatures of horses taken twice daily, animals with fever may be killed (welfare) **or "housed in insect-free stables to prevent spread of the disease",** vaccination of all susceptible animals (S Africa vaccinates with modified live vaccine even in the face of an outbreak – probably not acceptable to Europe). Movement restrictions plus vaccination in the Protection and Surveillance zones, and vector insect control**. No euthanasia of suspected AHS (unless welfare grounds).** This policy encourages reporting and reduces the urge to move animals illegally. S Africa now has 20 to 40% overreporting of the disease as a result of this approach. The crucial benefits of this approach are that it stops the movement of animals and encourages cooperation in effecting the disease control policy.
For more detail see "African Horse sickness" by J A W Coetzer and AJ Guthrie, Page 1231 onwards, Volume 2 Infectious Diseases of Livestock edited J Coetzer and R C Tustin, OUP.

Outbreaks outside Africa e.g.
1950s-60s Middle East and South West Asia – had huge effect on disadvantaged communities.
1987-91 European outbreaks. Costs in excess of £30 million. Virus overwintered. On June 1, 1987 shipment of zebras left Namibia, arrived in Europe on 16 June, and in Spain on 18 June. Destination a safari park. On 14 July first deaths occurred of horses adjacent to the safari park. Disease not diagnosed until 14 September – thus awareness paramount.
Vaccination used in Europe including Onderstepoort Biological Products Combination Bottle 1 (modified live vaccine), and Equipest (Merial) inactivated monovalent vaccine for strain 4 (no longer commercially available).

Types of vaccine:
Live Vaccines
Advs:
Very effective
Simple production
Stable for years. Major impact in endemic areas.

Disadvs:
Variable attenuation – some reaction and even death.
Variable immune response
Vector transmission – fears that in these polyvalent vaccines insect vector can pick up other strains in the vaccines and transmit them.
Reassortment possible.
Possible maternal antibody interference by vaccine in foals born to immune dams, so vaccination not recommended until foals 6 months old. However, levels of antibodies in foals depend on immunity of dam, and with foals which only acquire low levels of antibody they could be vulnerable when not vaccinated or only vaccinated once (it is recommended that horses should be vaccinated twice – as weanlings (6 to 12 months), and then as yearlings (12 to 18 months) and then yearly.

Inactivated vaccines

Advs:
Efficient
Safe
No transmission

Disadvs:
Complex to produce and store
Monovalent – one serotype
Slowish response
Have to balance inactivated/immunogenicity
Costly
No stocks
Low shelf life
Less useful in endemic areas because of the above factors.

Recombinant vaccines (See Jules Minke, Merial notes also)

There is an urgent need to develop recombinant vaccines for AHSV. At present none available commercially for AHSV but the success of these vaccines with West Nile Virus and BTV (which is closely related to AHSV) gives real grounds for optimism. **Hence trials in South Africa over the next two months with experimental Merial AHSV recombinant canarypox vaccine.**

There are many advantages of the recombinant canarypox vectored vaccines listed in Merial literature including:
i) safe and effective
ii) swift onset of immunity
iii) non replicating thus eliminating the risk of viral shedding and reversion to virulence
iv) provides broad protection – generates both a humoral and cell-mediated immune response
v) provides highly targeted immune response
vi) can be used as part of DIVA strategy

Questions
Tim Morris of the British Horseracing Authority asked Prof. Guthrie about quarantine and **vector protected conditions.** The precautions were rigorous e.g. sealed buildings, double door entry, forced ventilation systems with evaporative coolers with filters. Horses were allowed to exercise from 2 hours post sunrise to 2 hours pre sunset. Insecticides applied.

Dr Jules Minke, Merial, Lyons.
Adviser to The Horse Trust Working Party on AHS and WNV.

NEW VACCINE TECHNOLOGIES FOR AHS AND WNV: CURRENT STATUS AND FUTURE POSSIBILITIES

Dr Minke gave an extremely stimulating presentation about the new vaccine technologies, providing much optimism. Both he and Professor Guthrie, together with Dr Josie Traub-Dargatz of Colorado State University, provided the vital expertise and pragmatism needed in any discussion on West Nile Virus and African Horse Sickness Virus disease control policies.

I quote from the summary provided by Dr Minke on the new vaccine technologies. Below that I list some of the main points from his presentation. As has been said earlier, due to a combined effort by Dr Minke, Professor Guthrie and Professor James McClachlan of UC Davis, trials of a new recombinant vaccine for AHS, using the canarypox technology, are scheduled to take place in the next two months in South Africa. If successful this would have enormous implications for the control and eradication of the disease, not only in South Africa but in the EU, where up till now the modified live vaccines currently in use for AHSV are viewed with some concern.

Abstract from Newmarket Conference brochure
"One of the most significant changes in the field of veterinary medicine has been the introduction of several recombinant vaccines based on the canarypox (ALVAC) vector platform. Its high safety profile and ability to induce both humoral and cellular immune responses against the transgene without the need for adjuvants have been the driving forces for the generation of a number of commercial vaccines.

The Alvac technology platform facilitated the rapid generation of new constructs and as soon as the sequences of the protective genes of a micro organism are know, synthetic genes can be made and inserted into the ALVAC genome. This has proven to be an advantage in the case of emerging diseases such as WNV and Nipah virus and could be a major asset for the development of safe and efficacious vaccines for AHS. Notwithstanding the evident success of the polyvalent modified live vaccines against AHS in endemic areas, there are concerns about their use in epidemic situations because of their inherent biological safety risks. Therefore, their deployment in case of an outbreak in Europe would be viewed with concern by some veterinary authorities. The recent successful demonstration of efficacy of a canarypox vaccine expressing the VP2 and VP5 proteins of the related BTV confirms the viability of an ALVAC vaccination strategy for AHS. An additional advantage of the use of the ALVAC platform is that tests based upon the non- structural proteins will enable differentiation between naturally infected and vaccinated animals."

Supplementary notes from presentation

West Nile Virus vaccine
New vaccines are now in use in the USA e.g. Merial Recombitek (canarypox recombinant), and Intervet Chimerivax. Very safe. Used routinely as one of the core vaccines. Can be made commercially available under temporary licence in Europe.

African Horse Sickness Virus vaccine
Currently use modified live attenuated polyvalent vaccines in S Africa supplied by OBProducts. There is some nervousness in Europe about these vaccines – the perceived possible risks include reversion to virulence, possibility of establishing the vaccine virus in the midges (as polyvalent, many strains besides the current threat) and thus spreading other strains), risk in pregnant animals.
There are no inactivated vaccines produced now. "Equipest" produced by Merial for Spain in 1990s against AHSV 4 is no longer commercially available. There is a need for second generation inactivated vaccines because there are many disadvantages including:
i) at present inactivated vaccine production requires Level 3 (high level containment)
ii) there is limited production capacity
iii) no DIVA capability
iv) relatively slow onset of immunity
v) short shelf life
(For more details see Prof. Guthrie's presentation).

New vaccines
1. **Baculovirus platform** recombinant vaccine (see work by Roy and Sutton). Effective for bluetongue so have potential be effective against AHS.

2. Live vectored vaccines:
a) Replicative type have been used to combat rabies in foxes by air dropping food packs with vaccine inside. Also fowlpox.
b) Non replicative vector: canarypox virus (ALVAC). This **canarypox platform** used for Equine Influenza (Proteqflu, used in Australia 2008), West Nile Virus (Merial Recombitek, used in 2002 onwards USA), feline influenza. ALVAC platform vaccine has also been used to protect endangered species e.g. Santa Catalina Island Fox

This ALVAC canarypox platform has many advantages:
i) Rapid onset of immunity
ii) Efficacious
iii) Can be used as part of DIVA strategy – tests can differentiate between vaccinated and non vaccinated animals.
iv) Long lasting protection
v) No replication, shedding, and reversion to virulence
vi) Convenience – stable longer shelf life than inactivated vaccines
vii) Biosecurity level lower – no need to work with highly virulent organisms (just selected genes).

These new vaccines thus provide a platform of choice regarding emerging diseases enabling the rapid generation of new constructs (4 months) i.e. relatively quick production of vaccine possible from platform.
See Veterinary Record 2005 Edlund Toulemande.

Summary
1. New technology platforms are very promising e.g. canarypox, baculo, and chimera (Intervet Chimerivax for WNV and yellow fever)
2. Canarypox very promising/significant because of rapid immunity onset and DIVA technology e.g. sheep gain good immunity against BTV8.
3. Feasibility to be demonstrated for AHS in S Africa trials (Guthrie, Minke, McClachlan).

Professor Josie Traub-Dargatz, Pofessor of Equine Medicine at Colorado State University (CSU), College of Veterinary Medicine and Biomedical Sciences, Fort Collins, Colorado.

LIVING WITH WNV IN THE USA: EPIDEMIOLOGY, CLINICAL ASPECTS AND CONTROL.

Prof. Dargatz has worked at the CSU veterinary hospital since 1983, and her principal interests are in respiratory and gastrointestinal tract infectious diseases. However she is also closely interested in government networks for monitoring diseases and how information is communicated. She gave an extremely informative presentation on WNV, and the arrival and spread of the disease in the USA. Particularly valuable was the insight gained from the study, spearheaded by Professor Dargatz, into the outbreaks of West Nile Equine Encephalomyelitis (WNEE), caused by WNV, in Nebraska and Colorado. Her main conclusions are listed below.
Prof. Dargatz also provided additional supporting written material:
"Living with West Nile Virus in the USA: epidemiology, clinical aspects and prevention 199 through 2007" by J Traub-Dargatz and T Cordes.
I have also included supplementary notes which were taken by me at the presentation.

Conclusions
1. Vaccination is the single most effective means of preventing WNV disease.
2. WNV vaccine is now considered a core vaccine for horse owners in the USA.
3. Vets and owners play a critical role in disease detection i.e. in detection of the unusual.
4. There must be a plan in place for efficient communication of information to equine owners and vets e.g. website. (During the outbreaks the veterinary hospital received a huge number of phone calls, but was at the same time expected to be nursing cases of WNEE at the hospital).
5. There must be an effective laboratory infrastructure for rapid diagnosis with options for fast track development and approval of prevention and treatment options when emerging diseases occur.
6. **It is absolutely critical to have pharmaceutical companies poised ready to take action.**

Supplementary notes taken at the Conference.

WNV is an arbovirus now endemic in the USA. It is the causative agent of West Nile Equine Encephalomyelitis (WNEE). It is seasonal, occurring mainly in late summer and autumn.

Insect vector and host
Many varieties of birds act as the amplifying host for the virus. Mosquitoes are the insect vector, primarily Culex. The main cycle is thus between bird and insects. However, horses and humans can contract WNV if bitten by a mosquito, but they are regarded as "dead end" hosts because they do not have enough virus in their blood to act as source for mosquitoes.
NB
i) Person to person transmission possible? – blood transfusion, pregnant mother to foetus, breast milk of mother.
ii) Other animals besides horses may become infected: squirrels, alligators
iii) May be bird to bird infection without mosquito vector. This has only been demonstrated under laboratory conditions, so it has been suggested there may be a remote possibility in the wild e.g. perhaps birds eating birds, faecal material
iv) Cats and dogs are susceptible – may eat dead birds – rarely show signs of disease

Symptoms
Ataxia (unsteady), twitching muscles, altered mentation (ranging from hyperexcitability to extreme lethargy), weakness and unusual gait. May become recumbent (at greater risk of death). Can be confused clinically with other neurological diseases. Relatively high mortality rates without vaccination – 30%, but 40% in some areas. Those that recover may have residual problems, which may be long lasting.

Diagnostic tests
1. Blood: MAC ELISA (antibodies) on serum (no confusion with vaccination). Illustrates recent exposure. This test available in many veterinary diagnostic laboratories.
2. Blood: PRNT or Microtiter for antibodies. Different from MAC ELISA. Diasadvantage: can confuse with vaccination, and also "one gets a positive result to wild virus".
3. Tissue tests to identify virus in tissue. NB precaution.

History
1999 Unusually large number of bird deaths in USA. John Andresen (New York state vet) was the first to spot higher than usual cluster of horses with neurological problems. WNEE can be difficult on clinical signs alone to differentiate from other neurological diseases, so if disease is not endemic i.e. not expected (this was the case before 1999) this can cause problems re diagnosis. The message is preparedness, with dissemination of up to date knowledge, plus vigilance, regarding emerging diseases.
Equine cases
1999 25
2000 60
2001 738
2002 15, 259 (actual figure probably 3 times this)
2003 5000
2004 1000
2007 468
Further details available at www.aphis.usda.gov/vs/nahss/qeuin/wnv
With use of vaccination in 2002 and 3 there was a dramatic drop in horse and human cases. 2006 equine population in USA c 6 million, and 4.1 million doses of vaccine manufactured **NB**. **Equine industry and pharmaceutical companies responded quickly,** meaning that there was a rapid and widespread response to challenges posed by the disease.

Vaccines
Only 3 are licensed and commercially available for use in horses in US:
1. **Killed**/inactivated vaccine with adjuvant 2001 granted a conditional licence. In February 2002 a full licence was granted. Licensed for prevention of viraemia (i.e virus development in blood) but not disease.

2. 2003 January Merial Canarypox **recombinant** vaccine with adjuvant in which WNV protective proteins are expressed by canarypox vector. 2 doses are given, 3 weeks apart, then yearly after that. Licensed for prevention of viraemia.

3. The **chimera** vaccine consisting of "protective proteins of WNV spliced into an attenuated human yellow fever virus (WN-YF)" Intervet vaccine "Chimerivax". Single dose and annual booster.

NB In 2005 DNA vaccine was licensed but it is not yet commercially available.

Efficacy
Very efficacious. Vaccines never 100%.
No safety issues to date. Some initial concern re pregnant mare e.g. loss of pregnancy, but this was not substantiated.
Most cases of WNV are in unvaccinated horses.
See www.aaep.org

Nebraska and Colorado Study of WNV Outbreaks

This was spearheaded by Professor Dargatz as it was obvious the government was not in a position to do this as it was overwhelmed. Also cost was a factor. Veterinary students at CSU were employed as this was less expensive, but also beneficial to their studies. They conducted telephone surveys, and contacted 536 individuals. There was a 92% participation rate. NB A clear plan for collecting data is essential, so for countries not yet affected, but at risk, it is vital that a system is already in place before disease strikes.

Information gained/lessons learned (see also Conclusions on first page)
Relatively high fatality 30%
Of those with clinical signs majority were not vaccinated or only partially vaccinated.
Fatalities more common in older horses.
If disease at stage where animal recumbent, then more likely to be fatal.
Other animals infected e.g. mules.
Residual problems of disease – some animals who survive never return to normal.
Vaccination the most effective means of preventing WNV
Better to prevent than to wait until disease arrives.

Regional occurrence
First season just a few cases
Second season – amplification explosion.
Then reduction to maintenance levels.

Decline in disease due to:
Widespread, regular use of vaccination.
Preventive management and control – mosquito control – insecticides, repellents, vector-resistant housing.
Natural immunity because of exposure.
Stagnant water removal, dung and tall weeds.

Costs
In Nebraska and Colorado the cost of treatment, prevention and lost use was in the order of 4 million dollars. See avma and usda links below for further details.

Further information
This can be obtained through the link
http://www.avma.org/onlnews/javma/jun03/030615l.asp

"In 2002, 378 and 1,100 equine cases of West Nile infections were confirmed in Colorado and Nebraska, respectively….. Of the cases studied, 8 percent were mild, 58 percent were moderate, and 34 percent were severe….. Approximately 47 percent of equids with WNV in Colorado and Nebraska received at least one WNV vaccination in 2002, according to the owner survey".

Regarding costs:
*"… researchers estimate that prevention **costs of WNV vaccination** likely exceeded a combined $2.75 million in Colorado and Nebraska in 2002…"*
*"Veterinarians then estimated the **cost of treatment** for each category and determined that it costs approximately $200 to treat animals with mild disease, $400 for those with moderate disease, and $250 for equids with severe disease. Severe cases cost less, on average, because many severely infected equids were likely euthanatized before incurring high treatment expenses…."*

The entire report on costs to the equine industry in Colorado and Nebraska can be viewed and/or downloaded at www.aphis.usda.gov/vs/ceah/cahm/Equine/wnv-info-sheet.pdf (PDF).

Professor Alan Guthrie, Director of Equine Research Centre, Faculty of Veterinary Science, University of Pretoria, South Africa, and served on OIE working groups for AHS, EI, WNV. Also AHSWG adviser.

WEST NILE VIRUS IN SOUTH AFRICA: COMPARISONS AND CONTRASTS WITH THE USA

Disease of humans and animals with an **insect vector** – the Culex mosquito e.g. Culex univittatus. **Birds are reservoir hosts.** Humans and horses get bitten and can become infected but they are regarded as dead end hosts.

1937 Ugandan woman discovered with West Nile Fever in West Nile province.
1974, 1986-7 virus isolated in humans in South Africa.
Extensive epidemics recently in Mediterranean and E Europe, North Africa, Asia and more recently North America. With climate change possible spread further north from the Mediterranean.

Neurological disease with 40 % fatality. However in S Africa an avirulent or mildly pathogenic virus strain (lineage 2 virus) in horses; in humans it can cause fever plus muscle/joint pain in humans – death very rare.

South Africa

1. WNV is endemic in S Africa with very widespread transmission of the virus.

2. Distribution is linked to vector distribution.

3. Rarely causes neurological disease in horses. S Africa has **avirulent or mildly pathogenic virus strains (Lineage 2 viruses).** This is in **contrast to the USA (Lineage 1 viruses), which result in major neurological disease.** Lineage 1 also in Europe and Asia epidemics. A serological survey of all thoroughbreds by SNT (Serum Neutralisation Test) showed that 75% adult mares were seropositive but had no clinical signs. (Also maternal antibody detected in colostrum).

4. Experimental infection with Lineage 2 virus led to nothing significant regarding disease – no observed clinical signs or viraemia.

5. Horses and humans are dead end hosts and therefore the disease should not be a barrier to International Trade.

Summary (Quote from Prof. Guthrie's Conference paper):
"Asymptomatic or subclinical WNV infection of horses (and humans) is common throughout much of South Africa, and the experimental infection of horses with a S. African Lineage 2 isolate of WNV resulted in neither clinical disease nor viraemia. The difference between the clinical signs observed following infection with WNV in horses in South Africa and those infected with WNV the USA is due to difference in pathogenicity between the virus strains that circulate in the two regions."
(See Guthrie AJ, Howell PG, Gardner I, Swanepoel R, Nurton JP, Harper CK, Pardini AD, Groenewald D, Visagé CW, Hedges J, Balasuriya UBR, Cornel AJ, MacLachlan NJ: 2003. West Nile virus infection of Thoroughbred horses in South Africa (2000 - 20001). Equine Veterinary Journal 35 (6), pp 601-605).

Matthew Hartley
Deputy Head of Exotic Notifiable Diseases, Defra.
Member of AHS Working Group.

Mr Hartley provided very useful information on both WNV and AHS: occurrence, symptoms, etc. which was supported by a very detailed article in the Conference brochure However, what was of particular significance was his information on current disease control policies in the UK, and possible future policy. He emphasised that policy was based on EU legislation, (1992/35 for AHS) which was now considered to be in urgent need of revision, in the light of advances in vaccine technology and diagnostic tests, increased global movements of livestock and people, climate change, potential for novel vectors (as illustrated by the BTV8 outbreak in northern Europe in 2006). We were urged to lobby the EU to review policy, as combined pressure from Defra, the equine industry and other stakeholders would be far more effective than Defra alone.

What however was not mentioned was the power given to the Secretary of State in the Animal Health Act 2002, to slaughter **any animal** (including dogs, cats, birds etc) he sees fit. This includes not only animals that are confirmed by laboratory diagnosis to be infected, but also those suspected of being infected (but not confirmed positive by testing) **and crucially those not infected.** This then opens the gates to firebreak killing of healthy animals to create a barrier around an infected area. This mass killing of healthy stock took place in FMD 2001, and as Elliot Morley acknowledged in his evidence to the EFRA Select Committee on 6 November 2001 when he said "We do not have powers for a firebreak cull", the legal powers for this did not in fact exist in FMD 2001. They do now. I mentioned this to the Chairman of the TBA after the meeting.

It was evident that there were those in the audience who feared that Defra might attempt to behave in similar fashion to FMD 2001 (and 2007) and slaughter on suspicion. We were not totally reassured after close questioning of Mr Hartley that this would not happen if an outbreak of AHS were to occur in the near future. Neither was a remark by Dr Oura reassuring when I understood him to indicate that if a few more horses had to be killed to prevent the disease spreading then so be it. The impression therefore was that, as things stood under current legislation, there may not necessarily just be slaughter of infected horses. For further details of the Defra control strategy see later in text under individual disease headings.

What was particularly encouraging was the initiative taken by the equine industry to set up an Animal Horse Sickness Working Group in 2007 to tackle these issues, working in partnership with government (see Brigadier Jepson's comments). Defra's presence at, and contribution to the Conference was particularly significant. Of course the setting up of the Newmarket Conference itself was another example of the tremendous cooperation and determination between a wide spectrum of interested parties to:
i) raise awareness/educate, by bringing together world experts with first hand knowledge of WNV and AHS and its control in order to provide us with accurate, up to date information at the cutting edge of science;
ii) to move the debate forward and to examine ideas for future strategy/policy, with a view to lobbying the EU. See also the closing speech by Kirsten Rausing, Chair of the TBA.

Mr Hartley mentioned the development of the AHS Strategy which would be a "model for the future" and cost sharing with industry. He also mentioned that government would set up a

Core Stakeholder Group on AHS to ensure consultation and "working in partnership". There understandably is a degree of scepticism about how successful this will be as experiences from some in the FMD Stakeholder Group regarding the degree/type of consultation have been worrying. Furthermore, **Mr Hartley made the point that Defra's resources and priorities had "to be factored in" to any strategy**, and that says it all. There is for example no equine surveillance in horses at present for as this is considered be too costly. See http://www.defra.gov.uk/animalh/diseases/vetsurveillance/profiles/west_nile-virus-full.pdf Furthermore, I understand that horse owners who submit samples for testing for WNV to the VLA have to pay for the cost of the tests.

Below is a brief summary of some of the points covered on the individual diseases. Quotes from the article by Matthew Hartley in the Conference brochure are indicated in italics.

West Nile Virus
Spread of disease
1. The mosquito vectors are primarily the Culex species which are known to occur in the UK.
2.The nearest outbreaks to the UK were in Italy (1998) and southern France (2000).
3. Mr Hartley stated that *"migrating birds are the most likely mechanism for the infection being introduced into the UK"*. This should be qualified as the introduction of WNV into the USA was apparently not likely to have been due to migratory birds. The US virus was very closely related to a lineage 1 strain found in Israel in 1998, but the migration routes of birds do not correlate with this. Some explanations suggest an imported (illegally) infected bird, or the "import" of an infected mosquito of the jet setting variety. Thus the UK could be at the same level of risk as the USA in 1999.
4. Regarding the bird hosts, some species are particularly susceptible e.g. the crow family, and can be subject to mass "die-offs".
5. No active virus has apparently been found in UK birds (Phipps et al Veterinary Record (2008), 162, 413-415), but evidence of antibodies has been found.

Action taken by Defra
1. VLA surveys on deaths in wild birds since 2001. No evidence of mass die-offs. See 5. above.
2. It is now possible to send samples to the VLA for testing via the local Animal Health Office. There is a charge for this service.
3. Research has been commissioned for a large scale study of mosquitoes in the UK.
4. The health Protection Agency (HPA) tests for the presence of WNV in mosquitoes.
5. The Health and Safety Executive (HSE) have carried out a study of the approved pesticides for use in mosquito control.

Disease prevention
"Prevention and control, in the event of clinical disease being identified in the UK, is primarily to reduce the numbers of vectors and prevent contact with them:
i) Vector control – eliminate breeding sites of mosquitoes (stagnant water, rainbutts etc) with possible use of insecticides
ii) Vector avoidance – for animals keep them away from vector sites, apply insect repellent, house in insect proofed accommodation when mosquitoes are active".

Control strategy
1. Statutory notification of suspected disease plus veterinary investigation.
2. Restrictions on infected premises

3. Local and national risk assessments.
4. Dept of Health contingency plan followed and an infection control team (ICT) set up locally.
5. Overall control at national level undertaken by Defra and DoH. The ICT will advise on further local actions e.g. vector control, extra surveillance, publicity.
6. **There is no requirement for statutory slaughter of horses.** If horses slaughtered there is no provision for compensation.

For further information see:
http://www.defra.gov.uk/animalh/diseases/notifiable/westnilevirus/index.htm

http://www.defra.gov.uk/animalh/diseases/vetsurveillance/profiles/west_nile-virus-full.pdf

African Horse Sickness
Risk of introduction/spread
Closely related to bluetongue, so sudden spread of bluetongue to northern Europe has led to reevaluation of potential for introduction – BTV8 does not require presence of C. Imicola in northern Europe. Local species of midge have become novel vectors. These are obviously adapted to local climatic conditions so climate change e.g. rising temperatures not necessary to maintain the vector.

Legislation
"*Council Directive 92/35 provides for compulsory notification, and the setting up of a protection zone of at least 100 kms radius around an infected premises. This, together with a surveillance zone of at least a further 40 kms, would have to remain in force for at least 12 months. AHS is included in The specified Disease (Notification and Slaughter) Order 1992 to implement the slaughter requirements of the EU council Directive 92/35/EEC which lays down control rules and measures to combat AHS. Imported horses from at-risk countries outside the European Union are routinely tested for AHS.*
The severity of disease and the controls to monitor and restrict movement of horses could significantly affect the equine industry in the UK".

When a suspect case is reported the horse(s) is brought indoors, isolated, tested. Insect eradication in neighbourhood.
If sample positive, horse killed. Horse may be euthanised at any stage on welfare grounds at veterinary discretion. (NB Animal Health Act gives additional powers of slaughter).
Control zone set up of 20 km radius around IP.
Protection zone of 100 km
Surveillance zone of 150 km radius around infected premises. Varies according to epidemiological and geographical factors.
Tracing investigations are carried out.
Equine surveillance in surrounding areas plus surveillance of midge vector.
Professor Mellor also warned that some Culicoides activity during the day, so just bringing in horses from dusk to dawn may not be good enough.

Vaccination
Mr Hartley stated that EU legislation "requires vaccination" in an outbreak but "there are no licensed vaccines" and there is a perceived risk that the multivalent live vaccines may

introduce other serotypes, but see comments by Prof. Guthrie regarding the value of these vaccines in the face of an outbreak.

According to Mr Hartley:
"The last outbreak of AHS in Europe, in Spain and Portugal in the early 1990s was controlled after an intensive local vaccination programme was instigated. There are 9 different serotypes of AHS and there are no vaccines available in Europe against any of them. Using currently available vaccination approaches, each of the 9 serotypes would require its own vaccine". (This refers to inactivated vaccines, none of which are currently available in Europe. However, the advent of new vaccine technologies e.g. using the canarypox platform, may well transform the situation in the near future). "*The only vaccines in regular use overseas in AHS endemic areas are polyvalent modified live vaccines which are not suitable for use in free areas such as Europe that would try to eradicate the disease – and might also result in introduction of new serotypes."*
NB AHS is not endemic in all parts of South Africa e.g. an area around Cape Town can export to the EU under a regionalisation agreement and specified quarantine arrangements. http://www.nda.agric.za/vetweb/Animal%20Disease/AHS_Image40.htm

For further details of AHS and vaccination in S Africa visit the AHS Trust website:
http://www.africanhorsesickness.co.za/default.asp

Control strategy
"Defra is participating in an equine industry led working group to develop a UK AHS Strategy. The strategy takes forward current legislation and describes how it would be implemented in partnership should AHS arrive in the UK. This is currently in the drafting phases and will be available for consultation later this year". See earlier references to this by Brigadier Jepson.

We were advised that the strategy was in two parts:
i) current disease control strategy based on current legislation
ii) an improved updated control strategy drawn up after consultation with industry, scientific experts, government and other stakeholders. This to be presented to the EU to lobby for change of Directive 92/35.

"The generic contingency plans for notifiable disease outbreaks in the UK have been revised to include the outcomes of the consultation on the Specified Type Equine Exotic Diseases (STEED) plan."

Questions:
Imports of horses
Mr Hartley was asked for details of import figures and source countries and questioned about the import checks. He was unable to give details, but the questioner himself made it clear that the UK was the biggest importer in the EU of horses from Asia, the second biggest from South America, and the third biggest importer of horses from Africa, and asked about import testing and surveillance as there were obvious risks. It seems that there is no import testing for emerging diseases and that the horses undergo clinical inspection only. There is also no equine surveillance. Mr Hartley advised the meeting that there is apparently no legal requirement for the location of horses to be registered, so this would pose problems for surveillance. (However, as one of Defra's own reports makes clear the critical factor is cost – see Defra report links in WNV section above).

The questioner continued to push on the issue of stopovers, where horses may be exported from a not at risk country or disease free zone within that country, and yet on stopovers these animals may be taken off the aeroplane. If this is in an affected country and precautions are not adequate then there is an obvious risk. It would appear that the details of stopovers (location, precautions, inspections) may not always be recorded. Also as there are apparently no specific checks/blood tests routinely carried out on import for these emerging diseases then this is cause for further concern. Certainly Mr Hartley was not able to provide reassurance on this. This would obviously be a point worth investigating further.

Kirsten Rausing, Chair of The Thoroughbred Breeders' Association

Kirsten Rausing brought the Conference to a close urging conference-goers to spread the word and to take urgent action. The industry could not afford to sit back. She said that the EU had to be approached to amend the "obsolete" 93/35 Directive. She advised us that the Commissioners are far more likely to take notice if the industry acted on a united front, and suggested that one such umbrella organisation could be the European Federation of Horse Breeders Associations, based in Switzerland (Chairman Hans Peter Meier. Vice Chairman Richard Jones, UK). The EU is far more likely to act if it has representations from an organization that it recognizes as supranational i.e. represented all member states. What are needed are written and physical representations to the Commissioners regarding the amendment of the Directive. (Jules Minke comment: possibly a consortium of the equine industry, EU, and the pharmaceutical industries could provide the mechanism for change).

Finally she reminded us that it was a case of when, not if, these diseases arrive, and action was needed now, despite the fact that the UK may be at apparently lower risk than elsewhere in Europe at present, and that other diseases such as bluetongue were more pressing.

My comment: the risk is very real and should not be ignored. We are at risk of unforeseen "parachuting in" of disease (in addition to the "expected" northward spread of some diseases) with the very real possibility of disease being maintained and spread by novel vectors. The consequences for the equine industry could be disastrous if we are not adequately prepared. There is the expertise/technology available and there is obviously tremendous commitment and determination within the equine industry to put a workable disease control strategy based on sound, up to date science, in place. What is needed is the political will within the EU not only to acknowledge the real threats posed by the emerging diseases, but to take swift action to employ the very best of its scientific expertise in order to bring its disease control legislation into the 21st century, with preventive vaccination at the fore. As the EC document "A new Animal Health Strategy for the EU (2007-2013)EU" states "Prevention is better than cure". Enough of the words, now we need the action.