Nature Conservation and Veterinary Problems

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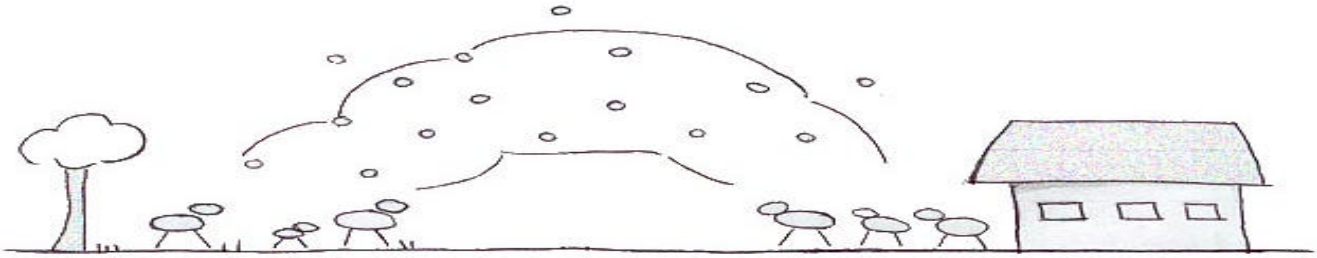

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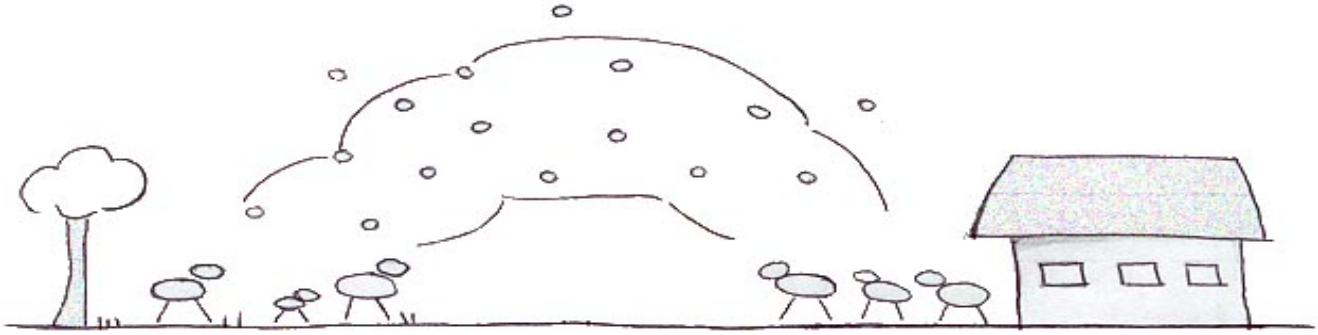

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Summary

Conflict between nature and veterinary policy

In 2003, the Large Herbivore Foundation (LHF) approached the Science Shop for Biology with a request to explore the conflicts between the EU veterinary legislation and LHF's core activity: nature restoration, involving introduction, conservation and reintroduction of large herbivores. Herbivores like deer and bovine species, and rooting omnivores like wild boar play an important role in shaping and maintaining natural landscapes. The EU aims at the creation and where necessary restoration of large interconnected nature reserves, where wild populations find a natural habitat and can maintain themselves without interference by humans. This goal is often frustrated by veterinary rules, which were designed for farm animals and don't take wildlife and nature conservation issues into account. Specifically, nature restoration with originally domestic species in a process of de-domestication, which are employed as ecological surrogate for once endemic but now extinct or endangered species like wild horse, aurochs, wild sheep and bison, are under the same rule as farm animals.

In this report, the problems veterinary policy incurs to EU's nature policy are studied, with a special emphasis to transmission risks of infectious animal diseases between wildlife and livestock. This is done by reviewing Foot and Mouth Disease and Classical Swine Fever. The worst conflicts between conservationists and veterinary authorities have to do with these two diseases. They are both capable of causing massive epidemics, running havoc amongst susceptible farm animals. Controlling an outbreak involves the killing and destruction of all infected and in-contact animals. Recent epidemics of Classical Swine Fever and Foot and Mouth Disease resulted in the mass destruction of millions of healthy animals, causing public outrage. Not only production animals were killed, but also pets and animals kept for hobby or recreational purposes. Valuable animals, including endangered species were in danger of extermination. Yet, protective vaccination was out of the question, because of trade interests. For the public, this was hard to understand, because vaccination could have saved a lot of animals.

Infection risks

We reviewed the literature using the following key questions:

(1) What are the major transmission routes of the disease and how are they to be ranked in order of importance?

(2) What do we know about transmission risks between livestock and wild or free-roaming domestic populations?

(3) What measures are available to prevent and control disease outbreak and how do they interfere with nature conservation, restoration and management?

(4) How can control and prevention measures be adapted so as to better serve nature management with large herbivores as well as the protection of wild species?

For *Foot and Mouth Disease* (FMD) it was found, that the major transmission routes are direct contact between infected and susceptible animals in the short range and airborne transmission over long distances. The latter, plus the fact that a great many species are susceptible makes FMD very hard to control. Primary outbreaks however, are almost invariably caused by illegal import of infected animals or their products, often followed by illegal feeding them to farm animals. There is no record of primary outbreaks caused by wildlife, and most transmissions between wildlife and livestock are from farm animals to wildlife, not the reverse. After eradication among farm animals, FMD naturally disappears from infected wild populations. As for prevention and control measures, from an ecological point of view it is best to have the disease run its course, should wild populations be infected. Endemic diseases like FMD and Classical Swine Fever have an important function in wild populations. Apart from killing infected and at risk animals, vaccination is

possible, either as a measure to suppress the disease and to quickly halt the epidemic, or to protect them from infection. In the first case, vaccinated animals will be killed afterwards, in the second case, there is a risk that vaccinated animals become carriers, which mean that they don't show symptoms but can transmit the infection. From a nature point of view, the best option is to isolate susceptible farm animals from wildlife. Ring vaccination of farm animals around nature reserves is an option in case of an outbreak. For endangered species, vaccination of wildlife should be considered. Monitoring wildlife for infectious diseases can be done in a non-invasive way and is an important tool.

Classical Swine Fever (CSF) affects only swine and is not capable of airborne long distance transmission. Main transmission route involves direct contact with infected animals or meat. Most primary outbreaks are caused by (illegal) feeding infected meat or *swill* (kitchen and restaurant waste) to domestic pigs. In a few places in Europe, CSF is endemic in boar populations. Depending on the strain, it can become endemic, which has happened in Sardinia. Yet, like FMD, in most places where CSF has been eradicated from domestic pigs, it disappeared from wild boar as well. During an outbreak, for wildlife it is best to follow the same course as in FMD. Where CSF is endemic in wild boar, good farm hygiene and prevention of direct contact are the best precautions. For the purpose of eradication of CSF from wild boar populations, oral vaccination sometimes has been successfully employed. Lowering the population density, in order to slow the spread of infection, and make it die out, has been considered but involves practical problems. Hunting is counterproductive in most cases. Monitoring is very important, because wild boar can carry CSF.

Disease policy

A leading role is played by the OIE, the World Organisation for Animal Health. OIE sets out guidelines to minimize veterinary risk from trade in animals and animal products. The World Trade Organisation (WTO), of which the EU is member, follows the guidelines from OIE and so does the EU. WTO prohibits protectionism and wants to eliminate trade barriers as far as possible. A disease-free status without vaccination is the most desirable from a trade point of view, because then trade obstacles are lowest. Countries are reluctant to vaccinate, because loss of trade status causes huge economic losses. After the 2001 FMD epidemic, both OIE and EU mitigated the rules for emergency vaccination, because it can help to control an epidemic more quickly. Where emergency or protective vaccination is applied, consequences for trade status have been limited as compared to the 2001 situation. Also OIE recognizes the need to develop animal welfare guidelines, not only for domestic animals, but also for wildlife.

While this is an improvement, nature conservation and ecosystem health have not been recognized as issues in their own right, and a co-responsibility of veterinarian authorities.

It is recommended that nature conservation organisations support the EU's and OIE's long-term goal of eradication of FMD, despite the fact that it is an endemic disease with an ecological role in wild populations. Modern farming systems are too vulnerable for diseases as contagious as FMD. Perhaps it is possible to maintain the ecological role of CSF to regulate boar density in large nature reserves, provided that the wild boar can be effectively isolated from domestic pigs. A precondition is, that in case of an outbreak corridors have to be closed off completely. In general, isolation of wildlife from domestic animals is important, given the transmission risk of infectious animal diseases, including non-endemic diseases like African Swine fever to wild populations from domestic livestock.

Vaccinated animals are usually killed afterwards, as vaccination is usually applied to halt an epidemic, not to save the vaccinated animals. After the 2001 FMD outbreak however, OIE and EU permitted protective vaccination for endangered species, but under very strict conditions, for which most animals living in the wild don't qualify. There is no valid reason why valuable species for other reasons than being endangered should not receive

protective vaccination, as long as they don't enter the food chain. From a conservationist point of view, the provisions OIE and EU have made for wildlife are not sufficient. They could be made less severe, without increasing transmission risk.
To encourage protective vaccination, it is necessary to diminish the economic penalties for this course of action. Main trade restrictions for veterinary reasons concern the trade in live animals. Given the frequency of outbreaks of priority diseases, it might be desirable to limit trade and transport of live animals in both vaccinated and non-vaccinated disease-free zones. It might be useful to re-examine the justification for trade barriers in the light of the current state of affairs and to take also into account other developments, such as the enormously increased trade and tourism volume.

Introduction

In 2003, the Large Herbivore Foundation (LHF) approached the Science Shop for Biology with a request to explore the conflicts between the EU veterinary legislation and LHF's core business: nature restoration, involving introduction, conservation and reintroduction of large herbivores. The Large Herbivore Foundation (LHF) was initiated by World Wildlife Fund International in 1998. As a project with the aim to form an independent network organization it was to give special attention to the conservation and restoration of Large Herbivore communities in the Eurasian nature. Focus of the programme is not just on the over 45 large herbivore species in temperate Eurasia, but also on the specific key role large herbivores play in ecosystems and the significance they have for the public.
Large herbivores have a key role in shaping and maintaining natural landscapes (Kuiters et al. 1996). Increasingly, such species are introduced, or reintroduced, in nature conservation and restoration areas, in order to achieve a more natural situation. They play a role in the dispersal of seeds, nutrient dispersal, and disturbance by trampling and species-specific grazing effects on the terrain.
The EU aims at the creation and where necessary, restoration of large interconnected nature reserves, where wild populations find a natural habitat and can maintain themselves without interference by humans. Often employed in nature conservation and restoration projects are hardened domestic species like Icelandic ponies and Scottish Highlander cattle, as well as species specifically bred to resemble wild ancestors, like Konik horses and Heck cattle, as ecological surrogates for once endemic but now extinct or endangered species. Veterinary rules however, have been tailored to agricultural activities, and do not take into account the different set of conditions for grazers in restored or natural landscapes.

In this report, the problems veterinary policy incurs to the EU's nature policy are studied, with a special emphasis to transmission risks of priority diseases between wildlife and livestock.

Controversies between veterinary and nature policy

Cromsigt (2001) has reviewed the problems posed by veterinary law to the introduction, keeping and conservation of wild and domestic herbivores in nature reserves. For wild and protected species, main problems are connected with transport, quarantine and identification requirements that enter into force whenever animals are transported. Transport of wild species, often over national borders, is inevitable, both with regard to reintroduction in their natural habitats, and because the local populations are not large enough to avoid inbreeding. Provisions, required for transport and pens for temporary housing and quarantine are often inadequate for wild species, where identification requirements are a source of considerable practical problems with wild animals. A second formidable problem is posed by rules concerning prevention and control of infectious diseases. Measures may include extermination of local populations, including protected species, even when kept in a zoo. Moreover, the presence of certain infectious diseases in wild populations may cost a nation its disease-free status, bringing about significant economic losses due to trade restrictions. Large, interconnected nature areas, allowing populations to cross-breed, as envisaged in EU nature policy, stand little chance of being realized, as long as they endanger a country's disease-free status.
Employing (originally) domestic species as substitutes for extinct or hard to come by wild species entails a host of veterinary complications. By default, domestic species, even if specifically bred as a reconstruction of extinct ancestors, like Heck cattle and Konik horse, fall under the same rule as domestic species kept for agricultural purposes, including identification and registration requirements and animal welfare laws. In addition, dead

carcasses have to be removed, which interferes with nature conservation goals for carrion eaters like vultures and ravens.
Individual identification of free-roaming animals poses huge practical problems, including stress to the animals and undesirable human interference in populations, which are meant to fend for themselves. Animal welfare rules, while not applicable to free-roaming wild species, do apply to domestic species, regardless the way they are kept. Under the law, they have to be fed and given medical care. Achieving a natural situation, where the population fulfils its role in the ecosystem, the same way a wild population does, is then impossible. To add more complications to this already difficult issue, it should be noted, that there is a whole spectrum, ranging from regular farming practice to 100% wildlife-like settings, in the way 'domestic' species are kept. In case of an outbreak of a priority disease, they may have to be killed as part of the control strategy. Also truly wild species run the risk of being killed and destroyed for veterinary reasons (Cromsigt, 2001).

Policy context

Veterinary laws serve both disease control and animal well-being. Disease control serves economic interests as well as animal well-being and human health. Countries can impose trade restrictions on animals and animal products for veterinary reasons. In general, the EU and the World Trade Organization, of which the EU is a member, are opposed to trade restrictions. OIE (Office International des Epizooties) is the World Organization for Animal Health It has the same position to animal health as the WHO (World Health Organization) has to human health. WTO has in its rules, that it follows OIE, and so does the EU (OIE, 2005, www.oie.int).
Guidelines concerning listed diseases of herbivores are set out in the Terrestrial Animal Health Code (OIE, 2004). OIE has since May 2004 changed its system, replacing the different classifications of animal diseases (list A, B, C etc) by one single list.
A listed disease complies with one or more of the following criteria

Basic criteria	**Parameters (at least one 'yes' answer means that the criterion has been met)**
International spread	Has international spread been proven on three or more occasions? **OR** Are more than three countries with populations of susceptible animals free of the disease or facing impending freedom (based on the Terrestrial Code provisions, especially Appendix 3.8.1)? **OR** Do OIE annual reports indicate that a significant number of countries with susceptible populations have reported absence of the disease for several consecutive years?
Significant spread within naïve populations	Does the disease exhibit significant mortality at the level of a country or zone/compartment? **AND/OR** Does the disease exhibit significant morbidity at the level of a country or zone/compartment?
Zoonotic potential	Has transmission to humans been proven? (with the exception of artificial circumstances) **AND** Is human infection associated with severe consequences? (death or prolonged illness)
Emerging diseases	Is there rapid spread and/or apparent zoonotic properties?

(Source: OIE, 2004, Terrestrial Animal Health Code, www.oie.int)

For each listed disease, OIE members are required to take 'adequate measures' for prevention and control, which may differ, according to the region. The Terrestrial Code provides guidelines per disease. Outbreaks and suspicions of outbreaks have to be notified to the regional veterinary administration. For some diseases, there exist regional or even global eradication programmes, aimed at attaining a disease-free status for the whole region.

Official disease-free status

'Since the early 1990s, the OIE has been given by the International Committee, composed of the Delegates of the OIE Member Countries, the responsibility of compiling a list of Member Countries or zones that are officially recognised as being free from certain diseases. For this purpose, a clearly defined and impartial procedure for declaring a Member Country free from a disease was necessary, accompanied by well-designed, science-based questionnaires.

In May 1995 a new procedure was adopted by the International Committee. Developed by the Foot and Mouth Disease (FMD) and Other Epizootics Commission (now called the Scientific Commission for Animal Diseases), which is elected by the International Committee, it permitted the OIE to examine in detail dossiers submitted by the Delegates of Member Countries in support of a claim that their countries or zones within their countries could be considered free of FMD in accordance with the provisions of Chapter 2.2.10. of the Terrestrial Animal Health Code (FMD was the first disease chosen in the light of its significance for international trade).

In 1996 the first official list of OIE Member Countries or zones that were FMD free without using vaccination was published after adoption by the International Committee.

While this mechanism applied to the recognition of national FMD status, the International Committee next recognised the need to apply the procedure to rinderpest and other diseases deemed to be of priority. To date, the OIE has a specific procedure for FMD, rinderpest and contagious bovine pleuropneumonia (CBPP). The conditions that apply to bovine spongiform encephalopathy (BSE) are close to being adopted.

Official OIE recognition of the absence of certain diseases, if the case arises, with or without the use of vaccines is essential to OIE Member Countries that engage in international trade.

Member Countries can also declare themselves free of diseases for which there is, as yet, no specific procedure for obtaining Official OIE recognition of Member Country status. In this case, they must provide the relevant epidemiological information to importing countries in proof of their position. The data provided must conform to the standard measures contained in the Terrestrial Animal Health Code, which is recognised by the Agreement on the Application of Sanitary and Phytosanitary Measures (SPS Agreement) of the World Trade Organization (WTO).'

(Source: OIE, www.oie.int)

The OIE only has a legally binding protocol for a disease-free status for FMD, rinderpest and CBPP (Contagious bovine pleuropneumonia), with a protocol for BSE coming soon. For other listed diseases, it offers guidelines, concerning a disease-free status and the requirement of animals and products offered for trade. EU and its major trade partners try to maintain a disease-free status for quite a few diseases. In practice, the former List A of OIE is a list of diseases on a programme of regional eradication and guidelines of OIE are quite authoritative. In the EU, animal health is the responsibility of the European Commission, specifically the Directorate General SANCO, consumer affairs and public health, while nature conservation and biodiversity belongs to DG Environment. The website of the European Commission provides information and contact details of responsible agencies and advisory committees within DG Sanco.

OIE guidelines are frequently reviewed and modified according to scientific advice in several areas. The OIE has three permanent working groups, on wildlife diseases, on animal welfare and on food safety, and it can appoint ad hoc working groups to address specific issues. The Animal Welfare Working Group is aware of the ethical dilemmas concerning wildlife and protected species. It is interested in contributions of stakeholders to their discussions, including animal welfare organizations and nature conservation groups. The Wildlife Diseases Working Group is primarily occupied with surveillance of wildlife disease and development of guidelines for management of wildlife diseases (OIE, 2002). Recently, they have established links with the IUCN and with the World Association of Wildlife Veterinarians, which may indicate that they are ready to adopt a wider perspective (OIE, 2004). Contact details of both working groups can be found on the OIE website, www.oie.int.

The objective of this study

The Large Herbivore Foundation and likeminded organizations call for a veterinary policy that also serves the interest of nature conservation, i.e. allows for the co-existence of wild and semi-wild free-roaming populations with domestic animals. At the root of the many problems reviewed by Cromsigt (2001) lies the transmission risk of infectious animal diseases between wildlife and livestock.
The objective of this study is to explore the possibilities of a more nature-friendly disease prevention and control strategy with respect to the risks of infectious diseases. This has been elaborated for two cases, foot and mouth disease and classical swine fever.

Key questions in this respect are:

1 What are the major transmission routes of the disease and how are they to be ranked in order of importance?
2 What do we know about transmission risks between livestock and wild or free-roaming domestic populations?
3 What measures are implemented to prevent and control disease outbreak and how do they interfere with nature conservation, restoration and management?
4 How can control and prevention measures be adapted so as to better serve nature management with large herbivores as well as the protection of wild species?

In the first introductory chapter background information is given about infectious animal diseases. In chapter 2 available control and prevention strategies are discussed, with special emphasis on the wildlife/ livestock interface. The above key questions are addressed in the next two chapters involving case studies on Foot and Mouth Disease (FMD) and Classical Swine Fever (CSF). Both diseases used to be classified by OIE as List A, but only for FMD there is an official OIE protocol regarding a country's or region's disease-free status. In the concluding chapter, the results are discussed in a more general perspective and recommendations are offered.

1 Infectious Animal Diseases

1.1 Introduction

An infectious animal disease is usually caused by external factors such as parasitism. Parasitism is a condition in which two organisms live together, one deriving its nourishment at the expense of the other. Parasitic organisms belong to a wide range of taxonomic groups. A heavy load of parasites is an infection and a disease is the outcome of an infection. The immune response against parasites consists of white blood cells that produce antibodies. These antibodies target the antigens (or proteins) present on the surface of the parasite or released into the host. Antibodies cost a lot of energy to produce and may damage the host's own tissues. Fortunately for the parasite, the immune response does not have to kill it to be effective. It only has to reduce the feeding, movement and reproduction of the parasite to a tolerable level (Smith and Smith 1998).
But the barrier posed by the immune response can be breached. Some parasites vary their antigens more or less continuously. By doing so, they are able to keep one step ahead of the host's response. The result is a chronic infection of the parasites in the host. Eventually, it may wear out the immune system, allowing the infection to spread and become pathogenic.

Balance between parasites and host

In order to continue its existence, the parasite has to strike a balance with its host. The parasite gains no advantage if it kills its host, as a dead host means the end of its parasites. On the other hand, the host can't afford to wear itself out in resisting the parasite. The host has to reach an optimum level of immune response, balancing beneficial and harmful consequences. It has to direct enough of its metabolic resources to minimize the cost of parasitism, without impairing its own growth and reproduction. The parasite has to achieve optimal growth and reproduction without overwhelming its host. Not to do so would be detrimental to both. In the course of evolution, host and parasite tend to develop a mutual tolerance (Smith and Smith 1998).

Parasites are distinguished by size and may be classified as microparasites and macroparasites. Macroparasites include flatworms, roundworms, flukes, lice, fleas, ticks, and fungi. Macroparasites have a comparatively long generation time and direct reproduction in the host is rare. They can spread by direct transmission from host to host or by indirect transmission, involving intermediate hosts and carriers. Microparasites include viruses, bacteria and protozoa. They are characterized by small size and a short generation time. They develop and multiply rapidly within the host cells and tend to induce temporal of lifelong immunity to reinfection in hosts surviving the initial infections. The duration of the infection is short relative to the expected life span of the host. Transmission from host to host is direct although other species may serve as a carrier vector. They require a high host density to persist (Smith and Smith 1998).

1.1.1 Virus diseases

Most rapidly spreading epidemics are caused by microparasites, in particular airborne virus diseases. Viruses are more dependent on their hosts than macroparasites and bacteria. They are obligate intracellular parasites, meaning that they can only reproduce within a host cell. Isolated viruses are merely packaged sets of genes in transit from one host cell to another. Each type of virus can infect and parasitize only a limited range of host cells, called its host range. This host specificity depends on the evolution of recognition systems by the virus. Some viruses have host ranges broad enough to include several animal species, while others are specified to one tissue-type in one animal species.

A viral infection begins when the genome of a virus makes its way into the host cell. The mechanism by which this nucleic acid enters the cell varies. Once inside, the viral genome can command its host, reprogramming the cell to copy the viral nucleic acids and manufacture viral proteins. FMD virus and CSF virus are RNA viruses, meaning that they use the host's RNA as a template for their own reproduction. The reproductive cycle of a RNA virus is given in figure 1.

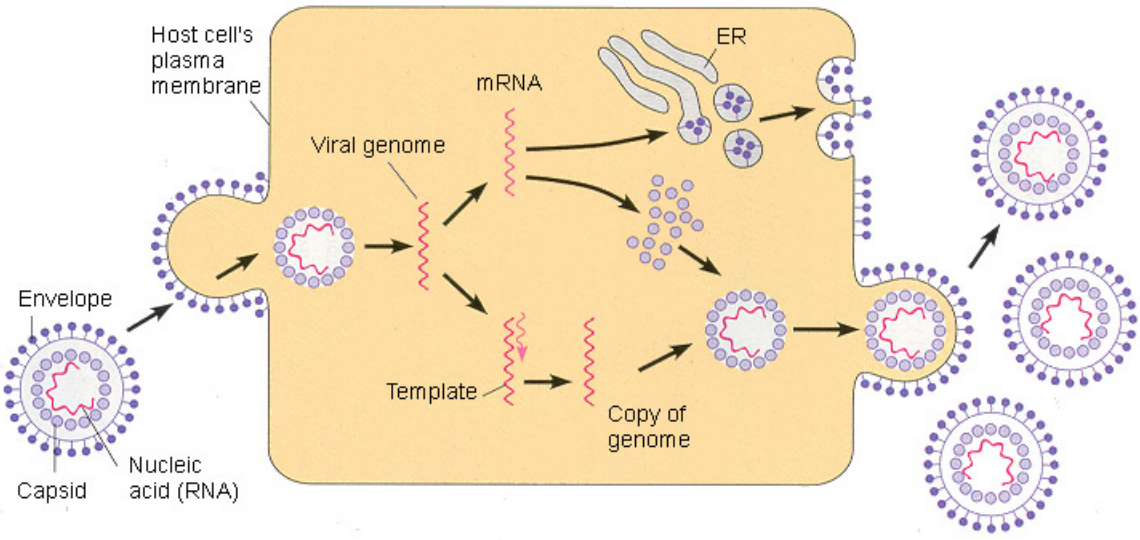

Figure 1. The replication cycle of a single-stranded enveloped RNA virus. The proteins of the viral envelope recognize and bind to specific receptor molecules (not shown) on the surface of the host cell. The envelope fuses with the cell's plasma membrane and the capsid and the viral genome enter the cell. The viral genome functions as a template for making complementary RNA strands, which have two functions: (1) they serve as template for making new copies of genome RNA and (2) they serve as mRNA. The mRNA is translated into both capsid proteins and proteins for the envelope. The cell's endoplasmatic reticulum (ER) synthesizes the proteins for the envelope that are transported to the cell's membrane by vesicles. A capsid assembles around each viral genome molecules and the virus buds from the cell.
(*After fig. 18-6, Campbell et al. 1999)*

This cycle is approximately the same for FMD virus and CSF virus, except that the FMD virus is not enveloped.

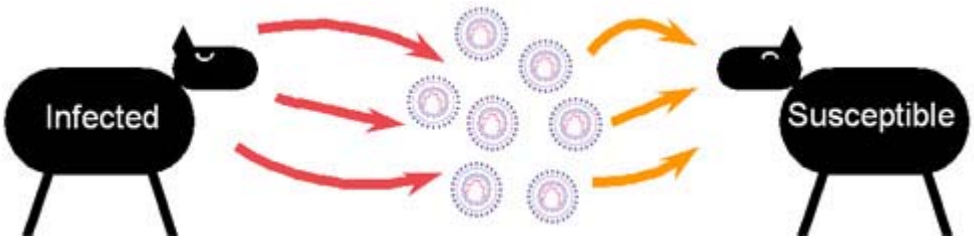

Figure 2. The transmission of a virus from an infected animal to a susceptible animal Transmission routes (arrows from infected animal) are different ways for a virus (balls) to get outside the body of an infected animal and be placed directly (or indirectly) in a position that enable a susceptible animal to possibly get infected. The infection routes (arrows to susceptible animal) are different ways for a susceptible animal to get infected with a virus when it is in the vicinity.

Thus, a virus diverts its host's resources for viral production. The simplest type of viral reproductive cycle is completed when hundreds or thousands of viruses emerge from the

infected host cell, which is often destroyed in the process. In fact, some symptoms of viral infections result from cellular damage and death and/or from the body's responses to this destruction. Cells may produce toxins after reprogramming, which can also cause symptoms. The viral progeny that leave a cell have the potential to infect additional cells, spreading the viral infection (Campbell et al. 1999). The amount of virus in the body of the host thus keeps building up and the virus may infect host cells that provide a way out, enabling the virus to be transmitted to other animals, for example epithelium cells.

1.1.2 The spread of an infection

Viruses find their way to another host via different transmission routes (figure 2). A transmission route is a way for a virus to get outside the body of an infected animal by excretions, secretions or dead body parts and be placed directly (or indirectly) in a position so that a susceptible animal could get infected. An infection route is a way for a susceptible animal to get infected with a virus when it is in the vicinity. The manner in which the virus gets into the body of a host needs not be the same way as the virus took in leaving its host. For example, a sneeze (transmission route) from someone who has a cold can release virus particles into the air and by eating (infection route) a sandwich on which virus containing droplets landed, a susceptible person can become infected.

A disease is highly infectious when the virus replicates rapidly inside the host and is excreted in large amounts by a transmission route that can easily infect susceptible hosts. Transmission risk is *density-dependent*, meaning that in circumstances of high density, such as in stables and meadows, the probability that excreted viruses will find their way to another host is high.

Pathogens can play an important role in the processes of the ecosystem. The pathogen may regulate the population of a host, by killing infected animals directly or by reducing the immune response so that secondary infections can take hold and kill infected animals. This reduces the population density to such a level, that transmission of the pathogen is minimal. It is also possible that the pathogen and host species are well adapted to each other. In these cases the host is persistently infected with the parasite, but is minimally affected by the pathogen. Such a host species may be a reservoir of infection for other species in the ecosystem. On the other side of the spectrum there are dead-end hosts. The animals of such a host species can become infected by the pathogen, but the animals are killed by the pathogen before the pathogen had time to reproduce and become transmitted to another animal.

The basic reproduction rate (Ro) of a pathogen can be helpful in assessing to what extent factors can influence the epidemiology of a disease. An infection will turn to an epidemic when the basic reproduction rate is much greater than one (Ro>1), meaning that more animals are infected in the new 'generation' than in the previous 'generation'. Thus, the number of infected animals increases constantly. The disease will prevail when the basic reproduction rate of the virus approximates one (Ro=1), meaning that the number of infected animals in the population remains constant. When the basic reproduction rate falls below one (Ro<1), the infection will die-out, because fewer animals are infected in the new 'generation' than in the previous 'generation'. This means that the number of infected animals decreases until there is no one left.

1.2 Transmission of Animal Diseases

The severity of an infectious disease commonly differs between species, but this difference is also dependant on the strain or serotypes involved in the infection. Strains sometimes show differences in preference for a particular transmission route. Virulence and infectivity also vary with the serotype of the pathogen. Strains causing only mild symptoms may pass unnoticed for some time, allowing the disease a wide proliferation, before control measures are put in place.

An endemic infection means that the pathogen can persist inside an area or population, without input from another infection source. This does not mean that every animal carries the pathogen all the time. The pathogen is transmitted from host to host like a baton in a relay race forever going round and round in a population or area with susceptible animal. Lowering the population density can stop this, because it diminishes the chance that infected animals meet susceptible animals. The disease will then eventually die out. An infection can also be self-limiting in host species, because after a while most animals are infected and either die or acquire immunity. After the immunity has worn off, reinfection can occur from an outside infection source. This infection source needs not be an endemic host. The relay race can be seen as an infection cycle from species to species as well as from infected animals to susceptible animals.
It is important to keep in mind that pathogens may not take all the routes of transmission and not every host species is fit for every transmission route. The amount of pathogen that animals excrete depends on the route of transmission, the type or strain involved, the species that is infected and the stage of infection. The amount of pathogen needed to infect a susceptible animal depends on the animal species, the pathogen strain or type and the route of infection. The first distinction in transmission routes can be made on the way a pathogen can bridge the distance between old host and new host. With direct transmission the distance between infected and susceptible animal has to be small. With indirect transmission the distance it can be far greater.

1.2.1 Direct transmission

When an infected animal meets with susceptible animals, transmission commonly occurs through direct contact. Infected animals may carry the pathogen in various excretions and secretions (such as saliva, blood, faeces, milk) that can come into contact with susceptible animals. Coughing and sneezing is the most common way for a pathogen to get outside its host's body. Contact with infected blood (from scabs and wounds) or milk sometimes infects susceptible animals.
A second route of direct transmission is through sexual contact. The pathogen may be present in the semen of an infected male animal. Infected sperm may transmit the disease by natural or artificial insemination. Infected females may transmit the disease to males through sexual intercourse. This route seems unlikely for transmission between different species, but inter-species sexual contact occasionally happens.
A third route of direct transmission is from infected mother to foetus. When a pregnant female animal gets infected it may infect her offspring if the pathogen can pass the placental barrier. In farm animals, transmission via infected embryos transferred to host mothers is also a route to be considered. Infected offspring may die in the womb or after birth due to the infection. Sometimes survivors are subclinically infected after birth, meaning that they excrete the pathogen without showing any symptoms of being infected. Also surviving young may be latently infected, meaning that they carry the pathogen with them without excreting it. Reactivation of the pathogen may take place in certain circumstances. These latter two types of infection even occur in adult animal.
The last route of transmission is direct contact with dead animals. Infected animals that die from their infection may hold the pathogen in large amounts for a long time. Latently infected animals or carriers that die of other causes sometimes pose a transmission risk. Scavengers (such as pigs and crows) and carnivorous predators may eat the meat of a dead animal and become infected. Pigs also eat dead pigs. This is a common phenomenon and may cause an infection to persist within a pig population. The question is, how long the pathogen can survive inside the tissue of a dead animal. Only then can the risk of transmission from dead animals in nature reserves be assessed. The transmission of prions causing Bovine spongiform encephalopathy (BSE) is suspected to occur through the feeding of animal by-products, such as meat and bone meal. BSE in cattle is suspected to originate from feeding of animal by-products of sheep infected with scrapie (a common prion disease of sheep) to cattle. Eating BSE infected meat may cause

Creutzfeldt-Jakob disease in humans at an uncommonly young age (for a review see Blanchfield 1998).

1.2.2 Indirect transmission

The routes of direct transmission occur more frequently, but the distance direct transmission is able to bridge is small. Indirect transmission routes may occur less frequent but have the potential of spreading over greater distances. This depends on modes of transport and the pathogen's survival outside its host. There are two routes of indirect transmission that are distinctly different from each other in some ways and similar in others. The first is through abiotic vectors and the second is through biotic vectors. Biotic vectors are commonly identified as animals or humans, as there is little known about plants and other non-animals as possible vectors.

Transmission via the abiotic environment

Long distance transport of a pathogen by the environment is dependent on meteorological conditions. As these conditions vary in time, pathogens survive longer in seasons that provide the better conditions. Viruses survive best at low temperatures, because their life cycle turnover slows down and they become dormant. Unlike bacteria they don't reproduce outside a living host.

Respiratory excretion, faeces and urine from an infected population with a high density can cause aerosol formation. An aerosol is a cloud of suspended particles that can be transported over long distances by wind. These particles can be saliva or droplet nuclei containing the pathogen. Dispersal by this route depends on many factors. Wind speed, stability, pollutants, precipitation, humidity, temperature and sunlight are the most important factors favouring or disfavouring transport of active pathogens over long distances (Gloster et al. 1982). The concentration of pathogens commonly decreases with distance due to diffusion and inactivation. The amount of pathogens produced depends on the animal species as well as the density of the infected animals. In some diseases, one animal can excrete enough pathogens to form an aerosol capable of infecting another animal (commonly from another species) that is kilometres away. A few studies indicate that airborne transmission over sea is also possible over tens of kilometres. Pseudorabies was shown to spread apart by airborne transmission between islands in Denmark that were 30 to 40 kilometres apart (Christensen et al. 1993).

Another route is the transmission via soil and surface water. Some pathogens can survive in soil and water for long periods of time when the environmental conditions are optimal. Introduction of the pathogen into the environment and subsequent survival and transport can occur through many routes. For example, infected faeces may enter the environment by droppings from infected wildlife, sewage from farms and by farmers fertilizing their land. Susceptible animals may come into contact with the infected soil by grazing or drinking infected water. The pathogen may also survive on tools, materials, machinery and cars.

In order to prevent unnecessary killing of wild animals, it is important to have quantitative knowledge of transmission risks. For example, American bison straying from Yellowstone National Park are shot for fear of transmitting Brucellosis to cattle. Most likely interspecies transmission route is contact with contaminated birthing or abortion sites, but the actual risk however is probably very small, as birthing behaviour, predators and weathering 'clear' the site of Brucella bacteria (Clarke 1998, Baskin 1998).

Transmission via animal vectors

Mechanical animal vectors are animals that are themselves unsusceptible to the pathogen but can transmit it to susceptible animals. For example pets, birds, rodents, insects, carnivores and humans. Some of these vectors may eat the meat of an infected animal and excrete pathogens with their faeces.

Another route of transmission is blood. Ticks and mosquitoes may suck blood of an infected animal and transmit the pathogen when it feeds from an uninfected animal. The transmission of malaria via mosquitoes is a well-known example for humans.
The last route of transmission is via clothing, hair, fur or feathers of an unsusceptible human or animal. This is similar to the abiotic vectors, the difference being, that animal vectors provide a microclimate that is more stable. This may prolong the survival of some pathogens.
For animal vectors to work effectively, it is said that there should be enough vector animals exposed to high amounts of pathogens. They also must have a fair chance of close contact with susceptible animals. In addition, the pathogen must be able to survive the period between hosts (Gloster et al. 1982).

1.2.3 Infection dose

Infection risk is expressed in terms referring to MID (minimum infectious dose) or PFU (number of plaque forming units), depending on the method employed to determine infectivity. The MID can be converted to the probability that one infectious particle infects an animal. For example, a MID50 of 100 TCID50 would correspond to a chance of 0.0069 that one individual virus particle infects one susceptible animal. This is relevant because theoretically, only one infectious virus particle is needed to infect an animal as long as the particle penetrates a susceptible cell. Looking again at the example, the chance that one animal becomes infected by one infectious particle is bigger when there are 200 susceptible animals present instead of 10 susceptible animals (0.75 and 0.07 respectively, Sutmoller and Vose 1997). Notably, these probabilities will differ between species and between infection routes.
Three different routes of infection can be distinguished. First, there is the respiratory route where the infectious particles are inhaled and commonly infect respiratory tissue (particularly the pharynx), from which it can spread through the entire body. Second, the oral route, by which an ingested pathogen enters the body through absorption in the intestines. Last, the venal route where the pathogen enters the body, through scabs and wounds in the skin, by vectors like mosquitoes or ticks, or through blood-blood contact (from mother to foetus, sexual contact).
The time it takes a susceptible animal to contract an infection via the first two routes, depends on the amount of particles in the air or food, as well as the amount of particles an animal can inhale (respiratory route) or consume (oral route). A short period of exposure to a lot of pathogens seems to be more infectious than a long period of exposure to a low concentration of pathogens (Alexandersen and Donaldson 2002).

1.2.4 Carrier state

After exposure to a pathogen, an animal can develop a subclinical infection, meaning that it excretes the pathogen without showing any symptoms of being infected. This is referred to as carrier state. A carrier state can also result after recovery from a clinical infection, meaning that the pathogen persists in certain areas of the body. Pathogen excretion may still occur, but this will be at lower level and there will be no visible symptoms. Trigger factors, like stress, may increase the excretion level. In certain conditions, vaccination may induce a carrier state. This is possible when the vaccine reduces the clinical signs of the disease by leaves the infection intact, allowing continued transmission of the pathogen.

1.3 Evaluation of transmission risks

With sufficient data, it is possible to quantify transmission risk for each possible route. Given the state of available data, in particular with regard to wildlife, a tentative risk evaluation is the best we can manage. It involves mapping all transmission routes between livestock and wildlife and assessing in which direction the transmission is most likely to occur. The next step is ranking them in order of importance The probability of wild

populations to become a reservoir of pathogens for future outbreaks is also important, because persistently infected populations can act as reservoir. There are different factors of concern for such an assessment, such as animal density, habitat type, species behavioural differences and human influence.

- Population density is important for the transmission within a herd, because it is correlated with the chance that an infected animal and a susceptible animal meet. It is also important for the transmission between herds, because aerosols commonly form when there is more than one infected animals.
- Farm animals are kept in a broad array of settings: from animals living in stables isolated from the outside world (by absolute air filtration) to animals extensively kept in meadows or woodland where they have free contact with wild animals. Similarly, wildlife and semi-wild populations live in a variety of densities and habitat types. The diversity can range from enclosed area, isolated from other wild populations and domestic animals, to habitats allowing free access to farm animals and other wildlife populations.
- The behaviour of sick animals and the behaviour of susceptible animals when confronted with a dead or dying animal may be significantly effect the risk of (direct) transmission. Other things like foraging behaviour (solitary or in herds) and behaviour towards animal vectors could also influence the actual risk of transmission. Behaviours can thus facilitate or diminish the chance of infection apart from excretion and infection doses. Differences in behaviour can occur between species.
- Humans also influence the transmission, either as involuntarily vectors of transmission, or by implementing preventive or control measures. The effectiveness of the different measures that are taken is very important. Preventive measures that can be taken are vaccination, fencing and hunting. Control measures that can be taken during an outbreak, may be stamping out, hunting and emergency vaccination. These measures are further discussed in the next chapter.

2 Disease Control and Nature

In this chapter, a number of prevention and control strategies are reviewed and discussed with respect to their impact on nature conservation goals. The EU aims at a non-vaccinated disease-free status for a number of important animal diseases. These are generally highly communicable diseases, capable of massive transboundary epidemics and considerable economic losses. The disease-free status without vaccination gives the best guarantee, that exported animals or animal products are truly pathogen-free. This status also warrants maximum market access to other member states and WTO partners beyond the EU. WTO member states are allowed to impose trade restrictions for veterinary reasons, like preserving their own disease-free status. The price of a non-vaccinated disease-free status is the 'stamping out' control strategy. Should an outbreak occur, all infected animals as well as potentially infected animals near an outbreak site are to be killed and destroyed.

In addition to measures to ensure good health of farm animals, like veterinary controls, farm hygiene, identification, registration and certification, there are a number of measures, specifically targeted to minimize veterinary risks from and to wild populations.

2.1 Preventive measures

Prevention involves a host of regulations, aimed at preventing infected animals or materials from entering the disease-free region. It involves restriction of contacts between animals from different farms and between farm animals and veterinary hygiene at the farm as well as in the food chain. Identification, registration, health certificates and quarantine rules serve to minimize infection risks during transport and to allow backtracking to the source of occasional outbreaks. Early identification of a listed disease is critical, as well as strict protocols in case of suspicious or proven outbreaks. Below, some measures with particular impact on wildlife are discussed.

2.1.1 Surveillance and reporting

In order to maintain a disease-free status, adequate surveillance and reporting systems are of the essence, in order to minimize reaction time (the time between infection and suspicion by the authorities). Given the consequences of control measures in case of an outbreak, farmers may be tempted to cover up first infections or to postpone reporting. Compliance with veterinary hygiene rules at the farm and during transport are also of the essence. An adequate surveillance system includes control and enforcement of these measures. This is most important for the protection of wildlife too, as outbreaks in a disease-free zone generally occur among livestock.

2.1.2 Monitoring program

An important measure to prevent outbreaks is monitoring wildlife, especially for diseases, including emerging diseases where wild populations can be a source. The quicker the disease is diagnosed in a nature reserve, the quicker control measures can be taken to halt transmission. In addition, keeping track of other variables during and before an outbreak (like herd size, age structure or movement) may give insight in factors that influence the occurrence or course of an epidemic. This may lead to better control policies in the future. Monitoring involves regular observation of live animal populations and the investigation of dead animals that have been found or shot by hunters (Mörner et al., 2002). Ecological processes seem unlikely to be influenced by monitoring.

Although EU directive 92/45/CEE recommends a country scale reporting system to assess the health of game species, only a few countries in Europe have their own wildlife disease 'surveillance' network (Artois et al. 2001). Monitoring of wild and semi-wild herbivores has among other places been implemented in the nature reserve Oostvaardersplassen, in the Netherlands. This monitoring exists of serological research on animals that are found dead

or are shot by gamekeepers (pers. comm. J.A.P. Heesterbeek). Officially recognized institutional networks for reporting mortality and diseases in wild species are nonetheless rare in the EU. For Europe, an informal network based on the members of the European section of the Wildlife Disease Association (EDWA) provides data from 16 country reports. From the available data, Artois concludes that there is an increasing interest in wildlife disease surveillance in Europe (Artois et al. 2001).

2.1.3 Prevention of direct contact

Where domestic animals roam outside, direct contact between wild and domestic animals is possible. Fencing can help to separate domestic and (semi-) wild populations. Such a fence can be placed to isolate a wild population from the surrounding domestic farms or it can be placed around farms to isolate them from the surrounding wildlife, depending on the situation. Contact through the fence is still not prevented, therefore double electrified fencing is recommended. In Zimbabwe, three large game reserves are isolated by double-electrified fencing for the control of FMD. The fences function as a barrier between infected zones (with African buffalo) and FMD-free zones with domestic animals (Sutmoller et al. 2000). This proved not as effective as expected because antelopes were found to jump over the fence. Costs of fencing, capable of keeping wildlife out, are an order of magnitude higher than the costs of regular fences, designed to confine domestic animals to their meadows. (author's remark).
Another disadvantage of fencing is that migration routes of wild animals may be blocked, causing ecological disturbance (Thomas et al. 2003). In the Kalahari Desert, fences caused hundreds of thousands of wildebeest to die during drought, because they were isolated from water sources. Examples of Eurasian species that migrate over long distances are Mongolian gazelle, Saiga antelope, and Reindeer. Mongolian gazelles regularly cross country borders. The border fences do not block migration, but a large number of animals are often severely wounded by barbed wire, leading to high mortalities in populations already weakened from harsh climatic circumstances (Cromsigt 2003).

2.1.4 Movement restriction

Movement of wild animals within and between nature reserves may occur, through deliberate introduction of animals or through spontaneous migration, enabled by the establishment of large, interconnected reserves (ecological network). This contributes to the risk of long distance transmission of diseases. The first type of movement can be regulated but the autonomous movement of animals is harder to control, especially when they do not live in an enclosed area. Fragmentation of habitats is undesirable, and the ecological network is an integral part of the EU nature conservation policy. But during epidemics the connections may have to be blocked to prevent transmission. It would be most achievable by using natural barriers and by blocking corridors in the ecological network. This way, movement restriction may have a minor impact on the ecological processes, especially when it occurs outside the migratory season as a temporary measure.

2.1.5 Preventive vaccination

By vaccinating the population, immunization reduces the number of susceptible animals. The choice of a non-vaccinated disease-free status precludes general vaccination programmes of farm animals. Vaccination of wildlife should be an option, as wild animals commonly do not take part in trade, but OIE only allows it for endangered species individually tagged and strictly confined to zoos or pens. Vaccination does not harm the host, which might be preferable for nature conservation agencies. Yet it disturbs natural ecological processes, as the role of the disease has been ruled out (e.g. regulating the population densities). Intervention in the host-parasite interaction is a disruption of the ecological processes in an ecosystem. 'Wild animals have always fought their own battles

with competitors, parasites, infections and with the rigours of environment and are as they are entirely because of this' (Kirkwood and Sainsbury 1996).
Vaccination of wildlife is not always feasible. They roam free in a large area and for intravenal vaccination they have to be caught or rounded up. This is neither advisable (because of stress to the animals) nor practical. Oral administration in the form of baits seems partly effective in wild boar (Kaden et al. 2000).
A problem with vaccinating is that the distinction between vaccinated animals and infected animals cannot always be made (Van Campen et al. 2001). Vaccination may therefore preclude monitoring. In addition, vaccination may give rise to carriers. These objections don't mean that vaccination is out of the question, but it should be applied with the necessary caution.

2.1.6 Preventive depopulation by hunting

Pre-emptive culling is meant to keep the population of susceptible animals at a low density. Meaning that the population is maintained at the threshold density at which an outbreak would be self-limiting. The frequency of contact between animals is diminished and the total number of susceptible animals decreased.
Nature organizations don't favour this option, because the density is often decreased far below the optimum population size. In addition, the culling of herbivore species is at odds with the main goal of nature conservation, preventing them from playing their part in the ecosystem.
In addition, keeping population density artificially low by hunting has often yielded unsatisfactory results, because of compensatory reproduction and immigration. As hunting alone cannot keep density levels as low as desired, additional measures like contraception should be considered (Artois et al. 2001). No research has so far been done on the feasibility of such a combined method. It may be possible to orally administer contraception to wild boars in the same way as done by Kaden and others (2000). Contraception would disturb the social order and mating cycle of wild boars (pers. comm. M. Montizaan). All attempts to maintain a population at a desired density with artificial means are a form of stewardship representing a great disruption in the ecological processes.

2.2 Control measures

Control strategies involve the closing off of all possible transmission routes as early and as complete as possible, as well as eradication the infection source, either by destroying the pathogen through vaccination or by killing and subsequent destruction of infected animals. It involves rapid identification of the primary infection source, as well as all secondary sources and susceptible animals that could have had contact with an infection source. Within a zone surrounding an outbreak source, very strict quarantine and eradication programmes enter into force. Maximum transmission rate, the minimum reaction time (i.e. time between infection and recognition) and incubation time determine how large the zones have to be. Below, some control measures and their impact on wildlife are discussed.

2.2.1 Isolation and movement restriction

In case of an outbreak, domestic animals are usually stabled or confined to a pen, in order to prevent contact with other animals. To close off transmission routes to or from wild populations during an outbreak, it is desirable to restrict movement of wild animals as well. The feasibility of this measure will among other things depend on the species (is it possible to confine them to a specific area?), as well as the size and landscape characteristics of the zone of restricted movement.

2.2.2 Stamping out

Stamping out is the mass destruction of all infected animals and animals that were potentially exposed to them. The number of animals eventually killed and destroyed maybe small under favourable circumstances, such as early detection, low incubation time, prompt and adequate implementation of outbreak scenarios, low density of susceptible animals and weather conditions that limit survival and transmission rate of pathogens. Sabotage or sloppiness in carrying out control measures in high density regions, or plain bad luck, like for instance a not too virulent strain where infected animals don't show too many symptoms, may cause a massive outbreak and the killing and destruction of millions of animals. It might even lead to the killing of all susceptible animals in a nature reserve. This would, of course, cause a lot of protest from authorities in charge of nature conservations, nature conservation and animal rights NGOs, as well as the public in general. For nature reserve managers, it would mean that their efforts and resources have gone to waste. Even protected species run the risk of being exterminated. Stamping out obviously is the worst-case scenario of any nature reserve manager.

2.2.3 Depopulation by hunting

Reducing the population to very low densities can also be employed as a control measure during an outbreak. Animals are shot until a threshold density is reached at which the infection fades out by itself. The difference with stamping out is that not all infected and in-contact animals have to be killed. The same arguments that are mentioned against stamping out also stand for depopulation, with the addition that depopulation by hunting may not be as effective in wildlife situations as expected. Hunting may cause an infected herd to disperse out of their home range, thereby increasing the risk of transmission to neighbouring groups (Damhuis et al. 2004). Driving prey with dog and noise to a specific place is discouraged in most parts of Europe, because this causes the great disturbances. It is recommended for hunters to stake out at a place that is preferable for animals, such as feeding places.

For wild boars, it is prohibited to shoot guiding sows or sows with piglets, because the social group falls apart and the remaining members disperse in multiple directions over large distances (more than 50km, Damhuis et al. 2004). Young boars and solitary sows without piglets should be targeted. Young boars are the main virus carriers (Kaden et al. 2000). Shooting the sows without piglets may restrict new progeny. It is unclear if this also applies to other ungulates, as other species do not seem to be as intensely studied on this point. It is however plausible that other wild species also move away from areas where they are disturbed.

Another reason for the ineffectiveness of hunting is that hunting might remove recovered immune individuals and favour compensatory reproduction and immigration. This may increase the proportion of non-exposed and therefore susceptible individuals. Additionally, if depopulation does not achieve the threshold density, the infection can remain endemic even at low incidence rates (Artois et al. 2001). Hunting may be allowed only after the epidemic peak has been reached in order to have a synergistic effect with the possible disease-induced reduction of population density and with the establishment of herd immunity (Laddomada 2000). At the beginning of the epidemic, hunting might be useful if carried out in the surroundings of the infected area as buffer zone, provided that the animals are not promoted to move outside the area itself.

2.2.4 Emergency vaccination

Emergency vaccination is often used to create a buffer zone around an epizootic to decrease the number of susceptible animals ('ring vaccination'). This can halt further transmission. In non-vaccination disease-free zones, these animals are often killed later, because of the risk of persistence (unnoticed carrier state in vaccinated animals) (Moennig 2000). Emergency vaccination of wild and semi-wild animals may be difficult, but

worthwhile to achieve protection of the wild animals and diminish their suffering. For endangered species or otherwise valuable stock, it may be the only option to save them. Emergency vaccination however poses the same constraints to the ecological processes as preventive vaccination.

2.2.5 No intervention

An important question is, whether a wildlife disease should be managed at all. Disease is a natural component of ecological systems, and having it run its course may in the long run more beneficial for population health than human intervention. Control measures may not always yield predictable results and wildlife disease managers should attempt to compare the real or expected patterns of infection in the presence and absence of control (Artois et al. 2001).

Wilderness is characterized as uncontrolled by humans and for the most part free of human influence. The main process in an ecosystem is natural selection. 'Survival of the fittest' is the rule that keeps a natural ecosystem healthy. Wild animals therefore need to fend for themselves, without care or disturbance from humans. This care may help the least fit animals, decrease the adaptation of the species to the disease and help to promote one species on the cost of other species.

The risk of non-intervention might be that the pathogen will persist in the wild population. The prevalence of the disease in wild populations that live near areas with livestock may pose a risk for future outbreaks in domestic animals. This has happened with bovine tuberculosis, which was introduced with cattle and has become endemic in many wild species, for example African buffalo populations, possums and ferrets in New Zealand, badgers in the UK and Ireland, bison in Canada and several deer species elsewhere in the

Ethical issues and wildlife

A popular view among conservationists is that humans are not responsible for the welfare of wild animals as long as suffering is unrelated to human activities (Kirkwood and Sainsbury 1996). Disease prevention measures may be important for the conservation of populations when endangered by man (Kirkwood and Sainsbury 1996). However, 'to intervene, to prevent or treat an infectious disease in a wild animal, is to arbitrarily take sides in what is likely to have been a host/parasite relationship that has evolved over a profoundly long time' (Kirkwood and Sainsbury 1996). 'Additionally, disease is important for the maintenance of biodiversity because it influences the species complement within established ecosystems' (Cunningham 1996). Human intervention may not be beneficial for the ecological whole. Intervention might impair population fitness by promoting the survival of the least fit animals in the population. Disease control in wildlife on the base of economical reasons will conflict with 'our moral duty to ensure that ecosystems do not lose vitality and that evolutionary processes promoting biodiversity in the long term continue with as little disturbance as possible' (Klaver et al. 2002).

There is however a dilemma when it comes to exotic diseases that are new to the ecosystem. Is human intervention permissible when it is to eradicate an exotic disease that has been introduced by humans and has a devastating effect on the original ecosystem? In other words, do humans have a responsibility to protect the current ecosystem or do humans have to allow the ecosystem to adapt to the new element (i.e. disease) through the process of natural selection?

Another problem is that semi-wild animals are neither wild nor domestic and there is an ongoing discussion about the extent of human responsibility for the welfare of these animals. Semi-wild animals have always been influenced by human activities and the process of de-domestication is actually the transition from individual care to a focus on animal populations and the role of the animal in the ecosystem. This transition is however not a distinct line. It is hard to assess when individual suffering (by a pathogen) is unavoidable, because the semi-wild population is adapting to fend for itself with natural selection taking its toll or when individual suffering justifies care for the welfare of animals that are under human stewardship. It is therefore necessary to further investigate the ethical aspects of intervening in (semi-) wild populations living in an ecosystem.

world. For some diseases, for example rabies and anthrax, there may be compelling reasons for intervention. (Bengis et al, 2002). In general, when it concerns highly infectious diseases, intervention in wildlife enters the picture. Yet, non-intervention is still the most common course of action in case of outbreaks (Wobeser, 2002)

3 Foot and Mouth Disease

Some of the biggest conflicts between agricultural and nature organizations arise in the control of foot and mouth disease (FMD). It is a highly communicable disease, affecting sheep, pigs, cattle and goats. Severe loss in body weight and a significant decreased milk production of infected animals can bring about economic disaster. These aspects call for severe control measures, although most adult animals survive the disease. Between 1962 and the late '80s, FMD was eradicated by annual vaccination of cattle in most European countries. No other susceptible species were preventively vaccinated. By the end of the 80s, there were no longer any endemic strains of FMD virus in the EU and its adjacent countries. Therefore, vaccination was abolished in the EU as well as its bordering countries. Since 1991, the EU maintains a non-vaccinated disease-free status for FMD.
In spite of the buffer zone in the Thrace region, Turkey appears to be a permanent source of sporadic outbreaks in the Balkans and thus a potential threat to the rest of Europe Russia was not able to maintain the buffer zones in the Caucasian region and the Central Asian States. FMD still persists in the Caucasian region, but the risk of spreading to Russia and Europe is considered limited (Leforban and Berbier 2002).
As demonstrated in recent years, the major risk for Europe is associated with animals and meat imported or smuggled from Asia or the Middle East and possibly Africa, but not from South America (Leforban and Berbier 2002). Lack of control measures during trade is the main cause that FMD can persist in all these areas. In South East Asia for example, the market-driven transboundary movement of livestock is unregulated and vaccination is not used widely as a control tool because of the expense (Gleeson 2002, Perry et al. 2002). Transhumance, the seasonal movement of people and their livestock to regions of different climate, is also an uncontrolled transboundary movement (Macpherson 1995). During the 2001 outbreak, which was mainly located in Great Britain and the Netherlands, hundreds of thousands of domestic animals were killed and behind the scenes scenarios were already discussed to exterminate all susceptible animals, should a FMD outbreak be observed near a nature reserve. Fortunately, this situation did not arise, but in the same period in Mongolia an outbreak of FMD was (incorrectly) attributed to the Mongolian gazelle (*Procapra gutturosa*). Plans were ready to exterminate a large proportion of the gazelle population. Only enormous pressure from different international organizations prevented this from actually happening (Cromsigt 2001). Nature conservationists maintain that preventive vaccination of livestock is a sound and more nature-friendly alternative, albeit at the cost of the coveted disease-free status (Cromsigt 2001). Recently a new directive has been adopted that allows emergency vaccination under certain circumstances. This may have consequences for a country's trade position, which is why countries are reluctant to apply it (pers. comm. E.G.M. van Klink).

3.1 FMD, the disease

Foot and mouth disease (FMD) is caused by viruses of the genus *Apthovirus*. Seven distinct serotypes, with indistinguishable clinical effects have been defined, namely types O, A, C, Southern African Territories (SAT) 1, SAT 2, SAT 3 and Asia 1. Recovery from infection with one serotype will protect from infection with the same serotype, but not from subsequent infection with another serotype. 'FMD virus has a wide host range, an ability to infect in small doses, a rapid replication, a high level of viral excretion and multiple modes of transmission' (Alexandersen et al. 2003). This RNA virus can infect most hoofed animals. The pathogenesis of the virus differs between host species and between virus strains. Common symptoms are lesions in the mouth and hooves. All even-hoofed ungulates are susceptible to FMD.
FMD can cause severe and potentially fatal disease in cattle (*Bos taurus*), pigs (*Sus scrofa*), roe deer (*Capreolus capreolus*) and muntjac deer (*Muntiacus muntjac*). Possible

symptoms are anorexia, shedding of hooves and unwillingness to move or possible lameness. Secondary bacterial infection of foot lesions sometimes cripples animals. In pigs the main symptom is acute lameness (Geller 2001). The mortality rate for mature animals seldom exceeds five percent, but young animals may suffer mortality rates as high as 50 percent (Meyer and Knudsen 2001) or higher. Mortalities of 64% in lambs and 55% in kids have been reported (Sharma 1981).
The disease is more severe in mountain gazelles (*Gazella gazella*), impala (*Aepyceros melampus*), roe and muntjac deer, with consequently higher mortality rates (Gibbs et al. 1975, Thomson et al. 2003). A more benign course with mild and subclinical symptoms seems to occur more often in sheep (*Ovis aries*), fallow deer (*Dama dama*), red deer (*Cervus elaphus*), African buffalo (*Syncerus caffer*) and goats (*Capra hircus*, Sharma et al. 1981, Gibbs et al. 1974, Thomson et al. 1992).
The development of atypical symptoms also depends on the virus strain involved (Sakamoto and Yoshida 2002). According to Haigh and others (2002) couldn't find any reports of FMD in European bison (*Bison bonasus*). Yet there have been FMD outbreaks among bison populations in Poland in 1946, 1951 and 1952, although during these outbreaks only few bison died (pers. comm. J. Kita). Symptoms in bison were similar to cows (pers. comm. J. Kita).
Goats, camels (*Camelus bactrianus*) and water buffalo (*Bubalus bubalis*) are frequently mentioned as susceptible hosts, but research data on the epidemiology of FMD in these species are very limited (Samara and Pinto 1983, Gomes et al. 1997, Barnett and Cox 1999, Kitching and Hughes 2002). Notably, camels showed clinical signs during the epidemic in Mongolia (Sakamoto and Yoshida 2002) and they are frequently vaccinated in Egypt (Aidaros 2002). No antibodies where found in camels after an epidemic in Kenya, whereas eland (*Taurotragus oryx*) did (Paling et al. 1979). Other species that can contract FMD are llama, blackbuck, sambar, nilgai, Tibetan black bear, yak and spotted deer (Kar et al. 1983, Musser 2004), but research data is also lacking.
There is no data on the origin of FMD. In 1545, it was first described in cattle in Italy. The disease was common in France, Germany and Italy in the 17th and 18th century. It did not spread to Britain until 1839. The early history of FMD in Asia and Africa is not known (Thomson et al. 2001). Nowadays, the disease is endemic in large parts of Africa, Asia and South America (Alexandersen et al. 2003). However, the only species known to be a maintenance host is the African buffalo, which means that FMD persists in some populations of buffalo without input from external infection sources. The SAT types are unique to Africa and have an intimate and probably ancient association with African buffalo. African buffalo are the usual source of SAT-type infections for domestic livestock and wild ungulates in southern Africa (Bastos et al. 2000). Persistently infected African buffaloes in the wild constantly generate variants of SAT 1 and SAT 2, causing the immune response to always be one step behind (Vosloo et al.1996). This complicates the eradication of the disease through immunization. Until now no major outbreak with SAT types have occurred in Eurasia, although sporadically infections in the Middle East have occurred (Aidaros 2002, Leforban and Gerbier 2002). This study mainly focuses on the situation in Eurasia, so the situation with endemic FMD in African buffalo is not discussed here.

3.2 Primary outbreaks

Primary outbreaks are probably inevitable (Leforban, 2002). Before the 50s FMD used to ravage Europe at intervals of 5-10 years. Between large epidemics, the disease continued to occur, sporadically or endemically, in regions with high animal densities or movements. From 1951, vaccination was widely applied, which led to a gradual decline of the disease. New outbreaks tended to occur from the Near East, with Turkey the most vulnerable region. In Asian Turkey, Anatolia, FMD is still endemic. Preventive vaccination of cattle only proved sufficient to eradicate the disease from the European continent. From the ban on vaccination in 1991 till 2000, 21 primary outbreaks occurred, mostly from the East with

type O strains, endemic in Asia. Most of these outbreaks were traced to illegal trade or transports of infected animals and products. Illegal immigration also counted for some outbreaks. Where the primary source was unknown, there was strong suspicion of illegal activities. One outbreak in Russia was due to an escaped laboratory virus, type A. The 2001 outbreak in the UK originated on a pig farm and has been attributed to illegal swill feeding. It was spread to the continent by the trade of sheep without apparent symptoms, before the FMD outbreak in the UK came to light (Leforban and Gerben, 2002). In primary outbreaks in the EU, illegal activities always lie at the root, as regular trade in animals and animal products, even from countries without the FMD-free status does not constitute a risk (Sutmoller and Casas Olascoaga, 2003. Leforban and Gerben, 2002).

3.3 Direct transmission routes

When infected and susceptible animals are in close proximity, the aerial transfer of droplets and droplet nuclei is probably the most common mode of transmission over short distances (Alexandersen et al. 2003). Direct contact between and within different animal species have frequently been reported to cause FMD infections. For example, transmission has occurred between deer, sheep and cattle (Gibbs et al. 1975, Gibbs et al. 1975b), between cattle, water buffalo and goats (Dutta et al. 1983) and between sheep and pigs (Amass et al. 2003).

Bouma et al (2004) found that direct transmission from one separately held calf to two other separately held calves could not be demonstrated with a type O virus strain. But in a recent experiment by Karin Orsel and others, FMD transmission did occur between calves housed together in a group. In this case, vaccination was able to significantly decrease transmission (pers. comm. K. Orsel).

Contact transmission also occurs through physical contact with infected excretions or secretions, including vesicular fluid or vesicular epithelium containing relatively large amounts of FMD virus. Donaldson et al (1982) found samples of FMD positive milk containing a maximum of 106.7 TCID50 /ml and blood samples with a maximum of 106.7 TCID50 /ml, both from cows. Gibbs et al (1975) found in deer that the amount of virus in the blood samples and in the samples of the respiratory tract were similar to those recorded for sheep and cattle in the same experiment during the course of the infection (table 1).

Table 1. Excretion doses of different species via respiration, blood and faeces for FMD.

Infected species	Respiratory tract	Viraemia (blood)	Faeces
Cattle	$10^{3.9}$ [a]	$10^{6.0}$ [b]	$10^{4.9}$ [f] $>10^{5.6}$ [g]
Sheep	$10^{4.0}$ [a] $10^{4.3}$ [e]	$10^{5.0}$ [b]	$10^{2.7}$ [f] $10^{3.2}$ [h]
Pigs	$10^{6.3}$ [a] $10^{6.4}$ [c] $10^{5.8}$-$10^{6.1}$ [d] $10^{6.1}$ [e]	$10^{7.0}$ [b]	$10^{2.9}$ [f, g]
Goats	n.d.*	n.d.	$10^{2.5}$ [h]
Roe deer	$10^{4.0}$ [a]	$10^{6.0}$ [b]	n.d.
Red deer	$10^{4.0}$ [a]	$10^{5.0}$ [b]	n.d.
Fallow deer	$10^{3.7}$ [a]	$10^{5.0}$ [b]	n.d.
Sika deer	$10^{4.0}$ [a]	$10^{6.0}$ [b]	n.d.
Muntjac deer	$10^{4.0}$ [a]	$10^{7.0}$ [b]	n.d.
Camel	n.d.	n.d.	n.d.
Water buffalo	n.d.	n.d.	n.d.
European bison	n.d.	n.d.	n.d.

* No data
[a] $TCID_{50}$ /animal/30min (Gibbs et al. 1975)
[b] $TCID_{50}$ /ml (Gibbs et al. 1975)
[c] $TCID_{50}$ /pig/hour (Alexandersen and Donaldson 2002)
[d] $TCID_{50}$ /pig/24h (Alexandersen and Donaldson 2002)
[e] $TCID_{50}$ /pig/24h (Donaldson et al. 2001)
[f] ID_{50} /gr (Sellers and Parker 1969)
[g] ID_{50} /gr (Parker 1971)
[h] $TCID_{50}$/gm (Sharma 1981)

The amount of virus from vesicles was usually in excess of 108.0 TCID50 /g tissue for each of fallow, red and roe deer (Forman and Gibbs 1974).
FMD virus has also been found in the faeces of infected animals, but the infectivity of excreted virus may decrease rapidly due to inactivation. The inactivation rate of FMD virus varies with the pH, temperature and ambient humidity. For pigs there is a consistently low level of virus in faeces (Parker 1971) and cattle seem to excrete substantially higher amounts of virus than sheep and pigs (Sellers and Parker 1969). Unfortunately, there is no data for the virus content in faeces of other herbivore species.

Sexual contact and mother-foetus

Transmission via sexual contact and from mother to foetus is an under-researched topic. There are just a few indications that these routes are indeed possible in cattle. For example, FMD virus is easily transmitted by the semen of bulls (Wentink et al. 2000). And there is a high probability that FMD infected cattle can yield high percentage of contaminated ova and embryos (Sutmoller and Wrathall 1997). This route is improbable in well-vaccinated cattle and carriers. In the experiment, washing the embryos was sufficient to remove the virus, meaning that the embryos themselves were never infected. A foetus may become infected with FMD in a later stage of gestation or when it comes into contact with contaminated maternal material (during or after birth). There is no transplacental transmission.
FMD virus has been found in the uterine lumen and oviduct of cows during the clinical stage of the disease (Sutmoller and Wrathall 1997). So bulls might get infected during sexual contact. The relevance for these transmission routes, however, is questionable. As mentioned earlier, this disease is highly contagious in direct contact and the contribution of these two routes to an FMD epidemic is probably negligible.

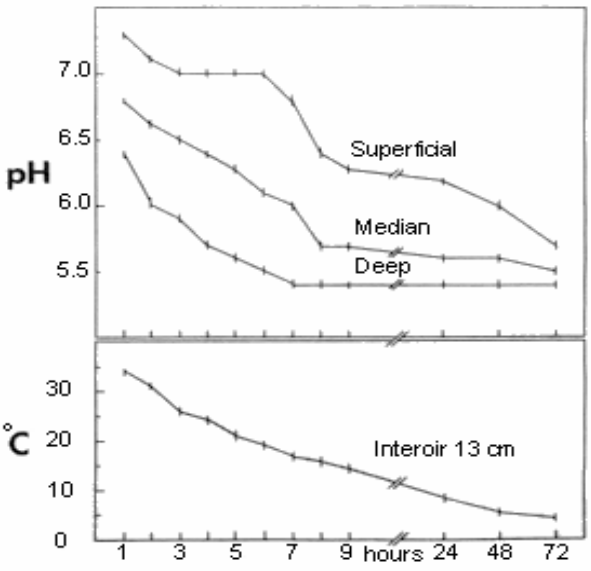

Figure 3. The pH and interior temperature changes in muscles of steer carcasses during maturation at 4 ºC. The superficial curve indicates the highest pH readings obtained in the superficial areas of the muscles and the deep curve represents the lowest readings from the deeper areas. The median curve represents the midpoint of observed pH values and is not the arithmetical mean. The temperature was measured 13 centimeters inside the muscle tissue (after figure 1, Cottral et al. 1960).

Dead animals

Post-mortal chemical changes reduce FMD infectivity. The acidity produced in carcass meat during rigor mortis in cattle will inactivate FMD virus. Cottral et al. (1960) measured the pH and interior temperature changes in muscles of steer carcasses during ripening at 4 ºC (figure 3). FMD virus can survive in the pH range 6 to 9. The superficial curve indicates the highest pH readings obtained in the superficial areas of the muscles and the deep curve represents the lowest readings from the deeper areas. The median curve represents the midpoint of observed pH values and is not the arithmetical mean. It appears that the threshold value where FMD virus becomes unstable is approximately reached after 3 to 48 hours. This means that the muscles tissue of dead cattle is probably free of active virus within 3 days at a temperature of 4 ºC. At higher temperatures this period of active virus in

dead bodies will be shorter as inactivation rate increases with temperature. Acidity of muscle tissue after rigor mortis in pigs seems to be variable, but has not been examined in detail for mutton (Alexandersen et al. 2003).
This doesn't imply that carcasses older than 3 days pose no risk factor at all, for the pH in bone marrow, lymph nodes and certain organs and offal does not decline (Donaldson 1987). Infective virus therefore can be found in these materials for an extended period of time. In carcasses that were stored at 4 ºC, active virus was found in rib bone marrow after 73 days and in lymph nodes and blood after 60 days (Cottral et al. 1960). Susceptible animals may ingest these parts of an infected carcass and subsequently become infected. However, a quantitative assessment of the virus was not executed (Cottral et al. 1960). Sharma (1981) reports that all tissues and organs of sheep and goats contain high quantities of FMD virus and could be potential disseminators of virus. Without a detailed course assessment after death, the risk of infection in time cannot be quantified.
Not only ingestion, but merely direct contact with dead bodies may induce an infection, through inhalation of the virus by sniffing the exterior of the body. Gailiunas and Cottral (1967) found that FMD virus remained infectious on the hide of steers that were killed after the development of clinical signs, for up to 90 days at 15 ºC and for 352 days at 4 ºC. In this study, the infectivity was demonstrated by inoculation, so it is unclear whether under natural conditions (i.e. sniffing the carcass) a susceptible animal could also get infected. Dehydration of the carcass may also reduce the survival duration.
Animal products from infected animals constitute an infection risk, especially when not cured at a pH lower than 6. Such animal products are, by definition illegal, as OIE prescribes slaughterhouses to subject carcasses to a maturation period of 24 hours at pH 6 and deboning carcasses from vaccinated disease-free regions. Products from zones, where an outbreak has occurred need extensive treatment in order to destroy the virus (OIE, 2004). Products from vaccinated animals or from carrier animals do not constitute a significant risk, because the antigens keep the infectivity low (Sutmoller and Casas Olascoaga, 2003).

3.4 Indirect transmission

A lot of policy measures are taken to prevent direct contact between domestic populations and between wild and domestic populations to diminish the spread in case of an outbreak. But with FMD, indirect transmission is also an important possible route. This is much harder to control than direct contact between animals.

3.4.1 Transmission via the abiotic environment

Conditions conducive to survival of FMD virus are high humidity, low temperatures and neutral pH. Pollutants and sunlight were not found to be of influence (Donaldson and Ferris 1975). For stable aerosols the temperature needs to be below 33 ºC (Gloster 2004). The actual value of the relative humidity depends on the virus strain involved (Donaldson 1972). The lowest value reported is 55% for a type A strain, but at a relative humidity above 70% each virus strain is certainly stable. Airborne virus is generally inactivated within a few hours when relative humidity falls below 55-70% (Donaldson 1972, Gloster et al. 1982). Mead (1979) however says that FMD virus is able to remain viable for long periods when dried on material around the farm; unfortunately no data was given on which he grounded this position. Drying will inactivate most but not all virus. The drying of fluids or organic material containing virus will also inactivate a relatively large proportion, but surviving virus may be more stable after drying, thereby creating a trace residue of infectious virus (Donaldson 1987). The virus can survive in a pH range from 6 to 9, especially at lower temperatures, but becomes increasingly unstable at pH values outside that range (Parker 1971, Thomson et al. 2001). The virus is most stable within the pH range of 7.0 to 8.5 (Bachrach et al. 1957, Parker 1971).

Airborne

Under certain climatic and meteorological conditions, short-distance droplet nuclei transmission may be extended to long-range airborne transmission. Distances of 60 km over land (Hugh-Jones and Wright 1970) and 250 km over sea have been reported during FMD outbreaks (Donaldson et al. 1982, Gloster et al. 1982). Airborne transmission was conjectured after exclusion of human interference, migrating birds and because of the relation between wind direction and dispersal.

This possibility of long distance transport over sea is probably due to the fact that air above sea has a high humidity and that the air is more likely to be stable over sea than over land (Gloster et al. 1982). The host's respiratory tract was determined as the main source of airborne FMD virus (Donaldson 1986).

Amplifiers, indicators and maintenance hosts

The role of hogs in airborne transmission has been characterised as FMD *amplifiers*, because they excrete a lot of virus by exhalation. Sheep are implicated as *maintenance host*s, because they are prone to subclinical infection, whereas cattle are seen as FMD *indicators* because they are the first to show lesions and other characteristic symptoms (Sellers and Parker 1969). Forman and others (1974) and Gibbs and others (1975) found that the amount of virus excreted as an aerosol during the course of the infection in different deer species was similar to those recorded for sheep and cattle. In general, deer are compared to sheep in their epidemiological role. This is however very poorly substantiated by experimental data. It is more an intuitive assumption, because they are both small ruminants (pers. comm. A. Dekker).

Donaldson and others (2001) modelled the effect that different number of animals at the source would have on the distance that a plume could travel and still be infectious for different species. They did this for pigs, sheep and cattle and used the meteorological conditions that are most favourable for airborne dispersion of FMD virus, namely; a constant wind direction, a wind speed of 5m/s, a high atmospheric stability, no precipitation and a relative humidity above 55%. They calculated that 1000 pigs could infect cattle at a distance of 6 kilometres downwind, sheep at 2 kilometres and pigs at less than 0.2 kilometres, whereas 1000 cattle or sheep could infect cattle at only 0.7 km and sheep at 0.2 km. These different distances can be explained by to the differences between species in virus excretion (table 1) and minimum doses needed for infection (table 2).

The parameters for this model were chosen to represent a worst-case scenario (for policy-makers), so the distance of transmission is supposed to be less under natural conditions. However, previous outbreaks have indicated distances far greater than 6 km (Hugh-Jones and Wright 1970, Sellers and Gloster 1980, Gloster 1982). Perhaps these reports represent exceptional events, but they show that transmission over larger distances is a very real possibility. Ninety percent of the secondary outbreaks occur within 10 kilometres of the source (Hugh-Jones and Wright 1970). The remaining 10 percent overland include distances up to and even greater than 60 kilometres. This shows that it may be difficult to predict accurately what will happen under natural conditions. The situation is more complex because animal variability and daily or hourly fluctuations in virus excretion were not included in the model and topographical conditions are more important with longer distance (Gloster et al. 2003). All in all, modelling has its limitations.

Another source mentioned for infectious aerosols is the mass destruction by burning of infected domestic animal bodies. In the FMD outbreak of 2001 in Great Britain, there were two cases, where a holding under the pyre plume became infected on a date consistent with the pyre set to fire. The estimated probability of infection by the pyre was calculated from a model. Based on this estimation, infection risk for the holding, which became infected under the plume, was lower than that for equally susceptible farms that were even closer to the pyre, but still did not become infected at the time of the pyre plume. From this it was concluded that the pyre was an unlikely source for the infection of the holdings. Notably, in the calculation, the infection source was assumed to be a worst-case scenario

with a total of 106.5 TCID50 FMD virus excreted during a period of 3 hours, regardless of the pyre size (Jones et al. 2004).

Soil and water

Usually soil and surface water are contaminated by infected animal excretes and other infected organic material. The content of ruptured blisters also ends up in the environment and may be an important source, because the amount of FMD virus is several orders of magnitude higher than for example manure (pers. comm. Aldo Dekker). There is however limited data on the survival of blister excretes in the environment. Given this lack of data and the small amounts of blister excretion, we'll assume that survival of the virus in animal faeces is the second most important source. This makes transmission by soil and water the second route of indirect transmission in order of importance.
Temperature and pH play an important role in the survival of FMD virus. For example, FMD virus can survive in bovine slurry for at least 70 days at 17ºC and for 84 days at 4ºC. The concentration of virus in cattle slurry after 56 days at 4ºC is 103.5 to 104.25 TCID50 /animal and at 17ºC is 103.0 to 103.75 TCID50 /animal.
In pigs slurry with an initial concentration of 104.8 TCID50/50µl, FMD virus can survive for 2 weeks under 20ºC and more than 4 weeks under 5ºC (Haas et al. 1995). These experiments are however done with liquid manure, so the humidity is optimal during the whole experiment. These data may be comparable to survival in water, but not on land. There survival may be lower, due to dehydration by sunlight and ambient humidity. Parker (1971) recovered virus from cattle and pig faeces, but he used a different method that make the data not directly comparably to the data with liquid manure (see table 1).
The review of Alexandersen et al (2003) reports survival times of FMD virus on different organic material. It can survive up to 20 weeks on hay or straw, up to 4 weeks on cow's hair at 18 to 20ºC, up to 14 days in dry faeces, up to 39 days in urine, up to 6 months in slurry in winter, up to 3 days on soil in summer and up to 28 days in autumn. Bartley and others (2002) give a more detailed review on the survival of FMD in animal excretions and fomites. They found for example, that FMD virus survived longer when the virus was located beneath the soil surface (more than 2 days) than on the soil surface (less than 2 days) and longer under leaves (19-30 days), then when it was situated on the soils surface (less than 5 days) or on plant stems (less than 24 hours).
Unfortunately, the decay rate of FMD virus under these different conditions cannot be estimated, because the initial amount of virus is not given for the majority of the experiments (Bartley et al. 2002, Alexandersen et al. 2003). Data on the actual survival of FMD virus in surface water under experimental or natural conditions is lacking too.
Cattle excrete much more virus by faeces than sheep or pigs (Sellers and Parker 1969, Parker 1971), while goats seem to excrete less than sheep (Sharma 1981). Unfortunately, there is no data available on the amounts that feral animal species excrete. Cattle faeces seem to be a potent source for contaminating the environment. In conclusion, transmission via soil and water is a likely route, but little can be said on the height and duration of this risk.

3.4.2 Transmission via animal vectors

Birds are often implicated as vectors of FMD. Especially when the primary outbreak is on an island like Great-Britain, migrating birds are suspected, because they travel long distances and may have an FMD-infected area. Mead (1968) describes the ways in which birds would be able to pick up contaminated material and the hundreds of miles that they can cross in one hop. In order to effectively transmit an infection however, it is necessary that a large number of birds be contaminated with high amounts of virus (Gloster 1982).
From the research on long distance transmission during a FMD outbreak the dispersal pattern appears often correlated to the direction of the migrating birds in that same period. But Mead's (1968) review on different case studies on the occurrence of primary outbreaks of FMD and bird migration concluded that the influence of migrating birds as

vectors over long distances could be dismissed. Gloster and others (1982) also discuss the possibility of birds acting as mechanical vectors for transmitting FMD virus over sea and added more recent case studies to the discussion. They too concluded that birds were unlikely to spread the virus over long distances. Even evidence for transmission over short distances is lacking. Mead (1968) was able to report from personal communication that the FMD virus could be recovered after 36 hours in the digestive tract of starlings (*Sturnus vulgaris*). It is unclear how much virus was recovered and what the probability is that such a bird infects a susceptible animal.

Long distance spread of FMD by human vectors has been discounted by reviewing case studies of long-distance outbreaks (Gloster et al. 1982). Amass and others (2003) showed that humans might act as mechanical vectors over short distances. Personnel were contaminated by examining infected pigs. They transmitted the FMD virus to susceptible sheep and pigs. Different decontamination measures were taken and only the whole-body decontamination could prevent transmission to sheep. For pigs hand washing and clean outerwear were sufficient to prevent transmission. Human movements were identified as the cause for the transmission of infection affected cattle to a nearby zoo in India (Sarma et al. 1983).

Sellers (1971) mentions that virus had been found in rat faeces and urine. In order to attain sufficient virus for oral infection, the faeces from 160 rats would be required. The animal species thus infected however was not mentioned. He also suggested that contamination of dust by rat faeces or urine might lead to infection by inhalation. Since then, there are no further indications that rats are indeed credible animal vectors for FMD. The normal home range of a rat is only 50 meters, although rats have been known to travel 2 to 3 miles (3 to 5 km) a night to forage in farmers fields at harvest time (Maust 2002, Ballenger 2001). Contamination with FMD may occur when their food is contaminated, supposedly infected carrion (Ballenger 2001).

It is possible to infect hedgehogs and they have been shown to transmit FMD virus after experimental infection. They can eat fresh carrion and may travel 3 km while foraging within a home range of 24 ha in a single night (Martin 2004). In addition, hedgehogs got infected by inhaling the virus that was exhaled by an experimentally infected hedgehog (Donaldson 1979). It is however unclear if exposure to other species under natural conditions would also cause an infection and if an infected hedgehog excretes enough virus to infect other susceptible species.

No further animal vectors have been investigated in the literature. Sutmoller and others (2000) speculate on lions and other scavengers that act as mechanical carriers of virus after the kill of viraemic buffalo calves. Experimental data is however lacking. No mention has been made about the possible partaking of pets, insects or other possible vectors.

3.5 Infection dose

Of the different infection routes, the most is known about the respiratory route. There is only sporadic data on the oral route and no data at all on the minimum infection dose (MID) for infection via the venal route. Infection via the venal route is difficult to simulate under experimental conditions, whereas the contribution to the transmission of FMD virus compared to the other routes is probably negligible. The chance that an animal is infected by direct contact with its blood and not first by digestion or inhalation seems very slim. The respiratory route is generally considered the most common infection route (Thomson et al. 2001), because infected animals commonly excrete the virus by exhalation. The risk of inhaling aerosols is more likely to cause an infection in certain species, due to different MIDs.

As can be seen in table 2, the MID for pigs via respiratory infection route has not been determined well. One of the reasons is that previous studies used artificially generated aerosols to assess the MID for pigs. ‘The pathogenesis of FMD virus in animals exposed to artificially generated aerosols is however markedly different from that in animals exposed to natural aerosols’ (Alexandersen et al. 2002). Furthermore the assay used was

less sensitive for quantifying FMD virus than the one used nowadays. Even in a later study, the MID could not be determined, because the authors were unable to produce natural aerosols containing high enough doses of FMD virus (Alexandersen et al. 2002). The minimal infection dose to infect 50 percent of a population (MID50) has not even been quantified accurately for pigs (table 2). These results in fact reflect the low probability that aerosols can infect pigs. Alexandersen and Donaldson (2002) showed that 650 TCID50 in 5 minutes was not enough to infect ten pigs, meaning that this dose might be near the threshold level for the immune response to clear FMD in pigs.

Table 2. The minimum infection doses of different species by different infection routes for FMD.

Susceptible species	Oral route	Venal route	Respiratory route	
	MID	MID	MID	MID50
Cattle	$10^5 – 10^6$ [a] $10^{5.8}$ [b]	n.d.*	10 [c] 25 [e]	5 [d]
Sheep	n.d.	n.d.	10 [e, f]	7 [d]
Pigs	$10^4 – 10^5$ [a] $10^{3.9}$ [b]	n.d.	n.d.	$>10^3$ [g] >800 [c]
Goat	n.d.	n.d.	20 [h]	n.d.
Roe deer	n.d.	n.d.	n.d.	n.d.
Red deer	n.d.	n.d.	n.d.	n.d.
Fallow deer	n.d.	n.d.	n.d.	n.d.
Sika deer	n.d.	n.d.	n.d.	n.d.
Muntjac deer	n.d.	n.d.	n.d.	n.d.
Camel	n.d.	n.d.	n.d.	n.d.
Water buffalo	n.d.	n.d.	n.d.	n.d.
European bison	n.d.	n.d.	n.d.	n.d.

MID = Minimum infection dose to infect nearly all animals * no data
MID_{50} = Minimum infection dose to infect 50% of population
[a] $TCID_{50}$ (Alexandersen 2003)
[b] ID_{50} (Donaldson 1987)
[c] $TCID_{50}$ (Donaldson et al 2001)
[d] $TCID_{50}$ (Alexandersen et al. 2002)
[e] $TCID_{50}$ (Donaldson 1986)
[f] $TCID_{50}$ (Gibson and Donaldson 1986)
[g] $TCID_{50}$ (Alexandersen and Donaldson 2002)
[h] $TCID_{50}$ (Kitching and Hughes 2002)

The published MIDs for FMD virus for sheep and cattle are 10 and 25 TCID50 by natural aerosol (Donaldson 1986). However the calculations of these MIDs were based on the number of clinically affected animals. In order to compare the sensitivity of cattle and sheep with pigs, these MIDs for cattle and sheep are converted to MID50 with the addition of the number of subclinically-infected animals. Alexandersen and others (2002) re-analyzed the data and calculated values of 5 and 7 TCID50 as MID50 for cattle and sheep, respectively.
Pigs have shown to be relatively resistant to infection by airborne FMD virus compared to sheep and cattle (Donaldson and Alexandersen 2001). They however seem more susceptible to infection via the oral route, then sheep and cattle (Alexandersen 2003).
There is no direct data on the epidemiology of FMD in goats. The minimum infection dose reported on goats by Kitching and Hughes (2002) does not seem to be based on experimental data. As in all the literature involving goats Sharma (1981), Barnett and Cox (1999) also Kitching and Hughes (2002) seem to consider sheep and goats as equals. This assumption is however not substantiated by any data. Goats are no substitute for cattle, because goats clear the virus much faster than cattle (Sharma 1981). The main focus of these articles is on sheep.
Unfortunately, there is also no data on the minimum infection dose for deer or other wild animals for either infection route. The course of the disease in deer seems comparable to sheep and cattle (Gibbs et al. 1975). For example, cattle that were in contact with the small deer species (roe, muntjac and sika) reacted at the same rate to infection as the

conspecific deer. Similarly, the bigger fallow and red deer developed subclinical FMD and the appearance and distribution of lesions were similar to those in sheep (Forman and Gibbs 1974), so these deer species resemble sheep in their reaction to FMD virus infection. In addition, sheep and cattle have the same levels of virus excretion and the same infection doses. Because of this, in absence of real data, it might be reasonable to suppose that MID in deer is of the same magnitude as in sheep and cattle.

3.6 Carrier state

Alexandersen and others (2003) report in their review the maximum duration of carrier states in different species. This is 3.5 years for cattle, 9 months for sheep, 4 months for goats, 5 years for African buffalo, 2 months in water buffalo. Some deer species may become carrier, but the duration of the state has not been determined. In the experiment of Gibbs and others (1975) red deer, fallow deer and sika deer were virus carrier during the duration of the experiment (28 days). Pigs are considered to not be FMD carriers, although there seems to be recent research that suggests that pigs too can become carriers (Musser 2004). The FMD carrier state is always accompanied by virus antibodies in serum and OP fluid (Wittmann 1990)

The percentage of animals that become a carrier under experimental conditions is variable but averages around 50%. The amount of virus from the respiratory tract is usually low in carriers, 10-100 TCID50/ml (Alexandersen et al. 2003). Persistence of virus in pharynx was found for fallow deer, sika deer, sheep and cattle at levels of 104.0 TCID50 /sample and occasionally in red deer at levels of 102.0 TCID50 /sample (Gibbs et al. 1975). The carrier state for smaller deer, like muntjac and roe deer was not shown in the experiment (Gibbs et al. 1975).

Carrier animals are not very infective. Attempts to demonstrate transmission from known carrier cattle or sheep to susceptible animals held in close, direct contact under controlled experimental conditions have all failed (Wittmann 1990, Alexandersen et al. 2003). There is evidence in African buffalo that although transmission from carrier to susceptible animal is inefficient it does occasionally happen (Dawe et al. 1994, Hedger and Condy 1985), but only with prolonged and intimate contact (Thomson et al. 1992, Thomson 1995, Vosloo et al. 1996). In this experiment carrier and susceptible animals were kept for months in the same pen.

Vaccination has shown to protect animals against disease development and reduce contact transmission in infected animals (Salt et al. 1998, Cox et al. 1999). Virus replication is thus limited, but not stopped. The transmission is reduced to minimum levels. Vaccinated pigs and sheep were unable to transmit FMD to the in-contact susceptible pigs or sheep, respectively. The aerosol produced by these pigs had a maximum of only 2.1 TCID50/ml (Salt et al. 1998). No data was presented on the virus excreted by vaccinated sheep (Cox et al. 1999). Vaccines have not been tested on deer and bison, so it is unclear if this preventive measure could be effective for these species (Haigh et al. 2002).

3.7 Ranking transmission routes

For primary outbreaks, illegal trade or transport of animals and animal products is the most likely source. Also, the ban on swill feeding proved its rationality.

Direct contact is the most important route of transmission for FMD virus, commonly by exhaled respiratory nuclei. Contact with ruptured blisters may facilitate the infection of animals that are near an infected animal. Transmission seems to only be dependent on the duration of exposure. A very short period of exposure may not cause the virus to invade the body of a host and to infect a host's cell and replicate. Sheep and goats are important in this type of transmission, because infection often doesn't show.

The second most important transmission route is the formation of aerosols from these respiratory nuclei. The focus of research is mainly on airborne transmission, because this route can cross long distances and make an epidemic difficult to control. Theoretically, the

biggest risk comes from infected pigs in the winter, as pigs excrete a lot of airborne FMD virus and the conditions for virus survival are more favourable in winter on the base of the relative humidity. That is, when pigs become infected during an outbreak, which is not necessarily the case, because they are less susceptible to the respiratory infection route. Cattle and sheep and goats are very susceptible to respiratory infection. Deer, sheep and bovines presumably excrete 100 times less airborne virus than pigs and wild boars but may cause infected aerosol formation in sufficient densities. The susceptibility for deer may be comparable to cattle and sheep as the virus excretion is also comparable. No assessment can be made for the remaining feral animals that are susceptible to FMD.
The third most important transmission route might be the survival of FMD virus in the environment, i.e. soil and water, as the virus can remain viable for prolonged periods. This depends on various environmental conditions and it has never been identified as a route of transmission during an epidemic. The rest of the transmission routes are hard to rank according to importance. The survival of FMD in dead animals depends on the temperature and the chemical changes during rigor mortis. It is unclear how these chemical changes take place in each host species and how infectious the meat may be. Hedgehogs are the only identified animal vectors that may act as a reservoir for FMD, but it is unclear if they can be infected by other species under natural circumstances and subsequently transmit it to other species. For this reason, the role of hedgehogs as vector remains obscure. Humans, birds and rats may mechanically transmit FMD virus. Rats and birds may produce contaminated faeces. Humans and birds do not seem to facilitate long distance transmission.
The role of animal vectors is however complex and the available data are too limited to assess their feasibility. No research is done on the occurrence of sexual transmission under field conditions. This route may be irrelevant, compared to direct contact transmission. The FMD carrier-state implies that FMD virus is mainly restricted to the respiratory tissues, thus this route is even less relevant for carriers. Trans-placental transmission from mother to foetus does not seem to occur.
For pigs and wild boars, oral infection is the most likely route of infection. Determination of the minimum infection dose via the respiratory route may not be possible with the current techniques, because it is so high.
The outbreak of FMD in the UK in 2001 is a good example of the roles that different species can play in an epidemic. This outbreak was the first since 1968 and the primary infection was in a pig farm, probably caused by swill feeding (the exact source still unknown, Leforban and Gerbier 2002). Airborne virus from these pigs infected nearby cattle and sheep. Luckily, the virus strain caused the infected pigs to emit less airborne virus than expected from previous outbreaks and thus spread by airborne transmission was over much shorter distances (1 to 9 km, Gloster et al. 2003). The sheep only showed mild symptoms. This fact, combined with the decreased vigilance after so many years without FMD outbreaks, caused the disease to pass unnoticed. These sheep were taken through a number of markets, resulting in a major spread that reached Ireland, France and the Netherlands.

3.8 Interface of domestic and (semi-) wild animals

Above, a ranking is given of the routes by which FMD virus is transmitted. By knowing this, it may be possible to assess the transmission risk when either wildlife or livestock is the source of infection. The risk that domestic animals infect wildlife and vice versa depends on opportunities for direct contact, presence of vectors and in absence of both, on the distance between the populations. While airborne transmission from domestic animals to wildlife is perfectly possible, the reverse may be an unlikely event. Wild boars (the amplifiers of airborne spread) live solitary or in groups of 6 to 20 individuals with a median density of 3 wild boars per 100 ha (Cromsigt et al. 2001). Although modelling by

Donaldson and others (2001) showed that 10 infected pigs were (in theory) sufficient to infect cattle at 0.5 km distance, long-distance aerosol transmission in the order of kilometres from free-living wild boars is quite unlikely. Not only because of the low density of boars, preventing rapid spread of infection among the boar population, but because infection in swine occurs most likely by direct contact with an infected animal or by eating infected meat, not by inhaling infectious aerosols.
Semi-wild cattle and sheep, as well as some deer species, due to their tendency to flock together in herds, might become a source of infective aerosols. The small roe deer and muntjac deer live solitary or in small groups in forest areas (Forman et al. 1974, OmniShift 2004). Roe deer have their own territories and the median density is 15 individuals per 100ha (range 0 to 35 deer per 100ha, Cromsigt et al. 2001). Only in winter do they form herds of 10-15 individuals in open fields (pers. comm. M. Montizaan). The risk from the bigger fallow and red deer may be more substantial. They tend to herd in open woodland and graze with cattle and sheep (Forman et al. 1974, Gibbs et al. 1975). Female red deer live in groups of 2 to 50 individuals, whereas male red deer generally live separate from the females in groups of 2 to 40 individuals. The herd size of red deer depends on the food availability. In forest areas, the group size is commonly between 5 to 10 individuals (pers. comm. M. Montizaan). The median density of red deer is approximately 4.9 individuals per 100ha (Crowmsigt et al. 2001). Fallow deer commonly gather in herds of only 4 to 5 individuals. But in good feeding areas 70 to 100 deer may gather (OmniShift 2004).
Separately, these species do not seem to represent a risk for the formation of aerosols that can disperse over long distances, but the congregation of all these species in open areas during the winter (food shortage) or during periods of concentrated food availability may pose more substantial risk for airborne transmission from wildlife to domestic animals. The density of all susceptible animals under such conditions needs to be assessed. For now, we can observe that the nature reserve Oostvaardersplassen in the Netherlands contains considerable higher herbivore densities than nature reserves elsewhere in Eurasia, but still considerably less than domestic animals in the meadow. The total populations of Heck cattle, red deer and wild boar in 2004 were respectively 681, 1430 and zero (pers. comm. F de Roder). Thus about 2000 susceptible animals may inhabit the reserve, which has a surface of 5600 hectares. In theory, 1000 cattle or sheep could infect cattle at 0.7 km and sheep at 0.2 km (Donaldson et al. 2001). Even when these animals flock together, the herd won't be large enough to transmit aerosols to domestic population, as 1.4 km may not even be sufficient to transmit an aerosol outside the borders of the nature reserve. Moreover, it is unclear if all susceptible animals will congregate during winter and all animals will need to be infected at the same time.
As densities of domestic animals are far greater than those of populations in nature reserves, formation of highly infectious aerosols is more likely to occur. Ninety percent of the secondary outbreaks were found to occur within a 10 kilometres radius around a domestic infection centre (Hugh-Jones and Wright 1970). The infection risk for (semi-) wild animals within this radius may be higher as these animals have more contact with the open air than animals that live in stables. Domestic animals in meadows or enclosed outlets may play a considerable role in the epidemiology, because they live in high densities in the open air. So aerosols are formed easily and aerosols also easily infect these populations.
The infection risk for (semi-) wild animals may be different, due to the lower densities and difference in habitat structure. Forests diminish the exposure to wind (i.e. aerosols), whereas aerosols can easily reach susceptible animals that roam in open landscapes. The probability, that airborne virus infects a roe or muntjac deer, is therefore small because they live solitary or in small groups in forest areas (Forman et al. 1974). In winter, the infection risk is higher as they congregate in open fields and FMD virus is more stable. Airborne transmission from livestock to wildlife seems more likely than vice versa.
The transmission between domestic animals and (semi-) wild animals via soil seems unlikely when they are spatially separated. Commonly, soil does not disperse over long

distances in large amounts, unless there are natural phenomena like sandstorms and hurricanes. The dissemination of contaminated soil may be negligible for the transmission between livestock and wildlife. Nevertheless, transmission via water is conceivable. Contaminated manure may run-off into the water of nearby ditches or small rivers. The water may flow to another area and if the dilution is not too great, it may be infectious enough to infect animals that drink this water. The lack of field data on the survival rate of FMD virus in water makes it hard to assess a risk. Even so, it can be presumed that livestock form the most potent source, as they have the highest probability of producing contaminated soil and water with high virus concentrations (e.g. by farmers land spreading manure over their land). Wild animals tend to spread their excretions and secretions over a large area.
Some animal vectors may be more likely to get contaminated with FMD virus if either the domestic animals or the (semi-) wild animals are the infection source. However, it remains questionable whether animals that have access to either domestic or (semi-) wild animals also have access to the other. Rats are associated with humans, so contaminated rats are more likely to originate from farms. Contamination with FMD seems most likely to come from contaminated carrion (Ballenger 2001). Farmers however are supposed to remove dead animals as quickly as possible and to control rats at the farm. The density of rats in nature reserves is smaller, so the chance that a substantial number of rats become infected through contaminated meat is unlikely. Hedgehogs and birds are most likely to have access to (semi-) wild animals and domestic animals that live in meadows and enclosed outlets. Humans may facilitate transmission over short distances. But what is the chance that a naive person is first in close contact with (semi-) wild animals and thereafter in close contact with a domestic animal (or the other way around)? It seems possible for veterinary staff, but they probably apply decontamination measures. On a more general note, the notion of animal vectors that transmit FMD by their faeces may be unlikely for susceptible species other that swine. Wild boars and pigs may eat (substantial amounts of) faeces, but other species don't.
In conclusion, the probability of FMD virus transmission over long-distances is higher when livestock is infected then when wildlife is the infection source. Thomson et al (2003) support this view in their review on FMD in wildlife. Wild animals were not responsible for any of the re-infections in South East Asia, South America and Western Europe. Moreover, there are a lot of examples where FMD has been observed in wild animals with a nearby outbreak in livestock as the infection source. A few examples are wild goats in Ireland (Griffin and O'Reilly 2003), an outbreak in an Indian biological park (Kar et al. 1983) and all outbreaks of FMD in European bison in Poland (pers. comm. J. Kita). Many situation-specific factors influence actual infection risks, such as geography, habitat structure, population size and density. In general, wild animals have a lower risk of becoming infected, because the animal densities are considerably lower in nature. Even if a population becomes infected, it will probably die out naturally, due to these low densities (Thomson et al, 2003, Wobeser, 2002)

3.8.1 Persistence in wildlife

A major concern is that wild populations may act as reservoir for new FMD outbreaks in livestock. This may be possible when a FMD infection persists. In a nature reserve, infected animals may go unnoticed by gamekeepers, even more when it is subclinical. In the persistence of FMD the carrier state may play a role. FMD carriers excrete about 100 times less than when the same species are clinically infected. Thus the transmission rate from carriers is slower, as exposure time between infected and susceptible animals needs to be longer. This decreased spread may cause the infection to be self-limiting, when the basic reproduction rate falls below one (Ro<1). The disease will however persist when transmission has not decreased enough (Ro=1).
Dead bodies do not seem to facilitate persistence, as the mortality rate is very low with FMD and carcass meat seems to be infectious for only short periods of time. Carcasses

only form a serious infection risk when there is a big chance that wild boars find the carcass quickly after the time of death. This maximum interval between death and recovery is dependant on the time of year (just as the survival of FMD in the abiotic environment). In the summer, the temperatures are high and the humidity is low, meaning that survival is low. In the winter, the temperatures are low and humidity is high, so survival is higher. These favourable conditions are more common in Eastern Europe than in Western Europe.
From this information, it cannot be assessed how long FMD is likely to persist in wildlife. More information is needed for this assessment, for example, the threshold density under which FMD fades out and the probability that carriers infect susceptible animals. Nevertheless, 'a general observation has been that wherever in the world FMD has been eradicated from livestock it has generally disappeared from wildlife in those regions' (Thomson et al. 2003). The virus is unlikely to persist in wildlife and probably needs constant re-infection from domestic species. The African buffalo is the only known exception. This is also supported by field evidence from the 2001 epidemic. FMD did not persist in populations of wild boars and roe deer in the conservation areas in the Netherlands (Elbers et al. 2003).

3.9 FMD policy

The 2001 outbreak in the UK engendered a lot of discussion, because it was the first major outbreak in 20 years. The killing of so many healthy animals caused a lot of protest outside as well as inside the agricultural sector. The impending threat to valuable or endangered species, the obligation to kill cattle that took a lifetime's work to breed, as well as the killing of pets raised a lot of protest (Pluimers et al, 2002). As for endangered species, a joint conference of OIE and FAO recognized the obligation to conserve wildlife and endangered or otherwise valuable species and recommended to consider the option of vaccination of such animals (OIE/ FAO, 2001). The Terrestrial Animal Health Code now provides more protection to endangered species (OIE, 2004).

FMD free zone where vaccination is practised

An FMD free zone where vaccination is practised can be established in an FMD free country where vaccination is not practised or in a country of which parts are infected. Vaccination of zoo animals, animals belonging to rare species or breeds, or animals in research centres as a precaution for conservation purposes is an example of implementation of such a zone. The free zone where vaccination is practised is separated from the rest of the country and, if relevant, from neighbouring infected countries by a *buffer zone*, or physical or geographical barriers, and animal health measures that effectively prevent the entry of the virus must be implemented. A country in which an FMD free zone where vaccination is practised is to be established should:

(OIE, Terrestrial Animal Health Code art 2.2.10.5, 2004

Even without such provisions, emergency vaccination could have halted the epidemic much quicker than killing without vaccination. However, even though the OIE FMD protocol as well as the EU did permit emergency vaccination, authorities proved very reluctant to use this option. Uncertainty about the trade consequences were the main reason. In the UK, veterinary authorities were still discussing vaccination six months after the outbreak (Leforban, 2002). The Netherlands resorted to emergency vaccination when stamping out failed to control the epidemic. It was necessary to ask permission from the European Commission, which gave some days of delay. Afterwards, the law permitted them a choice between observing the compulsory waiting period, or to kill and destroy all vaccinated animals. Where the cattle farmers preferred the first option, the hog farmers voted against it, because a prolonged quarantine status would delay them from restocking their stables. So, the farmers' organization LTO recommended killing, which was carried

out accordingly. Authorities were so eager to quickly recover the disease-free status that measures to preserve valuable breeding stock, for instance through embryo preservation, were not even considered (Pluimers et al, 2001).
According to EU directive 85/511/EEC, the FMD-free period after stamping out all vaccinated animals was 3 months, in case vaccinated animals were kept alive, 12 months, but only for member states with FMD status with vaccination. In 2003, the EU adopted a new directive on FMD (2003/85/EC). Following OIE guidelines it allows establishment of vaccinated FMD-free zones, where vaccination is practised to protect valuable animals. After an outbreak, the waiting period in the protected zone before recovery to a vaccinated FMD-free status, is shortened to 6 months. The directive requires that all member states develop FMD contingency plans for FMD and have them approved by the European Commission. There will be a central authority in charge, with full competence to carry out any measures deemed necessary.
The British Department for Environment, Food and Rural Affairs (Defra) has developed such a plan, following extensive stakeholder consultation after the 2001 epidemic. It leaves the choice between protective vaccination or stamping out open and provides for stakeholder involvement at local or regional level (Defra, 2004). The Dutch contingency plan identifies different scenarios and prescribes measures to both domestic and non-domestic animals. In all scenarios, only Red List and CITES species are allowed protective vaccination; all other vaccinated susceptible animals have to be killed (Ministry of Agriculture, Nature and Food quality, 2002). Managers of nature reserves have to relinquish their authority. In case of an FMD outbreak, the Ministry of Agriculture, Nature and Food quality takes over the authority from the State Forestry Management. This means in practice that the entire nature reserve will be closed and that only two persons are allowed to enter, with special protective clothing, disinfected cars and upon exiting they have to shower and change clothes. When the outbreak spreads to the Nature reserve, the Ministry has all authority and nature managers fear that it will decide to kill all herbivores (pers. comm. F. de Roder).
Emergency vaccination has been investigated for FMD in cattle, pigs and sheep (Salt et al. 1998, Cox et al. 1999). Pigs are not considered carriers of FMD virus (Salt et al. 1998). Pigs were seldom protected before 21 and 28 days after vaccination, therefore 'the circumstances under which emergency vaccines could be expected to prevent virus spread would highly depend on the circumstances' (Doel et al. 1994). Emergency vaccination has provided a rapid and protective immunity in sheep as early as three days following vaccination, meaning that it limits the transmission of the disease to susceptible non-vaccinated sheep. Cattle were protected four days after vaccination, but a subsequent challenge with virus caused the virus to persist and there appeared to be a correlation with the time interval between vaccination and challenge. Even with a time interval of 4 months persistently infected cattle occurred, although the number of animals was considerably lower (Doel et al. 1994). From these experiments it seems that emergency vaccination can reduce the transmission rate, but can also cause infections to persist. Emergency vaccination may therefore only be implemented to reduce the transmission rate, but afterwards animals must either be killed, or kept in quarantine for an appropriate period.

3.9.1 Prevention and control

During the last epidemic in 2001 many veterinarians called for preventive vaccination (Cunningham 2001, Foster and McNaughton 2001, Stephenson and Davies 2001, Wardrope and Windsor 2001, Wood et al. 2001). A meticulous evaluation about the desirability of general preventive vaccination in the light of the 2001 outbreak led to the conclusion, that it would probably have made no significant difference, had the UK practised preventive vaccination (Leforban, 2002). As to wildlife, there are various reasons why preventive vaccination is unadvisable. It may be impossible to discriminate between animals that have been vaccinated and subsequently infected animals and animals that have been vaccinated, but not infected. Furthermore, even the best FMD vaccines appear

to be relatively inefficient and the reliance on vaccines exclusively is dangerous, as has been shown from experience in southern Africa (Thomson et al. 2003).
The danger lies firstly in that the immunity only lasts a short period of time. FMD immunity lasts approximately 4 months in cattle and a few weeks in pigs (Ferguson et al. 2001, Alexandersen et al. 2003). Only when individual cattle have received several inoculations does the level of immunity remain high against challenges with homologous virus. Thereafter annual vaccination is required to keep levels of immunity at a satisfactory level.
The second problem is that as recovery from one FMD virus type does not protect against another type, it is necessary to vaccinate with multiple types and serotypes. Notably, the vaccination of wildlife has never been conducted in Europe and an attempt in Southern Africa gave inconclusive results (Thomson et al. 2003).
Animals that are infected with the FMD virus usually survive, but may suffer a high morbidity. Emergency vaccination may protect animals from the clinical signs and may reduce the transmission rate during an outbreak, but this measure is not effective in protecting wildlife, as long as as they have to be shot afterwards.

3.9.2 Best policy for wildlife

The best wildlife policy is to prevent an infection from occurring. In general, it is best to prevent regular contact with domestic animals as much as reasonably achievable. During an outbreak domestic animals should be isolated from wild animals by stabling or confining them in a pen. Emergency vaccination to domestic animals should be applied immediately to prevent the disease from spreading. Wild animals should be restricted in their movements by closing off corridors and disturbing the animals as little as possible. In case of an outbreak among wild animals non-intervention is probably the best option. The disease will presumably die-out among wild populations when the domestic infection source no longer forms a risk. FMD seems unable to naturally persist in Eurasian wildlife and wild animals are unlikely to form a risk by aerosol transmission or other forms of long distance transmission. If direct contact between infected wild animals and livestock cannot be excluded, emergency vaccination of domestic animals in risk of contact can be applied.
In cases where the disease is expected to cause huge damage to endangered or exceptionally valuable animals, vaccination of the animals should be applied.

4 Classical Swine Fever

Like FMD, CSF is a bone of contention between nature conservationists and public opinion on the one hand and veterinary authorities on the other one. As with FMD outbreaks, CSF outbreaks are accompanied with massive slaughter and destruction of healthy farm animals and impending threat of extermination to wild boars.
Another issue is the reintroduction of wild boar in many areas and connecting zones between local populations in Western Europe. Due to habitat fragmentation, the conservation status of wild boar is unfavourable in several countries (e.g. the Netherlands, Denmark and the UK). Habitat size is often too small for preservation in the long run. In the Netherlands the wild boar populations are isolated in the Veluwe and Meinweg conservation areas from other populations in Belgium and Germany. Enlargement is at least needed to keep the populations viable, but implementation of the ecological network would be a more constructive way of upholding genetic variance and population size.
Projects to that end are however often opposed for fear of classical swine fever (CSF). The conservation of wild boars is important, because they form an integral part of an ecosystem. These scavengers clear up dead animals and cause other types of dynamics than large herbivores. They root in the ground, while searching for food. This behaviour ventilates the soil and creates spots of bare soil, which facilitates the establishment of certain plant species. The wild boars' ecological role cannot easily be replaced by other species.

4.1 CSF, the disease

Classical swine fever (CSF) or Hog cholera is caused by viruses of the genus *Pestisvirus* of the Flaviviridae family. This very infectious disease only infects domestic pigs (*Sus scrofa domestica*) and wild boar (*Sus scrofa*). It has a high mortality rate, predominantly among young animals. Once recovered, the animal requires a long-time immunity. High mortality has also been reported in wild boar. Whether wild populations harbor chronically infected animals without clinical signs has never been demonstrated, though it seems possible (Artois et al, 2002). CSF is endemic in wild boar and free-roaming domestic hogs in Sardinia since 1983. Also elsewhere in Europe, some populations of wild boar are not CSF-free (Artois et al, 2002). It is feared that via the ecological network infected wild boars may spread the disease to domestic pigs in disease-free countries (Damhuis et al. 2004). There are different strains with different virulence, but there are no serotypes defined. Domestic pigs and wild boars are the main hosts and the course of the disease is similar in both species.
Different strains can cause an acute, chronic or congenital form of CSF (OIE 2002). The acute form causes severe symptoms like anorexia, lesions of the skin, fever and death 5-15 days after onset of the illness. The mortality of young pigs can approach 100%. The chronic form has less severe symptoms, like dullness, capricious appetite, pyrexia, diarrhoea for up to 1 month. An apparent recovery will occur with eventual relapse and death. The congenital form causes piglets to be clinically normal but the virus to persist in the blood with no antibody response. No apparent symptoms can be identified. Only poor growth over a period of weeks or months is observed, where after death ordinarily occurs.
Notably, maternal antibodies have been shown to passively protect piglets from a post-natal CSF infection for several weeks (Depner et al. 2000). Maternal antibodies protect piglets against mortality during the first weeks of life, but not necessarily against virus replication and shedding (transient infection). CSF is a very deadly disease with a high mortality rate, often eradicating 90% or more of the local population (Lang et al. 2000, Chenut et al. 1999).
Pigs that do not die within four weeks of infection either become convalescent and develop high neutralising antibody titres, or they become chronically ill and remain persistently infected with CSF virus. Pigs with chronic CSF may survive for more than 100 days (Artois

et al. 2002). The pigs that survive an infection develop immunity that can last for several years or even for life (van Oirschot 1999). Domestic pigs and wild boars are considered natural hosts of CSF and as yet no other species have been identified as reservoir hosts (Loan and Storm 1968, Dewulf et al. 2001). There are varying reports on wild boar as a reservoir for CSF.
Persistence of CSF virus seems to depend on the area and the interface with domestic pigs. In certain areas, CSF has occurred in wild boar for short periods of time (months to a few years), but the disease has gradually died out later on (Laddomada 2000). In other areas the disease has shown to persist for a long time. In Mecklenburg-Western Pomerania in Germany CSF has been found in about two-third of the region during the second half of the 90s (Laddomada 2000). The disease has been endemic in eastern Sardinia from 1985, due to the spread of CSF infection to wild boar and occasionally to free-roaming pig populations (Laddomada 2000, Artois et al. 2002). It is also endemic in regions of France on the border with Germany (Chenut et al. 1999). The situation for many countries outside Western Europe is unknown, but outbreaks have been reported in recent years (Laddomada 2000).
Earlier history of CSF in wild boar has been associated with the persistence of the disease in domestic pigs (Edwards et al. 2000). The first report of CSF was done in 1830 in Ohio. The first European report was done in 1860. This does not mean that before that date it did not occur, people probably did not know what kind of disease the pigs had. It seems plausible that it originally came from Europe or Asia, but the true source remains unknown. The UK and the USA succeeded in eradicating CSF by killing all infected animals. The EU started eradication in 1980 and succeeded in establishing a disease-free status in most of the region by mass vaccinations, followed by abandoning vaccination and the adoption of a stamping out policy. The CSF-free status did not prevent outbreaks from occurring. In 1997 a massive epidemic in a Dutch region with extremely high pig densities led to the killing and destruction of millions of animals, before the epidemic was halted. Despite provision of emergency vaccination, it was not used because of trade consequences. Public outrage raised ethical questions and gave rise to research into alternative control strategies (Klinkenberg 2003).
The role that CSF seems to play in the ecosystem is regulation of the population density of wild boar. This is due to the high mortality rates caused by the disease. The development of subclinical infections may cause wild boar to be a reservoir for domestic pigs or for wild boar when the population density is again high enough. Unfortunately there are only sporadic experiments with wild boar, meaning that most data are derived from experiments with domestic pigs. This data are extrapolated to wild boars, because wild boars and domestic pigs are so closely related.

4.2 Direct transmission

When pigs are infected with a virulent strain of virus, they usually show high levels of virus in both blood and tissues. Infected pigs can have 103.5 to 103.6 TCID50 /sample in their blood (González et al. 2001). Also, large amounts of virus are excreted in the oral fluids and smaller amounts in urine and nasal fluids. Viral excretion continues until death or until specific antibodies are developed (Terpstra C. 1988). Dewulf *et al.* (2001) mentioned that the average amount of virus excreted by an infected pig under field conditions is comparable to 103 TCID50/ml. The way by which this would be excreted is not mentioned.
The amounts of virus excreted with urine and faeces are low, as compared with the titres in blood, kidney and intestinal tissue (Ressang 1973). Additionally, preliminary results indicate that the virus is readily inactivated in manure under aerobic conditions (Have 1984). The virus may however survive for longer periods in manure and experimental studies suggested that inactivation occurred more rapidly in the liquid phase of slurry than in the solid phase, with infectivity being lost in about 15 days (Have 1984). The amounts that infected pigs excrete via faeces are not reported.

4.2.1 Sexual contact

Boars that are infected with CSF virus can excrete virus with semen and can, subsequently, transmit the virus to sows via artificial insemination and additionally to the foetuses that develop from this (De Smit et al. 1999). The CSF virus isolate used in this experiment did not cause any typical signs of CSF in the infected boars and sows, so the course may be different if a more virulent strain is used. However, in this experiment all piglets developed acute CSF and died or were killed (being at the point of death) within approximately three weeks.

4.2.2 Mother-foetus

Transplacental transmission has been observed regularly, but the effect CSF has on the foetuses or piglets depends on the virulence of the virus strain and the period of gestation the infection occurs (van Oirschot and Terpstra 1977, Terpstra 1987, De Smit et al. 1999, Kaden et al. 2001). The later sows are infected during pregnancy, the more uninfected piglets are born (van Oirschot 1979). Pregnant sows infected by virulent strains die or, when the sows survive, either abort or produce mummified, stillborn, weak or diseased piglets that succumb shortly after birth (Dewulf et al. 2001b). In the case of infection with a low virulent strain, pregnant sows are subclinically infected and generally pass unnoticed. Their healthy looking offspring are however infected with CSF. They may shed large amounts of virus for weeks or months without being detected (Terpstra 1987). The development of symptoms can appear also later in life (late onset, van Oirschot and Terpstra 1977). Piglets have been shown to develop a persistent infection until they died from the infection (van Oirschot 1979).

4.2.3 Dead bodies

'CSF virus may be regarded as moderately fragile, like many enveloped viruses, but is able to survive for prolonged periods in a favourable environment, cool, moist and protein rich, as found in stored meat' (Terpstra 1987, Edwards 2002). Virus survival times of more than 4 years have been recorded in frozen pork (Edgar et al. 1949). It has been reported that artificially contaminated slaughterhouse waste kept at 20 ºC for 3 weeks was already non-infectious after four days (Prost and Bojarski 1967). The infection risk of dead bodies in a nature conservation area may be substantial during winter, when virus survival is longer. The chance of susceptible animals taking up infected material is therefore bigger. Wild boars may eat the carrion and become infected. However a risk analysis has never been conducted on this subject.

4.3 Indirect transmission

It has been a mystery how CSF virus was able to spread over long distances without the help of humans. The movement of pigs and contaminated materials has often been seen as the cause of long distance transmission. A lot of research has been done on animal vectors, because animals have often been incriminated.

4.3.1 Transmission via the abiotic environment

The survival of the CSF virus in the environment is affected by many physical and chemical variables, including temperature, humidity, pH and presence of organic matter. The virus is generally stable in the range pH 5-10. It is rapidly inactivated below pH 3 and above pH 10 (Slavin 1938, Kubin 1967, Edwards 2000, OIE 2002). A sharp pH peak for virus survival in blood was found at pH 5.2 (Chapin et al. 1939). However, the virus survival seems to depend on the temperature as well as on the pH (Depner et al. 1992). The effect of pH below 4 on the survival of CSF virus is much more marked at 4 ºC than at 21 ºC. At pH 4, CSF virus has a mean half-life of 260 hours at 4 ºC against 11 hours at

21 ºC (Depner et al 1992). Notably, these half-lives may be far higher in meat. Additionally, ultra-violet light will rapidly inactivate the virus (Kubin 1967).
As mentioned above, CSF virus is very stable in a protein rich environment and hence in organic material. Survival in the abiotic environment seems therefore limited in absence of organic matter. However, Ellis et al. (1977) distinguished seasonal peaks of outbreaks in the spring and autumn. This could not be explained by larger numbers of pregnant gilts and sows in certain periods of the year, but might be associated with climatic conditions favouring a higher stability of the virus (Terpstra 1987).
It is however still unclear what the routes may be. Transmission via excretions seems unlikely in the early stages of infection from the experiment by Dewulf and other (2002b). Five pairs of pigs were experimentally infected with CSF virus. The pigs were removed from their pens when the pigs were viraemic for at least 3 days and 20 hours later susceptible pigs were placed in these pens for 35 days. During the whole experiment, the temperature in the pens was about 20 ºC and the pens were neither cleaned nor disinfected. None of the susceptible pigs became infected. Remarkably, no virus was isolated from the excretion samples that were taken in the pens at the moment of depopulation and at the moment of restocking.

4.3.2 Airborne

Aerosol transmission of CSF has been reported for short distances of one meter (Hughes and Gustafson 1960, González et al. 2001). This was done by an artificial airflow between two pens. In a situation without an artificial airflow, transmission did not occur over a distance of 3 ft (Hughes and Gustafson 1960). The virus probably needs the air currents to rapidly arrive at the susceptible animals before inactivation. Dewulf et al (2000) also used air currents and showed aerosol transmission over short distances inside a farm. One infectious pig was sufficient to infect other pigs within the same pen by direct contact, whereas seven infectious pigs were needed to transmit the virus towards an adjacent pen (Dewulf et al 2000). Laevens et al (1998) found the role of airborne transmission to adjacent pens to be far more important for the spread of the virus than the role of contaminated clothing or footwear. No research appears to have been done on aerosol transmission over longer distances than meters. This indicates that while air-current transmission is possible, it is probably not an important method of transmission of CSF virus.
It has been suggested that airborne spread from farm to farm could occasionally occur in areas with high pig densities, but that it would not be a major feature of the epidemiology (Hughes and Gustafson 1960, Terpstra 1988). Crauwels et al (2003) support this view, as they could not find an association between time down wind and infection risk for neighbouring farms. ‘Land spreading of liquid manure is unlikely to be a factor in the possible airborne spread of CSF virus, especially since only negligible quantities stay airborne’ (Terpstra 1987).

4.3.3 Soil and water

Little research has been done on the transmission of CSF via contaminated soil or water. This is probably because the survival outside protein-rich material is considered to be limited.
To test survival of CSF virus in. soil, artificially contaminated red bricks and chopped hay were exposed to air (but protected from direct sunlight and rain) Both materials still retained infective virus at 7 days but were no longer infective at 14 days (Slavin 1938). It is unclear whether the infection of brick and hay with spleen fluids represents any real risk of infection. Infectivity was not tested by placing susceptible animals in direct contact with the bricks or hay. Instead, the brick and hay were grinded and a filtered suspension was injected into a susceptible pig.
Bovine viral diarrhoea (BVD) virus is member of the genus Pestivirus that infects cattle. Using BVD virus as a surrogate for CSF, it was shown that survival times in various types

of water varied from 6 till 24 days at 20 ºC (Pagnini et al. 1984). CSF virus was shown to persist in foetuses and foetal placenta, meaning that large amounts of virus may be disseminated at farrowing (Terpstra 1987). However, blood and other fluids that are excreted are often eaten by the sow, as is the afterbirth (Graves 1984). Thus direct risk from these materials may be very limited.

4.3.4 Animal vectors

Birds have been suggested as possible animal vectors for CSF. Kaden and others (2003) inoculated five ravens, one hooded crow and two laying hens orally with 1 ml of CSF virus ($10^{4.5}$ $TCID_{50}$). The hens and birds were kept in a pen with pigs and other pigs were fed the faeces of the birds. None of the hens, birds or pigs developed clinical signs during the study. CSF virus could neither be detected from the faeces nor from the blood and organs of the birds. This may indicate that these species don't play a role in virus transmission. Pigeons and buzzards also do not seem to transmit the CSF virus (Dorset et al. 1919, Dawson et al. 1914, Loan and Storm 1968). English sparrow (*Passer domesticus*) have however been shown to transmit the CSF virus from infected to susceptible pigs (Hughes and Gustafson 1960). But sparrows don't develop antibodies against the virus and do not transmit the virus to other sparrows (Loan and Strom 1968). This may indicate that they do not serve as reservoir hosts for pigs, but probably as mechanical vectors. It could even be possible that in the experiment of Hughes and Gustafson (1960) the wind currents caused by sparrows promoted aerial transmission. Sparrows flew to and fro the houses of infected and susceptible pigs that were connected by a 6 feet (1.8 m) long flyway. The same authors had already demonstrated airborne spread of CSF virus by using artificial air currents. So birds are at best mechanical vectors, not reservoirs, but it is equally possible that their role is of no significance.

The role of cats, dogs and rats as reservoirs seems to be unlikely (Dewulf et al. 2001). Three cats, three dogs and four rats were inoculated orally and intranasally with the average amount that an infected pig excretes (103 TCID50/ml). After an observation period of 43 days, it was concluded that no or very minimal viral replication had occurred in the animals. This is in accordance with the previous studies (Schwarte 1959) rejecting the role of birds, rodents, dogs or cats. These animals may not represent significant biological reservoirs, but mechanical spread of CSF (i.e. via fur or feathers) remains possible. It has not been excluded that these animals transmit the disease by eating contaminated meat and excreting it via their faeces. There are however no reports on these types of transmission.

Sheep and goats have been shown to harbour the virus for as long as three weeks (reviewed in Hughes and Gustafson 1960). Infected sheep and goats develop antibodies, but did not seem to transmit the virus naturally to other sheep of goats (Loan and Storm 1968). Other species that were found to produce antibodies after experimental inoculation with CSF virus (but could not transmit it to animals of the same species in direct contact) are deer, peccaries and calves. Additionally, calves did not transmit the virus naturally to susceptible pigs in direct contact (Loan and Storm 1968).

Chenut and others (1999) found that hares and rabbits developed antibodies when a live CSF vaccine (C-strain) was administered. Only the rabbits clearly showed clinical signs, but they did not transmit the virus to animals of the same species nor did any animal die. Sheep were not found to develop antibodies due to the vaccine. The authors also reported the absence of clinical signs and antibody formation after oral delivery of C-strain vaccine to wild rodents, rats, foxes, goats and crows, but no data was shown.

Research has been done on the potential of insects to act as animal vector (Tidwell et al. 1972, Stewart et al. 1975). The biological transmission of CSF virus by mosquitoes (*Aedes aegypti*) could not be confirmed, but mechanical transmission was possible (Stewart et al. 1975). This means that transmission did not occur due to blood transfusion, but due to contact. The virus had persisted for 3 days in the mosquitoes. However transmission attempts of Tidwell et al. (1972) using 6 species of mosquitoes were unsuccessful. It is

noteworthy to emphasize that *Aedes aegypti* was not among these six species. Two species of horseflies (*Tabanus lineola* and *Tabanus quinquecittatus*) experimentally transmitted the virus to susceptible pigs within 2 hours after biting an infected pig. Three other *Tabanus* species were incriminated. An apparent 24 hours delayed transmission of the virus by horse flies occurred. There was however some evidence that biological transmission did not occur, but that it was mechanical transmission (Tidwell et al. 1972).
Hughes and Gustafson (1960) found that stable flies (*Stomoxys calcitrans*) and houseflies (*Musca domestica*) were not capable of transmitting CSF. However, Miller and others (1974) isolated CSF virus from houseflies and stable flies up to 72 hours after exposure of an infective blood meal. In addition, face flies (*Musca autumnalis*), which tend to live longer that the former two species, harboured large quantities of virus for up to 7 days (104.6 PFU/ml). Although biological transmission has never been demonstrated, it is conceivable that these Diptera species could occasionally play a role in the mechanical transmission, especially where infected and susceptible pigs are in close proximity and the flies are in abundance (Terpstra 1987).
Shope (1958) showed swine lungworms to be a reservoir of CSF virus, but no further research seems to have been conducted to assess the risk of transmission under natural conditions. Manure worms (*Heliodrilus* sp.) were not capable of transmitting CSF under experimental conditions (Hughes and Gustafson 1960).
Humans are seen as mechanical vectors. Hunting farmers are often identified as transmitters for taking home wild boars and swill feeding the offal to their pigs. Contaminated clothing could not significantly contribute to the spread of the virus (Laevens et al. 1998).

4.4 Infection dose and route

Domestic pigs and wild swine seem to be equally susceptible to CSF (Brugh et al. 1964).
Under experimental conditions, 'pigs have been infected by oral, nasal, aerogenic, conjunctival, genital and various parental routes' (Terpstra 1987). But pigs are usually infected via inhalation. The minimum infection dose via the respiratory route seems to be less than 10 TCID50 per pig (Liess 1987, Dahle and Leiss 1995, Dewulf et al. 2001).
The oral infection route is probably less important, because researchers had great difficulty with infecting pigs (Slavin 1938, Hughes and Gustafson 1960). Remarkably, a bacterial infection with *Salmonella choleraesuis* seems to be an important synergistic factor in the oral infection with CSF (Hughes and Gustafson 1960). All 10 pigs exposed to both virus and bacteria got infected orally, while the virus infected only 2 out of 10 pigs without the bacteria.
The venal infection route appears to be possible through abraded skin (Hughes and Gustafson 1960). However, in this experiment, the virus was deliberately applied to the abraded skin. It is therefore unclear whether this is an important infection route and what the MID might be.

4.4.1 Carrier state

There are different ways by which pigs can become carriers. The first way is the 'carrier-sow syndrome'. This is caused by strains of low virulence (Terpstra 1987, De Smit et al. 1999). The carrier-sow syndrome implies that the sows are subclinically infected with CSF and transmit the virus to the foetuses. The piglets from these sows are born apparently healthy, but shed large quantities of virus for 4 to 6 months without showing signs of disease of developing an immune response (Meyer et al. 1981). This carrier-state may occur in 43% of the pregnant sows in one herd (Terpstra 1987).
Post-natal infections with strains of low or moderate virulence may also induce a carrier state in piglets (Baker and Sheffy 1960). The maternal antibodies give the piglets a passive protection that masks the clinical signs. Transient infections have also been seen in piglets due to maternal antibodies against CSF (Depner et al. 2000).

Another way for a carrier state to develop is from vaccination. Normally, vaccinated pigs do not transmit the virus to susceptible pigs (Chenut et al. 1999). But when the immunization due to the vaccination has not yet established itself or because the vaccine is not effective enough, a carrier state may result (Kaden et al. 2001). Swine vaccinated by a vaccine of insufficient potency may develop a subclinical infection when exposed to field strains, and infected pregnant sows may still transmit the virus via the placenta, despite vaccination (Leunen and Strobbe 1977). For example, a recently developed marker vaccine did not prevent vertical and horizontal transmission of CSF virus (Dewulf et al. 2002). It only protected pregnant gilts from the clinical course of the disease, thus a carrier state was initiated. Even though it is known that CSF carriers exist and can transmit CSF to susceptible animals, the amounts of virus that CSF carriers excrete are still unknown.

4.5 Ranking of transmission routes

Like FMD, it seems hardly possible to prevent primary outbreaks from occurring. Primary outbreaks originate from swill feeding infected meat or introduction of animals from an infected region, before CSF was noticed, or illegal transport. The transmission of CSF virus is considered to be mainly by direct contact with infected pigs and contaminated meat. For example, all three primary outbreaks in 1986 in Great Britain were attributed to the feeding of unprocessed waste food containing imported pigs meat products. Further spread was attributed to markets where infected pigs were traded and mechanical transfer also occurred (Williams and Matthews 1988). Thus long distance spread of CSF was commonly assigned to human practices.

It is difficult to treat CSF-infected meat in such a way that it renders the material non-infective. In its guidelines for the treatment of CSF-infected meat, OIE prescribes lengthy heat treatment or natural fermentation processes of considerable length (OIE, 2004, Terrestrial Code, appendix 3.6.4). So, the only way to prevent this transmission route is to prohibit swill feeding and effectively enforce this prohibition.

In areas of high pig density, the situation may be different. For example, during the epidemic in the Netherlands 1997-1998, radial dispersion of CSF virus seemed more important in neighbourhood infection (within a radius of 1km) than dispersion along the road on which the infection source is situated (Crauwels et al. 2003). This implies that airborne transmission or transmission via animal vectors like pets, rodents, birds or insects were more important than transmission via, for example unknown or unreported social contacts between neighbours, which the authors had expected to be more associated with dispersion along the road. From the references reviewed in this report, transmission by animals as mechanical vectors seems possible and specific meteorological conditions allow for airborne spread. But during the epidemic there was no association found between time downwind and infection risk for neighbouring farms. Transmission by animal vectors seems therefore to be most likely route, although airborne spread cannot be excluded as possible contributing factor. No specific animal species were proposed to have caused the neighbourhood spread of CSF. The transmission distance was however limited within a radius of only 500m (Crauwels et al. 2003). Staubach and others (1997) found the same radius. Roberts (1995) found that in high pig density areas CSF transmission is usually limited to neighbouring herds within a 6 km radius. In the epizootics he referred to, the major modes of long distance transmission were movement of animals, equipment or staff (contact spread) and possibly airborne spread.

It seems likely, that animal vectors can play a role in short distance transmission. Mechanical transmission of CSF virus in clothing, feathers or fur may be unlikely for long-distance transmission, because the conditions (i.e. temperature and low protein content) may be unfavourable for virus survival. Transmission via faeces of cats, dogs and rats (after these animals have eaten contaminated pig's meat) depends on the virus survival in the digestive tract. Swine occasionally eat faeces, but the amounts are unknown. There are however a lot of lacunas in the data on the mechanical spread via animals.

The rest of the transmission routes cannot conclusively be placed in a ranking of importance. Congenital transmission and sexual transmission take place, but it cannot be assessed how much they contribute to the spread. Airborne transmission seems to only occur over short distances. Information about the survival of CSF virus in the abiotic environment is lacking, but is presumed to be low when the protein content is low.

4.6 Interface of domestic and wild animals

Direct contact, infected meat (i.e. dead bodies) and over short distances animal vectors seem to be important routes for the transmission of CSF virus. By knowing this, it may be possible to assess the transmission risk when either wild boars or domestic pigs are the source of infection. Wild boar populations can constitute a source of CSF virus. Direct contact between wild boars and hogs kept outside is possible in some regions, but only is a risk if the boar population harbours CSF. Furthermore, when measures are taken to prevent direct contact between wild boars and domestic pigs by spatial separation, the transmission risk seems minimal, because long distance transmission of CSF virus seems to be limited. Airborne spread has its limitations and so has CSF survival outside the host's body.

Feeding contaminated meat of wild boars to domestic hogs is the most frequent source of primary outbreaks in regions with CSF endemic in wild boar populations. Farmer/ hunters fed parts of the animals they shot to there livestock. Domestic pigs that die before slaughter are commonly disposed of by incineration, but sometimes illegally by burying them on the spot. Although the situation may differ in Eastern Europe, as there are more smallholder farms and state surveillance is less tight. Fortunately, rules have been implemented prohibiting swill feeding entirely or demanding decontamination by thoroughly heating the meat before feeding (Ferrari et al. 199, Laddomada et al. 1994, Fritzemeier et al. 2000).

> 'Transmission to domestic pigs from infected wild boars has never convincingly been proven to occur by aerosol or by contaminated natural products, like earth and grass' (Artois et al. 2002).

Taking into account the lack of data on the survival of CSF virus outside its host, it is still possible to discuss the risk domestic pigs and wild boars pose to each other by different animal vectors. Contaminated flies, rats and pets seem more likely to originate from domestic animals, rather than from wildlife, as the density of infectious particles is higher and these animals, including stray cats and dogs, commonly live close to humans. Stray cats are more likely to come into contact with infected pigs than birds or stray dogs. They are tolerated in stables, as they can help a farmer with rodent problems. However, stray cats are sometimes seen as a pest, just as stray dogs (common in Eastern Europe). Eating meat offal or carcasses of wild boars may contaminate these animals. The question remains how infective these carcasses and their faeces are and what the probability is that susceptible swine have access to these animal or their faeces.

Another factor that has to be mentioned in light of direct contact between wild boar and domestic pigs is that wild boars infected with CSF virus may exhibit altered behaviours, such as loss of natural shyness, approaching or even entering farm buildings, or leaving the forests during daylight (Loepelmann and Dedek 1987, Artois et al. 2001).

In conclusion, it seems that the probability of CSF virus transmission is higher when domestic pigs are the infection source than when wild boars are the infection source. Yet, wild boars are a possible infection source as long as there are wild boar populations persistently infected with CSF.

There are a few reports on the transmission of CSF from domestic to wild boars. For example, in the north of Mecklenburg-Western Pomerania (spring 1993) there were two outbreaks in domestic pigs and it was speculated that these were the source for an infection in wild boar (end of 1992, Fritzemeier et al. 2000). This virus type spread among the population and kept circulating in the wild boar population. Reinfection of domestic pigs with this strain occurred in 1998. The scarcity of reports is probably due to the lack of

interest for CSF in wild boar before the eradication program started of CSF in the 80s. The interest for the CSF in wild boar emerged after the eradication in domestic hogs was successful and wild boar populations appeared to be a risk factors for re-emergence of this economically devastating disease.
The CSF outbreaks in pig farms in Tuscany (Ferrari et al. 1991), in Sardinia (Laddomada et al. 1994) and in Germany (Fritzemeier et al. 2000) have been linked to contact with wild boars. In Germany, 59% of the primary outbreaks in the period 1991-1998 in domestic pig herds were due to direct or indirect contact to infected wild boar and wild boar meat, whereas 23% of the primary outbreaks were due to the (illegal) swill feeding of contaminated meat from abroad. The source of the infection of the remaining 18% could not be determined (Fritzemeier et al. 2000). Fortunately, the domestic pig herd density is small in the German regions that have infected wild boar population. The risk of large epidemic may therefore be small. No transmission was reported from wild boar to domestic pig in the epidemic of 1998 in Switzerland. This was probably due to the low density of pig farms in rough terrain (Schnyder et al. 2002).

4.6.1 Persistence in wildlife

CSF has been shown to persist in wild boar populations. It is still endemic in wild boar populations in regions of Germany, and in most cases does not seem to be self-limiting (Fritzemeier et al. 2000). In Sardinia, it has been endemic for at least 15 years and the transmission from wild boar to wild boar seems to play an important role in the spread and persistence of classical swine fever (Artois, 2002, Laddomada et al. 1994).
For CSF to be endemic, the basic reproduction rate of CSF must be approximate 1 ($R_o \approx 1$), yet not fall below 1, lest the infection will fade out. For an epidemic, the reproduction rate has to be far greater than 1 ($R_o > 1$). Persistence seems to partly depend on the virulence of the CSF strain. A highly virulent strain causes high mortality and pregnant sows tend to abort, thus recruitment is primarily via immigration. The epizootic will die-out when the recruitment is lower than the mortality rate, because the basic reproduction rate of CSF decreases as the number of available hosts decreases ($R_o < 1$). This was apparent from the epidemic in Switzerland in 1998, which was self-limiting (Schnyder et al. 2002). Key elements for this were the high virulence of the virus strain, which resulted in a drastic reduction of the population of wild boar and the establishment of a high level of herd immunity. The presence of physical barriers at the borders of the risk zone also helped to reduce the prevalence of CSF in the wild boar population by limiting immigration.
Mild virulent virus strains will decrease these population-reducing factors. Regularly, the mortality is considerably lower and there are more carriers in the population. The ratio between immune pigs and carriers presumably depends on the specific virus strain. Recruitment would for a part be in the form of chronically and persistently infected piglets, due to the ‘carrier-sow syndrome’. These aspects are responsible for maintaining the infection, especially in medium to large size herds (Terpstra 1988).
Infected carcasses may also present an infection source that contributes to the persistence of CSF in a wild boar population. The risk from this source also depends on the virus strain, as the amount of dead animals depends on the mortality rate. In addition, it also depends on the climate. Carcasses especially pose a risk in the event of an epidemic in winter. Frozen carcasses can harbour the virus for months or even years. For example, the epidemic may have died-out during the winter as a lot of animals die from disease, starvation or otherwise. Reinfection can occur in due to exposure to infected carcasses that were buried under snow. The infection risk however diminishes after defrosting, as the virus will presumably inactivate in a few days or weeks. ‘CSF virus may be more persistent in Eastern Europe than in Western Europe due to the climatic differences (lower temperatures) and/or the size of the populations of wild boar’ (Terpstra 1987).

Other factors that seem to play a crucial role in the shift from an epizootic to a persistent endemic situation are the size of the affected population, the density and the age structure (Artois et al. 2002). The larger the population size, the higher the density and the younger the age structure, the greater the chances of virus persistence.
A young age structure causes the immunity to be very low in the population as older animals may have immunity from previous outbreaks. A large portion of young pigs is a passively protected against the clinical signs due to maternal antibodies or they are persistently infected via the carrier-sow syndrome. This means that a large portion of the population will consist of carriers during an outbreak, when the age structure is young. Young boars can disperse the virus over long distances, as they leave the family group.
From model studies, the most conservative (= lowest) estimation of the density required for a CSF infection to fade-out would have to be about 200 susceptible animals in an area of approximately 220 km2 (0.9 animals/km2) or lower. This threshold density has also been stated in disease control programs for CSF (Artois et al. 2002). This density was in correspondence with the situation in Switzerland (Schnyder et al. 2002). Estimation of the threshold density depends on the method and assumptions employed. It also depends on various factors like climate and geography. For example, from models it is has been assessed that the threshold was 6.4 wild boar/km2 for an area in Pakistan and 0.6 -1.1 wild boar/km2 in Eastern Sardinia (Artois et al, 2002, Schnyder et al. 2002).
The persistence of CSF in wildlife depends on various factors, including the rate of transmission in the population considered. The danger of persistent CSF in wild boars is that it can spread from one area to another due to transmission among wild boars. It is however unclear at what rate dissemination from one area to another will take place. It seemed to take years before CSF had spread from one province unit to another unit in the province Nuoro, Sardinia (Laddomada et al. 1994). In the epidemic of 1998 in Switzerland only one wild boar was found that had contracted the infection within the infected zone and migrated into the non-infected zone. During this epidemic, CSF had never spread to the non-infected zone (Schnyder et al. 2002).
Artois and others (2002) state that the CSF virus does not appear to spread easily in natural populations of free-living wild boars. This low transmission rate is probably due to the ecology of wild boar, but can also depend situation-specific conditions, such as the degree of habitat fragmentation and the type of habitat. The rate of CSF transmission is presumed to be lower when the habitat is rough, impervious and fragmented, because the chance that wild boars meet is lower.
Wild boars live in social groups and the movements of these groups are commonly restricted to a home range. This home range is however not defended as a real territory (Graves 1984). Wild boar groups do not leave their home range as long as they are not disturbed. Groups usually consist of a sow and her piglets (Graves 1984). The larger groups are each led by a sow (the guiding sow. It is further composed of other sows, the piglets and maybe a few young males. Family groups do not fall apart until either the young are sexually mature (7 to 8 months) or a sow is ready to farrow again (Graves 1984). Males start living relatively solitary from an age 1 to 2 years. These adult or subadult males usually join female groups during the mating season (November and December) after which the piglets are born in March and April. Males have a larger home range than females (Graves 1984).
The entire distribution area is subdivided in home ranges for herds or individuals. Each has his own undefended territory. These home ranges border each other and there is contact between groups that have a family relationship. It is important not to disturb the social order (pers. comm. M. Montizaan), because it promotes long distance transmission of CSF. During periods of food scarcity or intense hunting, wild boars can cross a distance of more than 15 kilometres, while looking for a new habitat (Baubet et al. 1998). Different methods of hunting can have different impacts of the dispersal of wild boars.
In conclusion, the persistence of CSF in a wild boar population can be promoted by a mild virulent strain, a large population size, a high host density, a young age structure and/or a

cold climate. The transmission rate of CSF in a large area depends among other things on the ecology of wild boars. The rate of transmission can be low in wild boar populations, because the movement of wild boar groups is restricted to a home range, as long as the social order is not disturbed.

4.7 CSF policy

At the beginning of the 21st century, CSF remains widespread in parts of the globe, such as in South America and Asia. Successful eradication has been achieved in many countries, including North America, Australasia, and parts of Northern Europe and many countries have successfully maintained a disease-free status in the absence of vaccination with totally susceptible swine populations. Yet, complete eradication is still out of reach in parts of Europe (Edwards et al. 2000). There are a few regions where CSF persists in wild boars (for example, the French-German border area and probably in Poland, pers. comm. E.G.M. van Klink).
The OIE Terrestrial Animal Health Code distinguishes between a CSF-free status with or without CSF in wild pigs:

> **Article 2.6.7.5.**
> Country or zone free of CSF in domestic pigs but with infection in the wild pig population
> Requirements in point 2.6.7.4.2. of Article 2.6.7.4., as relevant, are complied with, but CSF infection is known to occur in wild pigs. Additional conditions for the free status are that in the country or zone:
> - a programme for the management of CSF in wild pigs is in place, and CSF wild pig control areas are delineated around every CSF case reported in wild pigs, taking into account the measures in place to manage the disease in the wild pig population, the presence of natural boundaries, the ecology of the wild pig population, and an assessment of the risk of disease spread;
> - biosecurity measures are applied to prevent transmission from wild pigs to domestic pigs;
> - clinical and laboratory monitoring (under study) is carried out in the domestic pig population, with negative results.

EU control programmes may include monitoring boar populations, oral vaccination and further restrictions in case of an outbreak (European Commission, 1999).
From 1990 onwards, no regular vaccination against CSF has been performed in the European Union (Vandeputte and Chappuis 1999). This also applies to the new countries after accession, as the eradication programs of all the countries of the EU have been harmonized. In principle, all EU countries have the same policy (pers. comm. E.G.M. van Klink). In the rest of Central and Eastern Europe vaccination is still performed. Stamping-out alone or combined with vaccination is used in response to a CSF outbreak in all of these countries. The epidemiological CSF situation in wild boar is unknown for the majority of the countries of Eastern Europe (Edwards et al. 2000). In Sardinia the strategy is to let CSF run its course in wild boar, but at least 14 years later, it is still enzootic (Laddomada et al. 1994, Artois et al. 2002).

4.71 Prevention

As mentioned before, there is a lot of uncertainty about transmission routes. Prevention could be more accurate, if the mechanisms behind neighbourhood infections were better understood. As to wild boar, there is still a lot of data lacking about CSF status, spread and persistence. Where monitoring programmes are at work, wild boars are regularly monitored for the presence of CSF, by (randomly) checking the blood or wild boars that are either found dead or shot by hunters.

Pre-emptive depopulation is a legal possibility in the Netherlands. This policy however is not considered to be effective. This is mainly caused by the high recruitment of wild boars. The wild boar populations have experienced explosive growth in recent years, due to rich mast years (Damhuis et al. 2004).
Farmers can do much to prevent CSF infection from wild boars

> 'An important factor for virus transmission from wild boar to domestic pigs is mismanagement in the pig holdings themselves, i.e. lacking hygienic measures (contaminated equipment and clothing), false handling of wild boar carcasses (swill feeding) and lack of protection against contact with infected wild boar' (Fritzemeier et al. 2000).

Farmers need to take responsibility in protecting their livestock. Therefore, good farming practices need to be encouraged. Isolating domestic stock from wild boar in regions with Although ecologists and nature conservationists generally don't favour eradication of CSF, OIE guidelines as well as EU economic policy in the long run aim at the highest CSF-free status, including a CSF free status of wild animals.

4.7.2 Control

Although emergency vaccination can slow the transmission, stamping out is generally preferred. Main reason is that vaccinated animals cannot be distinguished from infected ones. Another important reason is that vaccinated sows tend to produce carrier piglets, which pass unnoticed. Recently emergency vaccination for CSF has been allowed for domestic pigs under certain circumstances (pers. comm. E.G.M. van Klink). Unfortunately, there is still the problem of discriminating between pigs that are vaccinated and subsequently infected and pigs that are vaccinated but not infected. Furthermore, there is the risk that vaccinated pigs, as carriers, still transmit the disease. A marker vaccine that has been developed for CSF does not seem to prevent horizontal and/or vertical transmission (Depner et al. 2001, Uttenthal et al. 2001, Dewulf et al. 2002).
At the moment, there are marker vaccines available, that can considerably limit the spread of epidemics and thus prevent unnecessary killing of millions of healthy animals. The problem of carrier animals can be circumvented by not vaccinating the sows. Thus, most animals will be immunized and Ro will fall below 1 and stop the epidemic. The unvaccinated animals, i.e. the sows and the very young piglets will function as indicator animals and develop symptoms, should a carrier animal be present in the herd (Klinkenberg, 2003).
Yet the very reasons, as mentioned in the previous chapter on FMD, to prefer stamping out over emergency vaccination and keep the vaccinated animals alive, also apply to CSF. In theory, use of a marker vaccine allows trading of slaughtered vaccinated animals. But unless there is an OIE approved protocol declaring such practices safe, disease-free countries will probably not accept such meat. At the moment, ring vaccination and subsequent culling is probably the best option.
A persistent preference for stamping out bodes ill for wild boar, should they become infected in an intensive pig farming region. In the 80s there was an outbreak of CSF under domestic swine in the Netherlands. The epidemic spread to the wild boar population in the enclosed National Park 'de Hoge Veluwe', in the Netherlands. It was decided to shoot all wild boars in the National Park. CSF was successfully eradicated, but the total extermination of the wild boar population could not be achieved. A few wild boars survived and repopulated the enclosed area (pers. comm. M. Montizaan). Fortunately the outbreak of 1997-1998 in the Netherlands, did not reach the wild boar populations (Bernard et al. 1999, Dekker and Elbers 2000, Crauwels et al. 2003).
Depopulation by hunting is a regular method in wild boar population that is infected with CSF. This however tends to remove recovered immune individuals and to favour compensatory reproduction and immigration (Laddomada 2000, Damhuis et al. 2004). This will increase the proportion of non-exposed susceptible individuals as well as carriers through transplacental transmission. It is therefore now recommended that boar hunting is

stopped as soon as a CSF outbreak is detected. In the absence of culling, infected animals die from the infection or seroconvert, and the number of susceptible animals is more likely to decrease below the threshold density required for persistence. In this situation, a high proportion of the population will be composed of immunized animals and the probability of contact between susceptible and infectious animals shedding the virus will be low. 'Thus without hunting, the CSF would be expected to die out within a year or two' (Artois et al. 2001). This is only the case when the virus strain is highly virulent and the conditions allow a high transmission rate. Otherwise the basic reproduction rate of CSF may stabilize above 1, like in Eastern Sardinia, where CSF has remained endemic for at least 15 years without intervention by humans.

In some areas with endemic CSF in wild boar populations (e.g. Eastern Germany), it is common practice to establish feeding places for wild boars during winter, to make depopulation easier and to decrease the direct hunting disturbance. Supplementary feeding however unnaturally increases the population density and can cause high concentrations of wild boar in areas with artificial feeding places. Depopulation by hunting may thus promote the transmission risk among wild boars and help CSF to prevail.

It is possible to control a CSF epidemic in wild boar by other means. There have been various attempts to develop a method to effectively vaccinate wild boars. For example, aerosol vaccination against CSF has been tried but did not seem to induce an immune response (Beard and Easterday 1964). On the other hand, oral administration of vaccine by burying bait has been shown to immunize wild boar against CSF and be safe for rabbits, hares and sheep that may accidentally come across it in the wild (Chenut et al 1999). Unfortunately, in the evaluation of the first field study in Germany there appeared to be a bottleneck, as more than 50% of the young boars did not feed on the vaccine baits nor became immunized (Kaden et al. 2000). Fortunately, the rest of the wild boar population could be immunized properly, as 72% of the animals that ate the bait were serologically positive after the first vaccination campaign. To obtain a higher seroprevalence in the younger age group, it may be necessary to enlarge feeding places and to reduce the size of the baits. Another way to improve the immunity in young boars could be by summer immunization when maternal antibody titres are low or absent (Kaden et al. 1999).

Kaden and others (2000) have successfully eradicated CSF in wild boar population from the infected area in Lower Saxony by oral vaccination. Likely contributory factors for the eradication of CSF were a moderate wild boar density, low infection pressure and proper application of oral immunization In contrast, the eradication of CSF in wild boar populations of Mecklenburg-Western Pomerania and of Brandenburg using oral immunization was not possible within four years. In these immunization areas, the density of wild boar population is higher and the environmental conditions are also very different (Kaden et al. 2000). The pitfall in oral immunization is that the vaccination of an insufficient number (or proportion) of animals will not only lead to a failure of the eradication program but may even allow CSF to become endemic, because it does not prevent the spread of wild-type virus (Artois et al. 2001, Van Campen et al. 2001). The effectiveness of oral vaccination is therefore situation-specific and may not be applicable in areas where the bait is difficult to spread (e.g. mountain, large nature reserves).

A risk with emergency vaccination could be that pigs develop a persistent CSF infection. Kaden and others (2001) researched this risk for oral vaccination of wild boars or pigs in an infected area. Pigs that are vaccinated and infected simultaneously show a transient viraemia and shedding of virus for some days after which the animals die. Notably, pigs that were vaccinated orally and infected with a virus of low virulence at the same time can transiently shed virus and become immune. No virus could be found in any part of these pigs, meaning that such animals are not a natural virus reservoir.

4.7.3 Best policy for wildlife

There are strong arguments in favour of non-intervention, should a CSF outbreak occur in wild boar. It would keep CSF's ecological function intact and there is a good chance that the disease will fade out all by itself. Non-intervention will also prevent the disease from spreading to other wild boar populations. For practical purposes, it may even be the only alternative in many areas, covering a large surface and pretty rough terrain. On the other hand, wild boar may become a reservoir of CSF for a considerable length of time, testing tolerance of agricultural interest groups towards wild boar populations. Even so, in intensive pig farming area, the risk of wild boar to domestic pigs is minimal, since intensive farms are thoroughly isolated from wild boar. Endemic CSF is a risk, where domestic swine are kept outside and where sensible precautions are not taken. Yet there is no such a thing as 100% intensive or a 100% extensive pig farming area. Nowadays, industrial systems co-exist with organic farms, with pigs roaming outside. So the domestic-wildlife interface is never entirely without risk. Prevention of direct contact is always at the order, especially as wildlife in general has more to fear from domestic outbreaks than vice versa. Current practice in the EU of monitoring wild boar and taking measures, involving strict isolation have proven adequate in preventing CSF transmission from wild boar to domestic pigs. Educating farmers and enforcing the ban on feeding swill or boar offal to domestic pigs may still need more effort.

The risk of transmission from wild boars to domestic pigs is minimal when direct contact and illegal swill feeding or feeding of wild boar carcasses are not practised. It is necessary to protect domestic pigs, e.g. by fencing around the farmyard and by good management in pig holdings. Yet, given the gaps in knowledge of transmission mechanisms and the difficulty to keep swine (domestic as well as wild ones) on the proper side of the fence where domestic pigs are free-roaming, it may sometimes be desirable to eradicate CSF in a wild boar population, if feasible. Vaccination campaigns will be the first choice. It should be set up in such a way to favour vaccination of the young ones. Animals should be disturbed as little as possible (no hunting) and confined to the area, isolating them from CSF free populations by closing off corridors.

5 General discussion and recommendations

5.1 Discussion

- The objective of this study is to explore alternative prevention and control strategies that better serve nature conservation, in view of the transmission risk of infectious animal diseases between wildlife and livestock. This is a relatively new issue, because the European nature policy, aiming at a Europe-wide network of interconnected nature reserves is still far from realisation. Recent outbreaks of CSF, FMD and fowl pest have launched a public debate about mass destruction of animals, serving primarily trade interests. The 2001 OIE/ FAO conference on FMD recommended member states to include provisions to protect endangered species, in particular those covered by the Convention on International Trade in Endangered Species. OIE's own contribution would be to allow vaccination of endangered species, without losing the FMD-free status, under condition that such animals are individually identified and kept in a location where physical barriers and zoosanitary procedures effectively prevent direct or indirect contact with pathogens. Another provision OIE would consider, was compartmentalisation (OIE/ FAO, 2001). Also OIE's periodical Scientific and Technical Review published a special issue on the wildlife/ livestock interface in 2002.
- On initiative of OIE, the EU organized in 2004 a multistakeholder conference on animal welfare. The wildlife issue was briefly addressed and yielded three recommendations: 1) that OIE should work with relevant stakeholders on the subject of animal welfare standards for wildlife 2) that OIE should take into consideration endangered animal species when developing animal welfare standards 3) that OIE should provide guidance for the welfare of endangered species and exotic species in captivity. As for killing for disease control purposes, it was recommended to have species and disease specific plans and to consider wildlife or other disease reservoirs (European Commission, 2004). OIE has abandoned its former classification into list A, list B etc disease, with basic guidelines according to the list classification. There is only one list now, and guidelines are specific per disease. The current guidelines include zoning within a country and provisions for protective vaccination of endangered species. The EU directive has similar provisions in its new FMD directive.
- Although this has laid a more favourable ground for further work in this area, it is only the beginning. Nature conservation as an issue in its own right has not been acknowledged, nor de-domesticated species that are increasingly employed in nature management and restoration. As of yet, there is little room for improvement for wild populations, and none at all for domestic animals employed in nature management. The EU Habitat Directive (92/43/EEC) contains articles permitting exemption from the obligations under this directive, for example Article 16. Protection of listed species can be overruled in order to prevent serious damage to crops and livestock, or for other reasons of overriding public interest, including those of a social or economic nature. As the Habitat Directive offers no protected status to herbivores in a process of de-domestication, the position of these animals is even weaker.
- There is no compelling reason for not including valuable non-production animals in the OIE or EU rules allowing for protective vaccination, as long as these animals don't enter the food chain and don't form an infection risk. Such animals include pets, traditional breeds on recreation sites or animals kept for sport or hobby, as well as the semi-wild animals discussed in this report. Yet, in many situations,

domestic animals graze in nature reserves and are slaughtered for meat. This would be an argument to treat them as production animals. But if they are not, they should not be treated as such.

- It would be undesirable to narrow down a discussion on animal health and wildlife to 'welfare' and 'preservation of endangered species'. Animal welfare is, however, a most important issue. Current OIE animal welfare principles emphasize welfare of individual animals (OIE 2004bis). Yet, from an ecological point of view which is also stressed by conservationists, the individual perspective is not appropriate (cf chapter 2). In this study we have reviewed FMD and CSF. FMD has been implicated as the single most important constraint for trade in live animals and animal products, as well as the disease most feared in regions rich in modern, industrial animal production farms (Kitching, 2002). Given the importance of hogs in industrial production systems, CSF is also a good example of trade-anxiousness. Other former List A diseases (as well as some List B diseases) qualify for that reputation too, for example avian influenza, which has also zoonotic potential. During the epidemic of 2003 in the Netherlands, hundreds of thousands of production chickens and other poultry were exterminated, as well as thousands of pet-chickens and other privately kept birds, whereas little could be done about the possible transmission via wild birds. During this epidemic, a veterinarian died from infection with the avian influenza virus due to insufficient antiviral medication (Dutch government, 2003). Pigs are seen as the main intermittent for new human influenza strains (Wells et al. 1991, Rimmelzwaan et al. 2001) and it might be possible for avian influenza to develop into a serious public health threat due to multiple replication cycles in pigs and wild boars.
- Neither for FMD, nor for CSF wildlife constitutes a major threat to livestock. With only a few exceptions, eradication of FMD and CSF will cause endemic infection in wild populations to fade out. The risk of farm animals to wild populations is far greater. The conservationists'preferences for large self-sustaining populations, where endemic infectious diseases can freely run their course, are hardly realistic. There is no going back to extensive traditional farming, where outbreaks of highly contagious diseases like FMD and CSF would spread much slower than is currently the case. If wildlife populations and their home ranges are increased, which is a goal in nature policy, the risk from infected wild populations to livestock might increase as well. Non-eradication might be feasible for other diseases. If research turns out that this is indeed the case, non-eradication would be preferable. But a non-intervention policy for wildlife during an outbreak is not necessarily at odds with a long-term eradication strategy. Contingency plans for these diseases should take all possible precautions in order to prevent infection of wildlife or semi-wild populations. Local contingency plans for nature reserves to minimize transmission risk in either direction might be a good idea. In case a population becomes infected, preventive measures against infection of livestock from wildlife are often more feasible than mass vaccination or stamping out wildlife. Objections against vaccination, based on the ecological role of the disease should be re-examined in the light of the desirability of eradication. If there are vaccines and non-disturbing vaccination methods for wildlife available, applying them might be preferable over non-intervention, if it helps to eradicate a disease faster.
- Our evaluation of FMD and CSF indicates that there is room for a more nature-friendly approach. It also shows that there are still important knowledge gaps that need to be addressed. Especially the applicability of results found in domestic animals to wild species in a natural situation makes tailor-made protocols to better protect wildlife problematic. Stamping out wildlife during an outbreak is probably unnecessary in most situations. FMD has never become endemic, but CSF has. Yet, even where CSF is endemic, monitoring and adequate farm hygiene suffice to prevent transmission from wild boar to domestic pigs.

- An important obstacle to really implement protective vaccination, especially in animal-dense regions, is the length of the period before re-establishment of a disease-free status in a zone where vaccinated animals are kept alive. The difference in economic damage between stamping out and protective vaccination has decreased after the 2001 FMD epidemic, but has it decreased enough?
- New vaccines and better diagnostic techniques might give rise to more precisely targeted protocols involving vaccination as a control or prevention strategy. Animal products from vaccinated disease-free regions, if produced in conditions set out by OIE are safe for trade and consumption. Also, meat from animals vaccinated against CSF is only a risk when fed to pigs. As animal products, when fed to livestock, may pose a risk for many diseases, it would probably be helpful if OIE set out generic guidelines for the use of animal products in animal feed. Main trade restrictions for veterinary reasons concern the trade in live animals. Given the frequency of outbreaks of listed diseases, it might be desirable to generally restrict trade and transport of live animals in both vaccinated and non-vaccinated disease-free zones. It might be useful to examine the justification for trade barriers in the light of the current state of affairs and to take also into account other developments, such as the enormously increased trade and tourism volume. Unjustified trade restrictions are against WTO rules and WTO might do well to more closely examine the veterinary restrictions.

5.2 Recommendations

- It is recommended to extend the category of animals qualifying for protective vaccination to other non-production animals. Future guidelines should offer better protection to valuable animals in general, not only wildlife and zoo animals, but also breeding stock, and animals kept as pets, or for recreational purposes. There is a need to develop authoritative guidelines and protocols that take nature and wildlife into account. Given the OIE's willingness to play a leading role in this respect, LHF is advised to achieve consultative status with OIE.
- Since it is the EU's own nature policy that suffers from veterinary regulations without regard for wildlife, ecological infrastructure and de-domesticated species, LHF needs to lobby the EU as well. Apart from WWF, with which LHF has already close ties, there are wider NGO umbrella's, for example European Environmental Bureau (www.eeb.org) that could be helpful in this respect
- Research into practical implementation of the ecological infrastructure is needed, allowing for appropriate measures, like compartmentalisation in case of an outbreak
- To ensure implementation of nature-friendly guidelines, it is important to find ways of reducing infection risks, without compromising the disease-free status more than with a stamping out policy.
- Knowledge gaps as to transmission routes for priority diseases need to be addressed, especially with respect to the wildlife/ livestock interface
- More research into animal welfare ethics in relation to wildlife, including populations in various stages of de-domestication is urgently needed
- Given the importance of non-intervention in wild populations in case of an outbreak, it is recommended that decisions to eradicate a disease from the region, or decisions for interventions in wildlife will allow participation of ecologists and nature conservation NGOs.
- It is recommended to re-examine justification of veterinary trade barriers with respect to WTO rules against protectionism
- It is recommended that nature conservation organizations support the EU's and OIE's long-term goal of eradication of FMD, despite the fact that it is an endemic disease with an ecological role in wild populations. Modern farming systems are

too vulnerable for diseases as contagious as FMD. Perhaps it is possible to maintain the ecological role of CSF to regulate boar density in large nature reserves, provided that the wild boar can be effectively isolated from domestic pigs. A precondition is, that in case of an outbreak corridors have to be closed off completely.

- In general, isolation of wildlife from domestic animals is important, given the transmission risk of infectious animal diseases, including non-endemic diseases like African Swine fever to wild populations from domestic livestock.

5.3. Concluding remarks

Current veterinary rules are quite insensitive to nature restoration and conservation policy. Wildlife, for a long time only taken into account as a risk factor only, is now more and more seen as a responsibility, but at the moment still in a narrow and limited way. If OIE acknowledges the responsibility to help preserve endangered and valuable species, it is not too far-fetched to expect responsibility and support to nature conservation as an issue in its own right. This study shows, that even for the most fearsome of diseases, FMD and to a lesser extent CSF, there is probably room for a prevention and control policy that is less restrictive to wildlife. Conservationists and veterinary authorities should exchange their views and cooperate closely to achieve a new, more nature-friendly balance.

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Appendix

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Diseases formerly classified as List A

OIE code	**LIST A DISEASES**
A010	Foot and mouth disease
A020	Vesicular stomatitis
A030	Swine vesicular disease
A040	Rinderpest
A050	Peste des petits ruminants
A060	Contagious bovine pleuropneumonia
A070	Lumpy skin disease
A080	Rift Valley fever
A090	Bluetongue
A100	Sheep pox and goat pox
A110	African horse sickness
A120	African swine fever
A130	Classical swine fever
A150	Highly pathogenic avian influenza
A160	Newcastle disease

(Source: OIE, 2005. www.oie.int/hs2/report.asp)