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Proceedings & Abstracts

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Invited Speakers Proceedings
The disappearance of terrestrial rabies in large parts of Europe has been achieved through stepwise intensive efforts to control and eliminate the disease in both its canine and wildlife reservoir hosts. Europe-wide elimination was accomplished by strict implementation of hygienic measures, registration and mass vaccination of dogs in the 1970s (Müller et al., 2012). In contrast, the emergence of fox rabies in the 1940s posed a new challenge and required fundamental changes in rabies control policies. Early attempts to control fox rabies that aimed at a drastic decimation of the fox population to interrupt the infectious cycle failed. As a result, the number of reported rabies cases steadily increased Europe-wide until the 1980s, when fox rabies reached its western and south-easternmost expansion in Europe (Müller et al., 2012), hence emphasizing the need for alternative rabies control methods. Despite perceived challenges to controlling an infectious disease in wildlife, oral rabies vaccination (ORV) of foxes has proven a remarkably successful tool and a prime example of a sophisticated strategy to eliminate disease from wildlife reservoirs (Rupprecht et al., 2008, Freuling et al., 2013). The first ORV field trial successfully conducted in Switzerland in 1978 (Steck et al., 1982) was a breakthrough in wildlife rabies control and triggered further field trials in other European countries in subsequent years. Whilst at the very beginning appropriate tools had still to be developed from scratch, pioneering developments including efficacious and safe oral rabies virus vaccines, adequate fox-adapted ORV vaccination strategies, machine-made baits, and automated computer supported aerial bait distribution were technical milestones on the road to success in subsequent decades (Müller et al., 2012).

Since 1989, the EU has been co-financing costs for disease eradication in member states and neighbouring none-EU countries. This co-financing policy has been a strong incentive advancing the EU to the driving force for fox rabies control in Europe (Demetriou and Moynagh, 2011). As a consequence, during the past three decades, ORV programmes had been implemented in 24 European countries. The maximum total area ever covered at least once with vaccine baits in Europe between 1978 and 2012 encompassed almost 2.6 million km². A total of 10 different commercial modified live rabies virus vaccines, attenuated and recombinant, were used in ORV campaigns (Freuling et al., 2013). As a result, the number of animal rabies cases in Europe decreased from 18,588 cases in 1980 to 6130 cases in 2012 with fox-mediated rabies having virtually disappeared from vast areas of Western and Central Europe. Despite the tremendous success achieved, European countries had to face several setbacks resulting in delay in rabies elimination at the regional level. Reasons for setbacks were multifaceted and lessons had repeatedly to be learned (Stöhr and Meslin, 1996; Müller...
et al., 2012). A recent comparative analysis of implemented ORV programmes between 1978 and 2010 revealed that rabies control took between 5 to 26 years depending upon the country. The proportion of land area ever affected by rabies and an index capturing the size and overlap of successive ORV campaigns (Selhorst et al., 2004) were identified as factors having statistically significant effects on the number of campaigns required to both control and eliminate rabies. Repeat comprehensive campaigns that are wholly overlapping much more rapidly eliminate infection and are less costly in the long-term (Selhorst et al., 2004, Freuling et al., 2013). While it needs comparatively less money to reduce the number of rabies cases by 50% or even 90% using ORV, disproportionally greater effort is required in the final phase of an ORV programme, with a median of 11 additional campaigns required to eliminate disease once incidence has been reduced by 90% (Freuling et al., 2013). The impressive decline in rabies incidence after a few campaigns often resulted in overoptimistic interpretation of the vaccination success; however, spatial analysis shows that the proportion of land area affected by rabies still remains the same size and only has a delayed reduction until elimination is achieved (Müller, unpublished). Hence, if successive ORV campaigns span the entire affected area, rabies will be eliminated more rapidly than if campaigns are implemented in a less comprehensive manner, therefore reducing ORV expenditure in the longer-term (Freuling et al., 2013). These findings should help improve the planning and implementation of ORV programmes, and facilitate future decision-making by veterinary authorities and policy-makers.

Currently in Western Europe, all measures are directed towards the maintenance of a rabies free status by avoiding reintroduction of the disease by measures which include implementation of the pet travel scheme, risk-based surveillance and establishment of cordons sanitaire along borders to rabies endemic regions. Despite success in Western and Central Europe, however, complete elimination of rabies in meso-carnivores in Europe has not been achieved, and is far from becoming a reality (Rupprecht et al., 2008). Although the EU has spent more than 75 Mio € to support rabies elimination in the Turkey, Western Balkan region, Kaliningrad and north-eastern neighbouring third countries (Demetriou and Moynagh, 2011), the fight against wildlife-mediated rabies in Europe still needs to be won. On a global scale, considering the large areas that need to be treated to eliminate wildlife rabies in Europe, North America, and other parts of the world, economic costs are an important issue, especially for less developed countries, requiring novel cost-effective vaccination strategies to be applied under different ecological conditions. Furthermore, whilst ORV of foxes and raccoon dogs seems no problem, ORV of multiple species wildlife reservoir hosts, e.g. raccoon, skunks, mongoose in North America still remains a major challenge (Rupprecht et al., 2008). The fact that those species have also been introduced as alien species to Europe highlights the need of combined research efforts to find acceptable solutions to related issues such as the development of an all-purpose oral vaccines for multiple species as well as optimal baiting strategies in the presence of multiple reservoirs (Stöhr and Meslin, 1996).


New human emerging infectious diseases originate for 75 % from an animal source, either from domesticated animals or wild animals. For that reason the World Health Organisation introduced the ‘One World One Health’ concept. This concept is embraced by politicians, physicians and veterinarians. Primarily the One Health concept is focused on livestock animals, but for companion animals and exotic animals this concept is important too. Especially the enormous variety of animals still existing in the wild and thus their ambassadors exposed in zoos put a epidemiological challenge to overlook the zoonotic impact of microbial agents carried by these numerous possible hosts.

Exposure to zoonotic diseases is one of the most important health risks for zoo veterinarians, zookeepers and to a certain extent to zoo visitors. The WHO has defined zoonoses as ‘those diseases and infections which are naturally transmitted between vertebrate animals and man’. There are about 1415 infectious agents causing diseases in humans, out of which 868 (61%) are known to be zoonotic in nature. It is important to notice that more than 70% of the emerging zoonotic diseases have wild animals as reservoir hosts and many are also seen in our domesticated animals with again an original source in wildlife. Recent outbreaks of Avian Influenza and SARS originated from wild animals, but Q-fever (Coxiella burnetti) outbreaks or Chlamydophilla infections resulting in psittacosis generally come from domesticated animals.

Wild animals act as reservoirs for several zoonotic diseases which could be bacterial, rickettsial, chlamydial, mycotic, viral, and parasitic in their etiology. The major zoonotic diseases that are transmitted from wild animals to humans include rabies, anthrax, leptospirosis, Q-fever, psittacosis, hendra virus, nipah virus, Ebola, herpes B encephalitis, toxoplasmosis, tuberculosis, Campylobacteriosis, Colibacillosis, Lyme disease, Plague, Salmonellosis, Tetanus, Typhus, Tularemia, etc.

Since ancient times, zoonotic diseases like plague, rabies and tuberculosis have devastated mankind and interestingly all of them have wild animals as reservoirs. The symptoms of zoonotic diseases in humans may range from a self-limiting disease to long term illnesses or may cause a high degree of mortality as in the case of Ebola. Although zoonotic diseases have been viewed as serious threat to public health, they are also a major concern with respect to endangered species of wild animals which are on the verge of their extinction.

Each disease has its own mode of transmission. Oral, respiratory, direct contact, vector-born, blood, vertical route, multiple routes, etc. The diseases transmitted from animals to humans are scientifically termed as anthropozoonotic diseases, like anthrax, psittacosis and rabies.
The diseases transmitted from humans to animals are termed as zooanthroponotic, like human tuberculosis, measles, giardiasis etc.

In European zoos every now and than outbreaks of Salmonellosis, Colibacillos, Psittacosis, Campylobacteriosis etc. occur, but are normally easily managed and controlled. Tuberculosis in Asian Elephants on the other hand still causes debate and problems. In most of these cases Mycobacterium tuberculosis and not Mycobacterium bovis is encountered, coming from a human source. Elephants transmitting the disease to zookeepers pose a major risk and measurements to avoid this in biosecurity and surveillance protocols in zoos are often lacking. The main problem is that there is not a valid definitive ante-mortem test to diagnose tuberculosis in an early stage and tuberculosis is often a finding seen at pathology and thus often measurements are taken too late to avoid transmission to humans. In Pinnipeds Mycobacterium pinnipedia had a great impact in the last decade and in some cases also lead to transmission of the disease to zookeepers. Cowpox infections in cheetahs and elephant pox in elephants have been encountered regularly and Toxoplasmosis and Yersiniosis may occur in a variety of animals and may be a threat to animal keepers and veterinarians.

Veterinarians and zoo staff come in direct and indirect contact with wild animals or their excrements during clinical examination, blood collection, post mortem examinations, cleaning procedures etc. These interventions leave them exposed to the risk of contracting zoonotic diseases. Salmonellosis by handling reptiles such as snakes is often seen and in the case of contact of reptiles with the public, zoo staff should be aware that esp. young kids may be exposed to Salmonella spp. Hand washing facilities should be available or even better abolition of direct contact with reptiles. Shigellosis from great apes (Orang Utans) infected zookeepers in Vienna and was a threat to zookeepers in Amsterdam when chimpanzees suffered from the disease.

Due to proper preventative veterinary care zoonotic outbreaks in the major zoos in Europe do not occur frequently. The important zoonotic diseases need to be notified and there is a clear picture what’s going on in European, Australian and Northern American zoos. The major zoos in Europe are united in EAZA (European Association of Zoos and Aquaria) and most veterinarians are member of EAZWV (European Association of Zoo and Wildlife veterinarians. In Northern America the equivalents are AZA and AAZV (American Association of Zoo Veterinarians). EAZWV has an Infectious Diseases Working Group (IDWG) who produced four editions of the “Transmissible Diseases Handbook” in which most zoonotic diseases are described in fact sheets. The fact sheets give a brief explanation of the disease, the agent involved, whether the disease is notifiable and/or zoonotic, the impact of the disease, who to call, how to diagnose, which national laboratory to consult and all relevant European legislation regarding the keeping of wild animals in captivity. It’s a tool for zoo veterinarians on how to react in case of a suspicion of an emerging disease. It does not replace proper handbooks but serves as a tool in cases of outbreaks of infectious diseases in a zoo. Since 2012 also AAZV produced their first edition with the same content, but more directed to the American situation. Both Handbooks are being reviewed in 2013.
SCHMALLENBERG VIRUS INFECTION: AN EMERGING VECTOR-BORNE DISEASE IN EUROPE

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Schmallenberg virus is the first orthobunyavirus of the Simbu serogroup detected in Europe. It was identified by metagenomic analysis in Germany and the Netherlands in autumn 2011. According to the current state of knowledge, ruminants and new-world camelids are susceptible to infection with Schmallenberg virus. There is serological evidence for infection of wildlife ruminants (roe deer, fallow deer, red deer, mouflon).

The genome of orthobunyaviruses consists of three single stranded RNA molecules (segments), designated as S(mall), M(edium) and L(arge). Exchange of segments by reassortment is possible between members of a group. As the M segment codes for the viral glycoproteins that induce neutralising antibodies in infected hosts, there is selection pressure leading to frequent reassortment events involving this segment. Molecular analyses have shown that Douglas virus, which was first isolated in Australia, Sathuperi and Shamonda virus represent close relatives of SBV, but can clearly be distinguished. The Simbu serogroup comprises of more than 24 viruses, most of which have been detected in ruminants. A number of these agents, e.g. Akabane and Aino virus, can cause disease in these animals, while human infections with orthobunyaviruses are rare and have only been described for two viruses (Oropouche and Iquitos virus). There is no evidence that humans are susceptible to SBV infection.

Adult animals may develop mild disease, if any. However, transplacental infection occurs frequently and may lead to severe congenital malformations such as arthrogryposis, malformation of the vertebral column (kyphosis, lordosis, scoliosis, torticollis) and of the scull (macrocephaly, brachygnathia inferior) as well as variable malformations of the brain (hydranencephaly, porencephaly, cerebellar hypoplasia, hypoplasia of the brain stem) and of the spinal cord in lambs, kids and calves.

Already in 2011, the infection had spread rapidly over large parts of North-Western Europe. In 2012, Schmallenberg virus infections re-emerged, although a substantial proportion of the susceptible animal population appeared to be immune upon natural infection, and spread to several countries surrounding the area that had originally been affected in 2011. Acute infections of adult ruminants or malformed SBV-positive offspring were detected in more than 5000 farms at least in Austria, Belgium, the Czech Republic, Denmark, Estonia, Finland, France, Ireland, Germany, Italy, Luxembourg, Norway, Poland, Spain, Sweden, Switzerland, the Netherlands and the United Kingdom. Suspect cases were also reported from Hungary and Slovenia.
Serological studies were conducted in Belgium, The Netherlands, Germany, France, Austria and Switzerland. In Belgium, serum samples obtained from cattle during spring 2010 and in spring 2011 were seronegative for SBV. The testing of sera from cattle randomly sampled on 209 farms in April 2012 revealed an apparent seroprevalence among adult cows of 90.8% (95 percent CI 88.3–93.2%). In the Netherlands, the estimated seroprevalence in cattle was 72.5 percent (95% CI 69.7–75.1%). in winter 2012. High (70–100%) within-herd seroprevalences were observed in two SBV-infected dairy herds and two affected sheep flocks. Similar within-herd prevalences were observed in cattle herds and sheep flocks in Germany. The seroprevalence observed in cattle during winter 2012 in Germany correlated with the spatial distribution of virus detection in malformed lambs and calves. In the core region of the epidemic in Germany, up to 98% of the tested animals had seroconverted, while the numbers of seropositive animals decline with the spatial gradient. Examination of sera collected before 2011 from susceptible species provided no evidence that SBV had been present in the affected area before 2011.

The virus is apparently transmitted by arthropod vectors. Biting midges (Culicoides spp.) seem to play a crucial role in transmission. Interestingly, there is evidence for SBV transmission during winter 2012/2013 at a time, when the population density of the putative vectors was low. Available evidence obtained from limited experimental infections suggests that direct horizontal transmission of SBV infection by contact does not occur. Oro-nasal inoculation of calves did not lead to infection and in several animal trials, contact animals remained seronegative. It is obvious, however, that SBV can be vertically transmitted from the dam to its offspring by transplacental infection.

SBV genome can be most readily detected by PCR. SBV-specific antibodies can be demonstrated in serum samples by virus neutralisation test, indirect immunofluorescent antibody test or ELISA. Acute SBV infection, which is expected to occur primarily during the vector active period can be detected by PCR in blood or serum samples. It is important to note that the extremely short viraemia observed in SBV infection (5 days maximum) requires that the affected animals are sampled when they show clinical signs, preferably during the very short phase when the animals develop fever. PCR can also be used to demonstrate SBV in malformed neonates, preferably by testing brain samples from at least two different parts of the organ (cerebrum and cerebellum). While SBV genome is regularly detectable by PCR in the brain and in other tissues from lambs with SBV-associated malformations, it is less likely to find SBV genome in calves with typical malformations. A negative PCR result in a malformed calf does therefore not rule out SBV as the cause of the malformation. In these cases, antibody detection in pre-colostral blood samples or body liquids, e.g. obtained by cardial puncture in stillborn animals, may be performed.

Although the losses caused by SBV infection were limited, the emergence of this new disease caused substantial trade disruptions.
This presentation will discuss lessons learnt from previous control / eradication campaigns, as well as future challenges to controlling viral diseases of animals. The major challenges faced in both controlling and eradicating animal viruses include the complex and rapidly evolving nature of viruses, the complexity of the immune response to viruses, the lack of effective and available vaccines, the presence of insect and wildlife reservoirs and the rapid and uncontrolled spread of viruses within developing countries. How these factors continue to affect the successful control / prevention / eradication of some of the most globally important veterinary viruses will be discussed. Examples will be given from viruses that have been successfully controlled and eradicated [Rinderpest (RPV) and bluetongue virus serotype 8 (BTV-8)], viruses that are currently expanding their geographical boundaries and are proving difficult to control [African swine fever virus (ASFV), Peste des Petits Ruminants virus (PPRV) and African horse sickness virus (AHSV)] and viruses that appear ‘out-of-the-blue’ like Schmallenberg virus (SBV) and some strains of bluetongue virus (BTV).

One virus that we can certainly learn many lessons from re: control / eradication is Rinderpest virus (RPV). In 2011 the OIE and FAO announced the global eradication of RPV, the first and only veterinary virus to be eradicated from the world through human intervention. Rinderpest was probably the most devastating disease of cattle in history and was circulating in Pakistan as recently as the 1994. The Global Rinderpest Eradication Programme (GREP) was initiated in 1994, the last confirmed case of Rinderpest was confirmed in Kenya in 2001 and the vaccination programme was stopped in 2006. The key to the successful eradication programme was the simple nature of the virus (one serotype), the presence of a thermostable vaccine that gave long-lasting protection, the lack of a significant wildlife reservoir, the presence of sensitive and specific diagnostic tests and international support for the eradication campaign with sufficient funding.

One question currently being asked is what should be the next veterinary virus on the list for eradication? PPRV, the cousin of RPV that affects small ruminants (sheep and goats) across much of Asia and Africa, must be a strong candidate. Like RPV, PPRV occurs as a single serotype. An efficient, long-lasting, single-use live vaccine is available that protects against all the lineages of PPRV, however this vaccine needs to be made thermostable. More studies across wider geographical areas are required, however initial data from wildlife (buffalo and gazelle) and cattle in Tanzania shows no evidence for the involvement of wildlife as
significant reservoirs of infection. These factors, along with the devastating consequences of PPRV infection across vast areas of the world, makes PPRV a very strong candidate for targeted eradication.

What lessons can be learnt from the recent successful control and eradication of BTV-8 from Northern and Western Europe? When BTV-8 first appeared in Holland in the summer of 2006 it came as a massive shock, as BTV had never previously been seen so far north in Europe. At the time no vaccine was available and it took two years from the time that the first case of BTV-8 was confirmed to the time that the first dose of vaccine was administered to an animal. During this 2 year period the virus spread rapidly across the whole of Europe, causing high levels of morbidity, and to a lesser extent mortality, in goats, sheep and cattle. Clearly, if a vaccine had been available in 2006, Europe would have avoided the devastating economic and welfare consequences of this outbreak. The fact the 26 serotypes of BTV currently exist, each needing a separate vaccine, makes it necessary to have seed stocks of all 26 serotypes available and ready to be manufactured into vaccines should the need arise. The reasons why it proved so relatively easy to eradicate BTV-8 from northern and western Europe are likely to relate to the long length of protection in both naturally infected and vaccinated animals (Eschbaumer et al., 2012, Oura et al., 2012, Batten et al., 2013).

Another member of the Reoviridae family, African horse sickness virus (AHSV), has also proved difficult to control. Live attenuated vaccines are currently available for all 9 serotypes, however there are very strong safety concerns related to these vaccines, making it highly unlikely that they would be used outside Africa. Data will be presented showing that live vaccine strains of AHSV are circulating freely in West Africa (Oura et al., 2012). For such a potentially devastating virus that is capable to decimating such an important industry, it is astonishing that killed vaccine stocks are not currently available in European vaccine banks.

What makes ASFV so difficult to control? In Sub-Saharan Africa, the abundance of reservoir hosts (warthog, bushpig and Ornithodorus ticks) makes control almost impossible. In Sardinia and Russia however, the reservoirs of infection are not present but control / eradication of the virus is proving extremely challenging. Question being asked include whether the virus may be being maintained in the environment through circulation within the wild pig populations. The main reason why ASFV has proved so difficult to control when it gets out of Africa is the lack of an effective vaccine. Multiple genotypes of ASFV occur with little or no cross protection between them, however a vaccine that protects pigs from infection with all the genotypes of ASFV has proved elusive. Many groups around the world are now concentrating on trying to produce a vaccine against the ASFV genotype that is currently circulating in Russia.

Finally, how can we be prepared for the unknown? Schmallenberg virus is an example of a novel virus that appeared from nowhere and has since spread far and wide across Europe and beyond. Many questions remain unanswered about how economically significant this virus will be, whether it will simply die out when it has infected all the susceptible ruminants in its
path, or whether recurring bouts of infection with different variants of the virus will continue in the years to come.

References:

C.A Batten, L Edwards, C.A.L Oura (2013). Evaluation of the humoral immune response in adult dairy cattle and adult sheep four years and 2.5 years post vaccination respectively with a bluetongue serotype 8 inactivated vaccine. In press, Vaccine.


Bovine respiratory disease is the most serious disease affecting housed cattle worldwide and is a major impediment to production as a result of morbidity, mortality and poor growth rates leading to huge economic losses. Over the last decade, it has become increasingly clear that infection by mycoplasmas, chiefly \textit{M bovis}, are major primary and contributory causes to BRD worldwide. Mycoplasmas have also been shown to be involved in a range of other clinical conditions including mastitis, arthritis, otitis and reproductive disorders but their true impact is unknown because of the lack of in-depth research in this area.

So what is the evidence that convinces me that \textit{M bovis} is being underdiagnosed? One of the best examples is what happened in both the north and south of Ireland which were \textit{M bovis} free until the early 1990s when the relaxation of import controls within the EU led to the influx of large numbers of cattle from parts of Europe where \textit{M bovis} was endemic. Within a year, \textit{M bovis} was being isolated from over a fifth of pneumonic lungs rising to over 40\% ten years later; nearly half of these only involving this mycoplasma (Blackburn et al 2007).

In Britain we estimate that \textit{M bovis} is associated with a quarter to a third of calf pneumonia as measured by serological testing and molecular diagnosis. With the increased international movements of cattle, there are few countries free of \textit{M bovis} with China describing the disease for the first time in 2008. In North America, \textit{M bovis} is an uncomplicated cause of mastitis leading to severe losses in the large dairy herd as well as a major problem in the huge feedlot industry. In Italy it has been consistently identified as the main impediment to the beef and dairy production in the north. In one study 100\% of veal calves and over three quarters of beef cattle became exposed to \textit{M bovis} during fattening which resulted in death, lungs lesions and poor growth (Nicholas et al 2008).

Great improvements have been seen over the last decade in mycoplasma diagnostics. Once it took up to a month to get a result because of the slow process of isolation and identification and results were not always accurate because of the ability on commensal mycoplasmas to overgrow pathogens like \textit{M bovis} and the even more fastidious \textit{M dispar}. Nowadays serological testing by ELISA will give a cheap and rapid result. Confirmation can be achieved equally as quickly with the use of PCRs which can detect \textit{M bovis} directly in clinical material like nasal swabs, lungs and milk. At VLA we use a modification of the PCR which includes a highly discriminating identification system called DGGE (denaturing gradient gel electrophoresis) which can detect mixed cultures as well as uncultivable mycoplasmas.
Research from our laboratory has shown that many mycoplasmas including *M. bovis* can form protective biofilms which enable them to survive and withstand host defences and chemotherapy (McAuliffe et al. 2006). They may also be more infectious in this biofilm state, requiring fewer organisms to initiate infection.

The spread of *M. bovis* is invariably the result of introducing infected animals which then transmit the disease by close and repeated contact to other animals; the cheapest form of control then is not to introduce the infected animal in the first place. In Ireland TEAGASC showed this was feasible when they began screening herds prior to restocking following the BSE culls in the 1990s and remained *M. bovis*-free.

While it is well known that the mycoplasmas are inherently resistant to the penicillins and cephalosporins, numerous studies have shown increasing *in vitro* MIC values for many of the commonly used antibiotics including the fluoroquinolones over the last 10 years indicating that *M. bovis* strains were becoming more resistant as measured *in vitro* (Nicholas et al. 2008). However the ability of some antibiotics with high MICs to successful control some infections *in vivo* may well relate to their particular mode of action possibly involving their anti-inflammatory effects and/or indeed to their action on other bacterial infections like *Histophilus somni* and *Mannheima haemolytica* that are often found in mycoplasma disease. Conversely the inability of some antibiotics with low MICs to control infections may indicate the late or chronic nature of the disease or indeed the virulence of the pathogen itself.

As in many disease areas, the use of vaccines to control disease will be a greener and more longer term option but work on vaccines for *M. bovis*, which was shown to be feasible nearly a decade ago (Nicholas et al. 2002), has been agonisingly slow with a commercial vaccine unlikely to reach the market in Europe for another 5 years. In the meantime VLA has been producing autogenous vaccines for use on individual sites. While success has been difficult to gauge because of the limited data available from these sites, evidence suggests that where vaccines are given at a sufficiently young age, say 2-4 weeks, before the majority become infected, then reductions in mortality and morbidity as well as treatment costs can be achieved (Nicholas et al. 2008).

**References**


CONTROL OF DISEASE IN EXOTIC ZOO ANIMALS

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The control of (emerging infectious) diseases in zoos may vary enormously depending on the size of a zoo, management demands, whether a veterinarian is fully employed or working on a consulting basis, the species of animals kept, the veterinary surveillance protocols used, the amount of exchange of animals, etc. But as a rule developed by EAZWV (European Association of Zoo and Wildlife Veterinarians) each zoo should have a written annual surveillance plan as a veterinary protocol to control and/or prevent disease. The Annual Disease Surveillance Plan and the measures based thereon must include:

Close observation of each animal at least once per day by suitably qualified staff, under the direction of the zoo veterinarian (in the case of large group species, such as fish in an aquarium, the veterinarian may decide that observation of the group is sufficient).

Immediate notification of the zoo veterinarian by zoo staff if any animals appear unwell or die (in the case of large group species, notification may be triggered by mortality above an agreed, expected level).

Laboratory examination to establish the infective agent in any live animals that appear to be affected by an infectious disease (in the case of large group species, such as fish in an aquarium, the veterinarian may decide that a representative sample is sufficient). In the case of suspicion of a disease that is notifiable under national and/or European legislation, the official veterinarian must be informed immediately.

Procedures should be included for newly arrived and diseased animals, taking into account the relevant risk factors and, therefore, including handling practices, clinical examination and specific tests as appropriate.

Regular parasitological examination of faecal samples (individual or group samples, depending on the housing system) in particular with regard to zoonotic parasites. It is recommended by EAZWV that all relevant groups should be checked at least once a year; the frequency of examination should be related to the prevalence of parasites.

Opportunistic examination and taking of appropriate samples from immobilised or otherwise restrained animals. EAZWV recommends all serum samples to be retained and stored at –18°C or below.

Post mortem examination without unnecessary delay to check for significant pathology, and as far as possible to establish the cause of death in every animal that dies or foetus that is aborted (but the zoo veterinarian may exercise discretion where there is clearly no suspicion of infectious disease, such as obvious trauma, or euthanasia of a healthy animal).
A vaccination programme should be based on the availability of safe vaccines. It should take into account the species involved and the risk of diseases likely to occur in the zoo.

Records must be kept in an easily accessible form, to be available as necessary for audit purposes, and retained for at least 10 years, to show at least the following information:

- All cases of disease, and treatment if applicable.
- Preventive actions such as vaccinations.
- Results of blood tests and other diagnostic procedures.
- Results of post mortem examinations including records of stillbirths.
- Observations during any periods of precautionary isolation.

Incoming animals must be isolated. The isolation quarters must be physically separated from other animal accommodation by a reasonable distance, taking into account the species concerned and the ability of the relevant viruses to spread on the air. This distance can be much reduced if the exhausted air is filtered. The limits of the isolation area must be clearly demarcated by walls or fences as appropriate. This does not preclude the possibility that specific areas or pens within the premises may be designated as isolation areas for a limited time and a particular purpose, provided that they meet the general requirements. There must be a double door system to prevent escape at the entry/exit with sufficient space between the doors to allow one to be closed before the other is opened. Entry/exit doors must be lockable and must display a notice stating: ‘Quarantine No Admission to Unauthorised Persons’. Facilities must be available at the entry/exit point for attendants to change overalls, to change and disinfect boots, to wash hands, and if appropriate to shower. Suitable facilities must be available to load or unload animals between transport crates and isolation pens without the risk of escape. Suitable crush or penning facilities should be available within reasonable access of the isolation area, so that animals may be safely restrained for clinical and diagnostic procedures such as blood sampling. The route from isolation to restraint must not put other animals at risk of infection from the introduced animals. The design of the pens or cages within the isolation area must be such that the animals may be visually inspected at any time, with adequate light and ease of access. The physical structure and all equipment must be made of such materials that they can be effectively cleansed and disinfected, or destroyed after use. The design must be suitable to minimise access by rodents, wild birds and insects, as appropriate for the species in question. Where drains are present, they must be fitted with rodent proof covers. The feed store must be suitably protected from vermin. Adequate storage facilities must be available to contain the litter and animal waste produced during the isolation period, and the storage facility must be bird and vermin proof. There must be facilities to dispose of the waste either during or after the isolation period in a way which will ensure that there is no risk of the spread of disease. Refrigeration facilities or equivalent must be available within the isolation area, or in a suitably disease-protected location nearby, to hold carcasses of animals that die until they can be subject to post mortem examination. Staff entering the isolation premises must always change into protective clothing and footwear. On leaving, the overalls and footwear must be removed and left within the isolation area, and the footwear must be disinfected. Hands must be washed, or otherwise disinfected, on entering
and leaving. Premises must have an effective programme, laid down in writing by the zoo veterinarian, for cleansing and disinfection after each isolation session; approved disinfectants must be specified and used in the programme; and an appropriate resting period (usually 7 days) must be specified after each cleansing and disinfection operation. Isolation should normally last for at least 30 days, unless a longer period is required to exclude specific risks such as rabies.

“Prevention is better than cure” is a statement that not only applies to the livestock industry but also to protect zoos with their variety of exotic species often endangered from incoming emerging infectious diseases.
TITLE: BLUETONGUE VIRUS: A CHANGING VIRUS IN A CHANGING WORLD

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Many different factors are likely to contribute to the increased levels of movement of viral pathogens currently being seen around the world. The biggest culprits are likely to be globalisation (the movement of animals and animal products) and climate change (affecting mainly insect-transmitted viruses). New viruses are appearing and known viruses are constantly changing and adapting to new ecosystems, which makes it extremely difficult for the scientific / research community to maintain the flow of accurate information needed to inform policy makers. Bluetongue virus (BTV) is a perfect example of a virus that presents all these challenges.

In some parts of the world (the Americas and the Caribbean), BTV is thought of as being a virus that does not cause significant clinical disease in infected ruminant species, so it is largely ignored by policymakers and veterinarians. Historically BTV has been confined to various parts of the world and its vectors (Culicoides sp.) have been found in relatively distinct global ecosystems. In recent years however, possibly due to climate change, the situation has become far more complicated with midge species and BTV strains / serotypes appearing in new geographical areas, causing serious outbreaks of disease in naïve ruminant populations. Additionally, to make the situation even worse, novel virulent strains / serotypes of BTV have appeared which are pathogenic in cattle and have alternative transmission mechanisms (transplacental, oral and direct contact). This has transformed BTV into a potentially more virulent, reproductive pathogen, with more serious consequences for policy makers and international trade.

The recent emergence of two unique BTVs [bluetongue virus serotype 8 (BTV-8) and bluetongue virus serotype 26 (BTV-26)] has completely changed scientific thinking related to the epidemiology and transmission of BTV. Clinical signs of BTV infection were thought to be confined predominantly to sheep (particularly the improved meat and wool breeds) and white-tailed deer, however, the strain of BTV-8 which recently spread across Europe also caused clinical signs in cattle and goats. This virus was also capable of being transmitted through both the oral and transplacental routes (Menzies et al., 2008, EFSA Panel on Animal Health and Welfare, 2011). This combination of transplacental and oral spread has extremely serious consequences for international trade and would enable the virus to survive a winter period (over-winter).

A second unique BTV, which was later identified as a novel serotype (BTV-26), was isolated from a sheep and goat flock in Kuwait (Maan et al., 2011). In order to investigate the
virulence of this novel BTV strain in sheep and goats, two experimental infection studies were performed (Batten et al., 2012, Batten et al., 2013). Sheep infected with BTV-26 exhibited mild clinical signs including conjunctivitis, reddening of the mouth mucosal membranes, slight oedema of the face and nasal discharge, however the levels of virus and viral RNA detected in the blood of the sheep was very low compared to that seen with other field isolates of BTV. In contrast, when goats were experimentally infected with BTV-26, they exhibited no clinical signs, however high levels of virus and viral RNA were detected in their blood. In the goat experiment, one in-contact uninfected control goat became infected 21 days after the other goats were experimentally infected, suggesting that BTV-26 may be spread by direct contact transmission. In a follow-up experiment, contact transmission of BTV-26 was confirmed to readily occur in goats sharing the same pen. A low level of BTV RNA was detected in nasal/ocular swabs taken from the infected goats, indicating that nose / eye secretions may be the source of the virus (unpublished). This is the first evidence of any BTV being transmitted through the direct contact route and has far-reaching consequences for international trade. Current understanding is that, during the midge-free winter period, BTV transmission stops. This will clearly not be the case for BTVs that are transmitted by direct contact.

In recent years the world has seen the emergence of two novel BTVs (BTV-8 and BTV-26) with unique phenotypes related to virulence and transmission. BTVs are segmented double stranded RNA viruses that are known to freely undergo reassortment in nature. There is current evidence from the field in Europe and North Africa (Morocco) indicating that co-circulating BTV strains can lead to the evolution of ‘reassortant’ viruses. The reassortment (mixing) of segments from strains with different phenotypes could result in the evolution of viruses that are both highly pathogenic and capable of being transmitted through the transplacental and the direct contact routes. If such viruses appeared in nature, current thinking on the epidemiology / transmission of BTV would need to change, and international trade regulations related to BTV would need to be rewritten.

References:


