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Classical Swine Fever in Wild Boar

Scientific Committee on Animal Health and Animal Welfare

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Glossary for this report

metapopulations   subpopulations with limited contacts with other subpopulations

epidemic   introduction of a highly contagious virus into a susceptible population followed by rapid spread limited in time and space

endemic   time unlimited chain of virus transmission in a certain geographic area

R_0   basic reproduction ratio of infection: average number of susceptible animals which are infected by one infected animal of a certain wild boar population

threshold level   number of susceptible wild boar, i.e. not immune to CSF, in a given area required to sustain the infection.

wild boar   the wild boar and the domestic pig are members of the same species *Sus scrofa*. Wild boar are native wild mammals in Europe but they can mate with domestic pigs, so fertile cressbred exist. Domestic pigs can also become feral. This report is concerned with uncontrolled populations of pigs in the wild, principally wild boar.

age classes   for the purpose of this report four age classes of wild boar were distinguished:

          0-3 months

          3 months-1.5 years

          1.5 years-2.5 years

          >2.5 years

1   REQUEST FOR OPINION

The Scientific Committee on Animal Health and Animal Welfare is asked to report on the eradication of Classical Swine Fever (CSF) in Wild Boar, including:

1. criteria for the surveillance of CSF in domestic pigs and for movement control measures for domestic pigs kept in areas where infected wild pigs exist

2. criteria for monitoring CSF in wild boar in an infected area and the effects of population levels on the disease.

3. factors which influence the evolution of CSF in wild boar in a given area, including control measures related to domestic pigs.

2   BACKGROUND

Classical Swine Fever (CSF) is a viral disease which has recently caused very serious economic losses in the European Union (EU). CSF virus infection occurs under natural conditions in all pigs, i.e. domestic pigs and wild boar (*Sus scrofa*), which are equally susceptible to CSF virus infection (BRUGH *et al.*, 1964; DEPNER *et al.*, 1995) and also causes poor welfare in affected populations.

After implementation of effective control measures in farmed pig populations, several countries, including Australia, Canada, New Zealand, United States of America (USA), succeeded in eradicating CSF. These measures succeeded in the absence of the disease in wild boar or feral pigs. In most other parts of the world the CSF virus is present, causing considerable economic damage. Within the EU, most Member States are practically free of the disease in domestic pigs.

CSF is currently present in parts of the wild boar population in several (geographic) regions of EU Member States, i.e. in France, Germany and Italy. It is also present in countries of Central and Eastern Europe that are likely to join the EU as well as in other Central and Eastern European countries (e.g. Switzerland). A survey of the situation of CSF in wild boar prepared by the OIE Regional Commission for Europe in 1998 is attached in Annex I. This actual situation might pose a threat for the pig industry of EU countries.

Sometimes the infection is self-limiting in the wild boar metapopulations (FERRARI *et al.*, 1998; FRITZEMEIER *et al.*, 1998) and sometimes the virus is circulating for years (LADDOMADA *et al.*, 1994; FRITZEMEIER *et al.*, 1998; KERN *et al.*, 1999). The role of wild boar as a virus reservoir and possible source of infection for domestic pigs is well known and epidemiological links between CSF virus infections in wild boar and domestic pigs have been reported repeatedly in recent years (WACHENDÖRFER *et al.*, 1978; KRASSNIG and SCHULLER, 1993; LADDOMADA *et al.*, 1994; TEUFFERT *et al.*, 1997). Virus isolation and molecular typing of CSF virus isolates have shown that CSF virus circulates and is possibly perpetuated for years amongst some European wild boar populations (FRITZEMEIER *et al.*, 1997, 1998). In the course of European CSF epidemics in wild boar the virus was transmitted from wild boar to domestic pigs due to direct and indirect contacts (TEUFFERT *et al.*, 1997; FERRARI *et al.*, 1998; FRITZEMEIER *et al.*, 1998). Taking this into consideration, prevention and control of CSF in wild boar is of prime importance in order to protect the domestic pig population.

Several attempts to eradicate CSF in wild boar, e.g. by reducing the population density or by experimental vaccination of wild boar using a live attenuated vaccine, so far have failed to yield convincing positive results.

In order to achieve a better understanding of CSF and its control in wild boar a meeting on measures to control CSF in European wild boar was held in Perugia, Italy in April 1998. The meeting was financially supported by the Commission of the EU. In addition, a subgroup of the Scientific Committee on Animal Health and Animal

Welfare was established. The task of this subgroup was to prepare recommendations on strategies for the control of CSF in wild boar.

The prerequisite and the basis of each promising strategy to control CSF in wild boar requires a thorough knowledge and analysis of the

a) wild boar ethology,

b) pathogenesis and course of CSF infection in wild boar,

c) influence of interfering human activities, e.g., feeding, hunting, vaccination.

Considering these parameters, useful strategies might be devised and implemented for each particular epidemic in wild boar.

3 ASPECTS OF THE ETHOLOGY OF WILD BOAR

The wild boar's basic social structure is a group of related adult females (more than 2 years old) and subadult females (ages between 1 and 2 years), accompanied by young males and females (less than 1 year old). This structure is stable during the summer, but is subject to considerable dispersion during the hunting season, where this exists. After this period, herds are constituted from the surviving members of the original ones. Young males and females become subadults and stay with the herd until the piglets are born (TEILLAUD, 1986, AHRENS, 1984, STUBBE and STUBBE ,1977, STUBBE, 1987).

Subadult males leave the herd either when the first adult females have their piglets or later when subadult females have piglets or in the course of summer. However, they stay in the native area and remain in contact with their herd. They do not start to disperse until the beginning of the hunting season in September, but once they do, rapid movements of over 15 km are observed (BAUBET *et al*., 1998).

Adult males tend to be more sedentary and do not leave the forest area (5,000 to 10,000 ha) in which they live. They roam over considerable distances during the mating season.

Subadult females may remain in their native herd for a second annual cycle or leave the herd either when the adult females have their litters or a short while afterwards when they themselves have them. They may either go back to their native herd with their piglets or constitute a new herd with other individuals from their herd. They do not associate with individuals from outside. In summer, subadult females although leaving their native herd temporarily or in order to constitute a new herd, remain in their native area, trying to stay within the home range of their native herd (AHRENS, 1984, JEANEAU and SPITZ, 1984, STUBBE *et al*., 1989).

If no animals are shot (e.g. in a reservation) or if the hunting pressure is low, association of surviving females among themselves may thus form herds of considerable size, attaining up to 30 or 40 individuals once the piglets are born. Conversely, when hunting pressure is high, especially among females more than one year old, small herds of 5 to 10 individuals are observed. The latter consist of one or two adult females with their offspring and a few subadults.

In summer, herds live in home ranges typically of 500 to 1,000 ha. When spatial distribution of food availability changes, home range sizes may increase, but no considerable movements are observed.

During the hunting season, where this exists, home range sizes increase. Movements (from 5 to 10 km) towards refuge areas with less hunting pressure are observed. Distances travelled increase with increasing hunting pressure and return movements are observed at the end of the hunting season. During these movements, herds are

stable when guided by adult sows which make best use of their knowledge of the area, using several refuges.

If the guiding sow disappears, the other adult or subadult females take over and lead the herd. However, if all the herd's adult or subadult females disappear, the youngsters are left to themselves and may move over distances of up to 50 km. Young surviving members of a herd are observed to remain together even during major movements and when they settle down.

4   CLASSICAL SWINE FEVER IN WILD BOAR

4.1 The virus

The causative agent of CSF is a small (40-60 nm) enveloped ribonucleic acid (RNA) virus with a single stranded RNA genome with positive polarity. CSF virus belongs to the pestivirus genus of the *Flaviviridae* family (WENGLER, 1991). The viral RNA codes for four structural and seven nonstructural proteins (ELBERS *et al*., 1996; MEYERS and THIEL, 1996). The virus is relatively stable in moist excretions and fresh and frozen meat and meat products, including ham and salami type sausages (SAVI *et al*., 1965; McKERCHER *et al*., 1978, McKERCHER *et al*., 1987; PANINA *et al*., 1992; MEBUS *et al*., 1993). However, it is readily inactivated by detergents, lipid solvents, proteases, common disinfectants and heat treatment. The virus can be transmitted from wild boar to domestic pigs and vice versa and has a broad spectrum of virulence.

The virus has been extensively analysed and biological and molecular tools for the development of new diagnostics and vaccines are available.

4.2 Clinical signs

The course of the disease, the pathogenesis of CSF virus infection, clinical signs and gross lesions have been extensively investigated in domestic pigs. Less is known about the course of the disease in wild boar. The manifestation of the infection may vary greatly depending on the age of the animal and viral virulence. In domestic pigs, after an incubation period of approximately one week, animals become febrile and may develop signs typical for CSF.

However, the clinical signs of CSF can be extremely variable (MOENNIG and PLAGEMANN, 1992; VAN OIRSCHOT, 1999). Infected pigs develop pyrexia and leukopenia. Severe thrombocytopenia is a consistent finding. Petechial haemorrhages of the skin and mucous membranes are the most typical signs although they are not observed consistently. Central nervous system disorders and constipation followed by diarrhoea may also be characteristic clinical findings. Adult animals may not show any sign of illness post-infection but they seroconvert, while young animals rather tend to develop the acute lethal form of infection. Therefore, mortality is rather low in adult animals and much higher in young animals. Apart from the animals´ age the clinical course of CSF may be influenced by the virulence of the virus and the condition as well as the constitution of the animals. Acute and chronic courses of CSF are known. All courses of the infection have in common that the animals are viraemic at least as long as they show clinical signs. Deaths occur two to three weeks after infection (acute course) or after up to three months (chronic course). Virus shedding occurs mainly during the clinical phase of disease, since young animals are clinically sick much more often than adults, they play a major role in the spread of CSF virus. Observations in wild boar show that at the beginning of an epidemic, all

age classes are affected and dead animals of different age groups are found, although younger animals constitute the majority. Later no more old animals are found dead, because the survivors of the infection are immune. Wasting, central nervous system disorders (ataxia, movement coordination), loss of fear and segregation are the most obvious signs.

In an endemic situation, almost only young animals (piglets) are affected and die. Field data from Germany based on 11,670 wild boar which were shot or found dead in the province of Brandenburg during the period March 1995 to December 1997 (KERN *et al.*, 1999) clearly demonstrated a gradual age-dependent decrease of CSF incidence in wild boar. Typically, the highest number of viraemic animals was found in the group of wild boar piglets younger than 3 months of age at the beginning of an epidemic. About 20 % of the wild boar piglets weighing less than 10 kg were viraemic, whereas no viraemic animals were found in the group of adult wild boar weighing more than 75 kg. These findings illustrate the important role young wild boar play in the epidemiology of CSF. Animals of this age group serve as a source of CSF virus within the wild boar population. Similar observations were made in Italy (GUBERTI *et al.*, 1998).

Like other pestiviruses, CSF virus may pass through the placenta of pregnant sows, infecting foetuses and leading incidentally to the birth of persistently viraemic piglets. The latter develop late onset CSF later in life and die from CSF. In domestic pigs the outcome of transplacental infection of foetuses depends largely on the time of gestation (VAN OIRSCHOT and TERPSTRA, 1977, VAN OIRSCHOT, 1979) and may result in abortions and stillbirths, mummifications, malformations or the birth of weak or persistently viraemic piglets (MEYER *et al.*, 1980). Although persistently infected offspring may be clinically normal at birth, they invariably die from CSF. In domestic pigs, survival periods of 11 months after birth have been reported. This course of infection is referred to as 'late onset CSF' (VAN OIRSCHOT, 1999).

While in postnatally acutely infected animals the period of virus shedding lasts only a few days until they either die or recover, chronically infected animals shed virus intermittently until they die. Persistently infected animals which became infected transplacentally or early in postnatal life usually shed virus for several weeks or even months before they die.

Experimentally, it has been shown that transplacental CSF virus transfer occurs also in wild boar resulting in persistently infected piglets. DEPNER *et al.* (1995) observed a postnatal survival time for a viraemic piglet of 39 days. Field data on the occurrence of persistently infected wild boar are not available, but it can be assumed that intrauterine infections occur in the field, especially in young adult females. But in contrast to domestic pigs, persistently infected wild boar might not survive for an equally long time because living conditions are much harder.

4.3 Pathology

Wild boar and domestic pigs display the same range of lesions after experimental infection. In postnatal infections, lesions are generally caused by widespread thrombosis or endothelial damage, inducing haemorrhagic diathesis and petechiation.

Pathological changes are most frequently observed in lymph nodes and kidney. The lymph nodes become swollen, oedematous, and then haemorrhagic. Haemorrhages of the kidney may vary in size from hardly visible petechiae to ecchymotic haemorrhages. They may be visible on the surface of the cortex. Petechial to ecchymotic haemorrhages can also be observed in urinary bladder, larynx and epiglottis, heart, intestinal mucosa, serous and mucous membranes, and skin. The skin may also become cyanotic.

Prenatally, at the early phases of ontogenesis, the virus affects organ differentiation which may lead to malformations.

In the field the most visible signs of CSF are the lesions described above. However, it takes knowledge and awareness of the observer (hunters, game keepers, etc.) to recognise typical signs and to interpret them properly.

4.4 Immunology and Vaccination

Little is known about the immune response of wild boar against CSF. However, it might be assumed that it is analogous to the immune response of domestic pigs. Like all pestiviruses, CSF virus is immunosuppressive during acute infection. Pigs that have recovered are protected against CSF for several years or even for their lifetime. They no longer serve as virus targets.

Neutralising antibodies are detectable two weeks after infection at the earliest. In pigs with chronic CSF, neutralising antibodies are detectable at the end of the first month postinfection for a few days and may disappear afterwards. Pigs infected *in utero* are immunotolerant to CSF virus and do not produce specific antibodies.

Passive immunity generally protects piglets against mortality during the first five weeks of life, but not against virus replication and shedding. Maternal antibodies have a half-life of about 14 days. Antibodies that are found after about 3 months of age can be assumed to be the result of an infection. Little is known about cell-mediated immunity against CSF virus.

In domestic pigs immunoprophylaxis has been an important instrument to control CSF. In the 1940's, the first trials were made to attenuate CSF virus by adapting it to rabbits (BAKER, 1946; KOPROWSKI *et al*., 1946). After initial setbacks this development ultimately led to a very efficient and safe generation of live vaccines. Most of these vaccines are based on the China-strain (C-strain) of lapinized CSF-virus. C-strain vaccines were and still are being used worldwide for the control of CSF. Currently, the C-strain is used in an oral immunisation field trial to control CSF in German wild boar. C-strain vaccines are highly efficacious, inducing neutralising antibodies and are safe when used in pregnant animals. Their efficacy is demonstrated by the observation that vaccinated pigs are protected against infections with virulent CSF virus as early as 5 days after vaccination, and probably for the rest of their life. However, there is a severe disadvantage of using live attenuated vaccines against CSF: vaccinated and field-virus-infected animals cannot be distinguished. The antibody pattern induced by the vaccine virus resembles that of reconvalescent animals; thus field virus infections may be masked by vaccination.

The use of a marker vaccine against CSF could overcome this dilemma, and a first generation of such vaccines – a subunit vaccine - has been developed in recent years. However, vaccines, which are to be applied parenterally, e.g. subunit vaccines, are not suitable for wild boar immunisation. So far, only conventional attenuated live vaccines are effective vaccines for oral immunisation. Improved, future live marker vaccines against CSF might have the potential for oral application, e.g., viral vector vaccines (RÜMENAPF *et al.*, 1991; VAN ZIJL *et al.*, 1991; HOOFT VAN IDDEKINGE *et al.*, 1996, VAN RIJN et al., 1996), or molecularly altered infectious cDNA clones of CSF virus (MEYERS et al., 1996, MOORMANN et al., 1996, RUGGLI et al., 1996).

4.5 Diagnosis

In wild boar, CSF diagnosis is mostly based on pathological and virological investigations of organ material or blood samples. Observation of wild boar meta-populations is important for the early suspicion and detection of CSF after introduction of the virus. Hunters, veterinarians and farmers must be highly aware of the risk of an infection. Abnormal mortality and sometimes obviously sick animals are first indicators of the introduction of CSF into a population. Laboratory confirmation of the suspicion of CSF according to the EU Council directive 80/217/EEC (EU, 1980) is required.

In order to discover CSF infected meta-populations at an early stage, it is important to identify the "right" animals for laboratory investigation. These are animals found dead, clinically sick and - rarely - growth retarded. They should be tested virologically. Random sampling of shot animals is suitable for epidemiological purposes, such as monitoring and serological prevalence studies (HANNOVER VETERINARY SCHOOL, 1998).

4.5.1 Virology

The "gold standard" for CSF diagnosis is virus isolation. CSF virus can be isolated from organ suspensions or body fluids of dead animals. Suitable organs are tonsils, parotid glands, lymph nodes, spleen and kidneys (EU, 1980, OIE, 1996). The virus isolation protocol requires at least three days and is labour intensive. A rapid diagnostic test for CSF is based on the demonstration of viral antigen in organ tissue sections using conjugated antibodies for direct immunofluorescence test (fluorescent antibody test, FAT) or similar techniques. To screen large numbers of animals, virus antigen capture enzyme-linked immunosorbent assays (AgC-ELISAs) can be used for the detection of viral antigen in blood samples. However, the latter tests are less sensitive than virus isolation.

Recently, detection of viral RNA has become an additional option for laboratory diagnosis. In particular, the 5'-nontranslated region of the genome has been used for amplification by the reverse transcriptase polymerase chain reaction (RT-PCR). Subsequent nucleotide sequencing of the respective region allows discrimination between different CSF virus isolates (HOFMANN et al., 1994; LOWINGS et al., 1996). The EU/OIE Reference Laboratory for CSF in Hannover, Germany, maintains

a large computer data base on CSF virus isolates, including epidemiological and virus type information data.

The 'molecular epidemiology' based on the above technique has become a useful tool and may well support epidemiological investigations (tracing on/back). Typing of CSF virus isolates of at least each primary outbreak in domestic pigs and isolates originating from wild boar provides useful information. Member States have been asked to send respective materials to the above-mentioned reference laboratory.

4.5.2   Serology

Serological diagnosis of CSF is important for the detection of the infection in wild boar populations, where acute clinical signs are absent. In addition, it is a tool for the monitoring epidemic and endemic disease situations.

The virus neutralisation test is the most sensitive and most reliable method for CSF antibody detection. Porcine serum samples are incubated with a CSF reference virus. If the serum contains antibodies for CSF the test virus will be neutralised. However, cross-neutralising antibodies specific for ruminant pestivirus infections of pigs are sometimes detected by this test. Differential diagnosis for ruminant pestiviruses should therefore be carried out using a second neutralising test with ruminant pestiviruses. The neutralisation test requires three to five days and is labour intensive. ELISA tests for the detection of antibodies are less time consuming and suitable for the screening of large numbers of sera. Positive or inconclusive results have to be retested using the neutralisation test, which is usually more sensitive and more specific.

4.5.3   Laboratory facilities

Most European countries are suitably equipped for the laboratory diagnosis of CSF. Almost all European countries with CSF laboratory facilities participate in annual inter-laboratory comparison tests in order to standardise and improve diagnosis. These tests are organised by the reference laboratories and the results are discussed at international conferences.

A list of addresses of National Swine Fever Laboratories of EU Member States is attached in ANNEX II. As representative for countries of Central and Eastern Europe the OIE Reference Laboratory for CSF in Pulawy (Poland) can be contacted.

4.6 Epidemiology

In countries that are free of CSF in domestic pigs, epidemics in wild boar are typically initiated by deliberate or accidental swill feeding. Often the farmers', hunters' and tourists' awareness of the risk and knowledge of the legislation are not adequate. Wild boar might pick up CSF virus contaminated material that had been thrown away at rest places and garbage dumps. In countries with CSF in domestic pigs wild boar might be infected also due to (indirect) contact to infected domestic pigs (carcasses, manure, etc).

Once introduced into wild boar, the virus is spread within the wild boar meta-population due to direct animal contacts and due to contacts with contaminated excretions and carcasses. Persistently infected piglets could contribute to virus circulation in wild boar.

From wild boar the virus may be transmitted directly (animal contact) or more often indirectly to domestic pigs (contaminated equipment belonging to farmers who are hunters as well, feed contaminated by wild boar excretions or carcasses, illegal swill feeding, contact of pigs with wild boar excretions etc.).

In Germany, all CSF virus types circulating in wild boar during the last decade have been isolated from domestic pigs in the corresponding geographic regions. Since 1993, 80 % of the 92 primary outbreaks were located in geographic regions where CSF is endemic in wild boar. For about 60 % of the primary outbreaks it was concluded that they were due to direct or indirect contact to infected wild boar and wild boar meat, respectively (HANNOVER VETERINARY SCHOOL, 1998). Similar observations regarding epidemiological links between infected wild boar and domestic pigs were made in Italy (RUTILI, 1997; FERRARI *et al.*, 1998)

When CSF virus is introduced into a wild boar population, usually a number of animals can be found dead, whereas others produce antibodies and survive. After the peak of the epidemic, the disease can die out spontaneously due to the acquired immunity of parts of the population and to the reduction of animal density due to mortality, respectively. From past experience it was thought that epidemics of CSF in wild boar are self-limiting. However, in recent years it was observed in several cases that epidemics turn endemic and that CSF virus persists in wild boar populations without signs of self-limitation. Possible reasons for the emergence of endemics are the involvement of viral strains of low virulence and the increasing density and size of the wild boar population. Endemic situations seem to become more prevalent in recent years (AUBERT *et al.*, 1994; DEPNER *et al.* 1998).

CSF virus is able to persist in a wild boar population only when a viraemic animal transmits the virus to at least one further susceptible wild boar (basic reproduction ratio: $R_0 \geq 1$). The more susceptible animals belong to a wild boar meta-population and the higher animal density in a certain area is, the higher is the probability that $R_0 > 1$ over a longer period of time. Principally, each animal which is not protected by antibodies against CSF virus is susceptible. In epidemic situations all animals are susceptible, whereas in endemic situations mainly young animals are susceptible.

Both the prolonged persistence of CSF virus in wildlife populations and the constant threat to pig holdings in the areas affected require an efficient control strategy.

4.6.1 *Threshold level of susceptible host animals*

Once the virus is established in a population, the goal of all control measures must be the reduction of susceptible animals in the affected area below a threshold level of susceptible hosts where R_0 is reduced to below 1 ($R_0 < 1$). However, no such strategy has been applied successfully so far.

The following factors influence the number of susceptible animals and thereby the threshold populations: size of the infected population, mortality of infected animals, natural immunisation, hunting, feeding (movement of animals), and oral vaccination.

4.6.2 Size of the infected population and virus circulation

The duration of virus circulation within an affected wild boar meta-population depends on the size and on the rate of reproduction of that meta-population. The larger the population, the more time is required to reach a prevalence of immune animals which is necessary to reduce the number of susceptible individuals below the threshold level. At later stages of a progressing epidemic, the number of infected animals is less important than the number of susceptible animals, because the latter are crucial for the outcome of an epidemic.

The period during which the epidemic is likely to last is approximately directly related to the size of the population. On the basis of several experiences 1,000 wild boar (at a population density of 2-5 animals/km^2 and in areas where hunting is allowed) can maintain the infection for approximately one year. After 12 months the epidemic is likely to fade out or to become endemic. It may be assumed that if hunting were forbidden for 12 months the infection could fade out earlier or not become endemic, respectively. That means that in populations smaller than 1,000 head the infection is likely to persist for shorter periods.

Once the estimated time period to extinguish the epidemic is over, an assessment of the seroprevalence should be carried out. The purpose of this investigation is to collect epidemiological information. A small number of skilled persons, who have been trained to handle carcasses of shot animals correctly, should collect the samples.

4.6.3 Mortality of infected animals and virulence of viral strains involved

The mortality due to CSF virus infection depends on the virulence of the virus and to a large extent on the age of the infected animals. The infection might cause up to 90 % mortality in piglets. A decrease of deaths is observed with age. Moderately virulent virus isolated from recent epidemics in both wild and domestic pigs kills only few adult animals. Highly virulent strains spread faster than viruses of lower virulence because the former replicate more efficiently in infected animals and high virus doses are excreted by infected animals (DAHLE AND LIESS, 1995). The virulence of the virus affects the shape of the epidemic curve and consequently the level of endemicity. Moderately virulent strains - inducing chronic CSF - are excreted by individuals for longer periods and therefore a smaller number of susceptible hosts is required to sustain the virus in the population. In any given condition an absolute number of susceptible animals exists under which the infection is unlikely to spread further. During an epidemic caused by a high virulent strain this number has been estimated to be 270 (i.e. 1.4/km^2) (HONE *et al*., 1992). In endemic areas with low virulence strains of CSF-virus this number has been estimated to be 207 (i.e. 1/km^2.). The threshold number is strongly affected by the mortality rate due to the infection (GUBERTI *et al*., 1998). The length of the time period during which restrictions are to be maintained is related to the size of the population involved (RUTILI *et al*., 1998).

In endemic areas, transplacental infections leading to persistently infected piglets are unlikely to occur at a substantial rate due to the low incidence of new infections in older animals. Additionally, persistently infected animals have a short life expectancy. Thus it appears unlikely that persistently infected animals play a significant role in the maintenance of the virus.

4.6.4 Natural immunisation and vaccination

After an outbreak of CSF and during the progression of the epidemic more and more adult animals acquire natural immunity against CSF virus. In addition, in the course of a CSF epidemic oral vaccination using baits might be used in order to further reduce the number of susceptible animals. The success of vaccination campaigns largely depends on the strategy of bait delivery. Young animals are the main target group for vaccination. However, so far seroconversion rates observed in young animals are poor, possibly because older animals are more likely to pick up the baits (KADEN, 1998; KERN, 1998; KERN et al., 1999).

4.6.5 Hunting

Recent experiences show that indiscriminate hunting fails to reduce the wild boar population to the threshold level. Wild animal species tend to saturate the carrying capacity of their habitat. Due to density dependent processes, every population substantially reduced by hunting, will have an increased reproductive possibility. When hunting decreases the population will often return to the carrying capacity. The effect is that a large number of young animals will be born. The latter are usually the most susceptible individuals that will develop the clinical form of infection with high fatality rates and that will maintain the virus in that population. In endemic areas adult animals show a high level of herd immunity. Therefore the killing of older (immune) animals reduces the population immunity as observed in Mecklenburg-Western Pommerania and Brandenburg, Germany (DEPNER et al., 1998; KADEN, 1998).

In addition, hunting increases the movement of individuals in their home ranges and this may lead to their dispersion (MAUGET et al., 1984). If animals are not disturbed, they will have less contact with other susceptible animals. This implies that the size of the affected population is smaller and the threshold level at which the disease dies out might be reached earlier due to natural immunisation. The shooting of older animals leads to a dispersion of the social groups, whereas shooting of young animals does not.

Trapping of young wild boar using cage traps should be taken into consideration in order to reduce the number of susceptible animals in the area.

4.6.6 Feeding

Feeding of wild boar is a possible source of virus introduction in the wild boar population when meat, meat products or kitchen waste is fed. After an outbreak all measures that might induce movement of animals must be banned. Feeding may contribute to further spread and perpetuation of the virus in the wild boar population because it leads to intensified movement of wild boar. Feeding places are visited by animals whose normal home range is far away (LOEPELMANN and SCHUSTER,

1997). Direct or indirect contacts between different wild boar groups are intensified due to common feeding places. The consequence would be an enlargement of the meta-population at risk. An additional effect of feeding might be an increased fertility being the result of increased weight and the improved general condition of the females (GAILLARD et al., 1993).

5 RECOMMENDATIONS FOR CONTROL MEASURES

In contrast to the situation in domestic pigs, control of CSF by depopulation of wild boar is rarely practicable. The recommended control strategies are not based on experimental or field trials and therefore should not be regarded as rigid guidelines. They are cumulatively derived and combined from experiences with single measures which have been applied. A distinction is made between the strategy to be applied after an outbreak followed by an epidemic and the strategy to be used in endemic situations, respectively. According to the above mentioned factors the two situations will be dealt with separately. However, some of the measures apply for both situations (e.g. public awareness campaigns).

5.1 Control strategy after outbreaks and in epidemic situations

When CSF virus is introduced into a wild boar meta-population, control measures should be established which take into account the local geographical and epidemiological situation. The measures might vary according to differing circumstances. The aim of all measures must be the prevention of further spread of the infection and the reduction of the reproduction ratio to values below 1 ($R_0 < 1$) in order to stop the epidemic, i.e. the reduction of susceptible non-immune animals. This can be attempted by the following measures.

5.1.1 Public awareness and education

Measures to increase public awareness for the disease are essential for the success of the control policy. Veterinary authorities, hunters, farmers and the public must cooperate for the control of CSF in wild boar. Therefore these groups should be educated properly in order to be aware of the risks and suitable control measures.

5.1.2 Defining a controlled area for the wild boar population at risk

In order to develop an effective control and eradication plan, the area and the wild boar population at risk have to be analysed and defined. Usually CSF infection in an area is detected after dead animals have been found that turned out to be virus positive. The time elapsing from the actual introduction of the virus into the population and the detection of the problem by hunters varies from case to case. The area at risk has to be defined and controlled as soon as possible because the infection is evolving in a given area. The area of risk should not be based on individual home ranges of wild boar, but rather on the geographical area covered by meta-populations taking into account the presence of effective natural boundaries, e.g. major highways, rivers, high mountains and feeding conditions, respectively. For safety reasons defined areas should not be made too small. Defining the area and the number of wild boar at risk should be carried out by experts with adequate competence, e.g., wildlife biologists, hunters, veterinarians and epidemiologists. Before any further action is taken the strategy for the control of CSF in the area(s) of risk should be carefully planned.

5.1.3   Reduction and prevention of movement of animals

During the course of an epidemic any disturbances, dispersion of animals and the associated spread of the virus must be avoided. Therefore any hunting activity - even for species that are not susceptible to the infection – has to be stopped. Feeding must be banned at all stages of the epidemic in order to prevent increased migration of animals. Except that, where low level feeding has taken place, this should be maintained, in order to prevent emigration from the area by animals in search of food.

5.1.4   Investigation in the infected area and in the periphery

Once the infection is confirmed surveillance in the defined area at risk should focus on the detection of dead animals, their removal and investigation in diagnostic laboratories. The main objective at this point in time is to assess the spatial diffusion of the infection. Serological investigations in the area at risk should follow at 6 month intervals according the criteria outlined above.

During the period of suspension of hunting activity in the area at risk, surrounding territories should be adequately investigated in order to detect the presence of the disease, both with virological investigations on animals found dead and by serology on animals shot according to the criteria described above.

Four possible scenarios are likely to occur as a result of an epidemic:

a) Extinction of the infection in the risk area and no further spread to surrounding areas. This usually happens when the infection has spread over a small area (approximately 200 km^2 with 400 to 1000 wild boar) and when the overall seroprevalence in wild boar varies from 35 % to 75 %.

Actions to be taken: Lifting of the restrictions. Hunting can be resumed and proper investigations of carcasses for CSF virus should be performed for at least one additional year. Serological investigations should be focused on young individuals (3-6 months) that can easily reveal recent cases of infection (see Chapter 6).

b) Persistence of the infection in the area with no cases outside. This normally occurs when the infected area is large and contains more than 1000 wild boar, cases of mortality still occur and serology reveals a low seroprevalence so that the estimated number of susceptible hosts is higher than 200.

Actions to be taken: Restrictions should last until the monitoring programme indicates that the threshold of 200 susceptible animals has been reached. Concurrently, a high level of surveillance in neighbouring areas should be maintained.

c) The infection has been recognised outside the area and also persists in the primarily infected one. This usually occurs when the infected area was not designed correctly, e.g. when some epidemiologically important factors were underestimated or not taken into consideration, respectively.

<u>Actions to be taken</u>: The infected area must be extended and redefined. In case the new area is larger than 200 km^2 it is necessary to subdivide the territory into subareas of about 200 km^2. The results of the monitoring according to the sample size required should be correlated with the subareas (see Chapter 5).

d) The infection died out in the infected area but cases of infection are detected in surrounding areas. This can occur when the infected area is small and no effective barriers are present in the territory. This stresses the importance of accurately defining the infected area.

<u>Actions to be taken</u>: Redefining the infected area with the same actions described under c).

Scenarios c) and d) are more likely to occur when hunting activity is not suspended.

5.2 Control strategy for endemic situations

In some areas of Europe the size of the territories involved in CSF infections in wild boar is very large (3,000-5,000 km^2). The emergence of large infected areas is normally the result of a long time of viral persistence in a certain wild boar metapopulation, as well as of intense hunting and low virulence of CSF viral strains involved. The management of these areas is particularly difficult and it is possible that the eradication of CSF will take several years.

5.2.1 *Determination of the dynamics of the infection in areas and subareas by age-stratified and GIS (Geographical Information System)-based serology*

Repeated serological investigations of large numbers of samples taken from the whole endemically infected area usually yield an overall low seroprevalence. However, as the infection may move back and forth within an area, the interpretation of the serological data alone does not give a complete picture. Instead the total area should be divided into subareas of e.g. 200 km^2 (with 400-1,000 animals each) and the age-specific seroprevalence in these different subareas should be analysed. Most likely the incidence of fresh CSF cases is not homogeneously spread over the entire area. Instead clusters of acute infections will be identified in smaller areas and low levels of fresh infections in others. The aim of this type of investigation is to pinpoint subareas where the virus is continuously maintained, thus serving as an endemic source of fresh infections for the surrounding subareas.

5.2.2 *Permanent endemic area*

The interpretation of sequential serological studies could be either that an area is permanently endemic or that it is or was temporarily endemic representing merely a segment of an ongoing epidemic wave. The verification of a permanent endemic area is reached by assessing the results and the profile of repeated (e.g. 6 months intervals), age stratified serological investigations. For permanently endemic areas this will result in identical slopes according to the distribution of the seropositivity in the different age classes (GRENFELL and ANDERSON, 1985). Once a permanently endemic subarea has been identified, restriction and control measures - like in an epidemic situation - should first be applied in this subarea.

5.2.3 *Epidemic waves*

If the infection is spreading in epidemic waves the serological profile and the infection rate of subareas will show different patterns from year to year. From and through these subareas the infection will spread in intervals with an unpredictable pattern according to the different wild boar numbers, habitat and dynamics. The case where the mode of transmission is such that epidemic waves are sweeping through the whole area the control of the infection in wild boar is particularly difficult and the use of oral vaccination might be a suitable additional tool.

5.2.4 *Oral vaccination*

Vaccination programmes should be targeted to the specifically identified metapopulation maintaining the infection in the wild boar in order to reduce the number of susceptible animals below the threshold of transmission (ANDERSON and MAY, 1992; BAGON et al. 1996). In wild boar it is supposed that the threshold density for CSF is reached by a number of about 200 susceptible animals in about 220 km^2 (GAILLARD et al. 1993). The required efficacy of vaccination is a function of the estimated population. If 1000 wild boar are present seroconversion rate in 80 % of the animals should be reached. If only 500 wild boar are present the rate should be at least about 60 %.

Vaccination programmes should last for at least two years. The efficacy can be properly investigated using criteria described in chapter 6. Application of oral vaccination is expected to decrease the number of susceptible host animals. In the "vaccinated" areas, hunting should be reduced or forbidden in order to maintain as long as possible a stable population in which the majority of animals is immune. Hunting might lead to a high turn-over of the population (MORETTI, 1985) and females tend to have more than one heat period per year, especially when their progeny are shot. As a result, a decrease of the effect of vaccination could be observed (ANDERSON and MAY, 1985). If selective hunting is applied, potentially immune (older) animals should be saved and potentially non-immune animals (young pigs, under 6 months) should be removed (shooting or cage trapping). Repeated or continuous serological sampling and evaluation should be performed.

5.3 CSF control in a wild boar population which is in a transient phase of infection

An area, which is in a transient phase from CSF infection to a CSF-free status is characterized by the observation that no CSF virus or viral antigen was found during the last 12 months. The wild boar population is CSF antibody positive in piglets (<3 months of age; maternal antibodies) and adult animals only. Pigs aged over 3 months remain seronegative after the disappearance of maternal antibodies.

Monitoring measures should be continued for three further years in order to make sure that the CSF virus has been eradicated. A serological survey should be carried out in the wild boar population. The majority of animals who are tested for antibodies against CSF should be young animals older than 3 months. All wild boar found dead are to be investigated for CSF virus or viral antigen (see 6.5). Killed animals must be investigated likewise when displaying suspicious clinical or *postmortem* signs.

5.4 CSF control in free-status area

An area may be considered free from CSF in wild boar when:

– CSF virus has not been present in wild boar during the last twelve months

and

– the wild boar population is CSF antibody free.

The "free status" of the wild boar population should be investigated and documented annually (see 6.5). The feeding of swill to wild boar must be forbidden, unless the swill is treated according to a licensed procedure for the inactivation of CSF virus (e.g. heat treatment). Only animals found dead should be tested routinely for CSF viral antigen. The threshold for sending in laboratory samples for a CSF exclusion diagnosis must be low, but should not immediately result in CSF suspicion with all its negative consequences.

5.5 Restrictions on domestic pigs in case of an outbreak of CSF in wild boar

CSF epidemics and endemics in wild boar pose a serious threat to domestic pigs. The following restrictions are recommended:

– effective separation between wild boar and domestic pigs;

– direct and indirect contact of wild boar with domestic pig holdings is to be avoided (e.g. by double fencing, hygiene and public awareness);

– movement restrictions for wild boar (no trade of live wild boar);

– trade restrictions for wild boar meat. Wild boar carcasses must remain in the affected zone;

– pathological material and biological products derived from wild boar have to be processed to ensure the destruction of CSF virus;

– movement restrictions for domestic pigs. In principle domestic pigs (or piglets) could remain in the area (there is still an element of risk involved). However, herds should be investigated clinically (suspicion $\Rightarrow$ virological investigation) before animals leave the zone affected by CSF in wild boar.

The following additional measures should also apply to outdoor holdings:

– establishment of new outdoor holdings for domestic pigs should be forbidden;

– existing outdoor holdings are to be reported to the veterinary authorities. They have to be organised and run in a manner that a direct contact between wild boar and domestic pigs is prevented. In particular, a double

fence should be built for this purpose (with at least 1 m between the fences);

– the farmer must provide sheds in order to keep the domestic pigs in closed facilities in case of a high risk of CSF virus infection (virologically positive wild boar in the vicinity).

5.6 Control measures in case of an outbreak in domestic pigs in an area with wild boar

In case of a CSF outbreak in domestic pigs, the infection must be eradicated according to the Directive 80/217/EEC. Geographical, epidemiological and logistic facts of the area have to be considered, when establishing the protection and surveillance zones.

– Monitoring (clinical observations, virological/serological investigations) in wild boar should be intensified (see chapter 6.);

– possibly infective material (carcasses, manure, etc.) unless inactivated must be kept inside the premises.

Farmers, hunters and veterinarians should be informed about risks of spreading the disease.

6   MONITORING

Each wild boar population should be monitored on a regular basis whether it is regarded as infected or not.

- Serological and virological investigations are the only tools available to gain insight in the evolution of CSF into a wild boar population. Therefore it is of uppermost importance that these activities are properly planned in order to obtain a solid data base essential for the management of the infection.

- It is important to define the aims of the sampling plan beforehand.

Possible objectives might be:

- detection of the infection in areas where it is not known if the virus is (or it has been) present or not

    - assessment of the seroprevalence in infected areas

    - monitoring of the evolution of the infection in affected areas

6.1 Target population

The target population to be sampled should first be identified in order to determine the sample size which should be based on the estimated number of wild boar living in the area.. Because most of the time data on population density and size are not available, it is simpler to estimate a geographic area within which to sample. The geographic area should be related to the continuous presence of wild boar and chosen according to the presence of natural or artificial barriers (such as rivers, major highways, etc.) efficient to prevent large and continuous movement of the animals. When such circumstances are not present sampling areas of approximately 200 km^2 should be used. In areas where wild boar are managed usually the density ranges from 2 to 5 (or more) wild boar/km^2; so a rough estimation of the population size ranges at least from 400 to 1000 wild boar per subarea (e.g., Brandenburg and Sardinia; data in: KERN, 1998, GUBERTI et al., 1998).

6.2 Samples

When CSF virus is circulating in a population of wild boar the probability of detecting infected animals is rather low, because at a certain time only a small proportion of animals is acutely infected. Only in those animals virus or antigen detection is successful.

So it seems more reliable to use serum samples collected from hunted animals, or according to specific sampling activity within a surveillance scheme, to assess the presence of antibodies.

Serology could give more detailed information, if additional data on sex and age of the sampled animals are collected.

6.3 Detection of the infection

The level of the infection that one wants to detect must be established in advance. According to the epidemiology of CSF in wild boar a seroprevalence of at least 5% should be reached in short time after an outbreak. Consequently this could be chosen as the level of the infection to be detected. For this purpose a sample of 59 animals is a sufficient number to detect at least 1 case of seropositivity with a confidence level of 95% if the prevalence is 5% or more (COCHRANE 1977). The criteria of sampling individuals must be randomised (CANNON and ROE, 1982).

The sensitivity and specificity of the diagnostic test is to be taken into account and - if necessary – the sample size should be corrected. In any case 59 is the minimum number of animals to be sampled.

6.4 Assessment of the seroprevalence

This method of sampling should be performed in endemic areas to assess the prevalence of the infection. If no information is available at all or previous data are not consistent a sample size of 96 animals should be taken (confidence level = 95%).

In areas where hunting is usually carried out 50% of the sampled animals should belong to the 3 months-1.5 year age class, 35% to 1.5-2.5 years age class and 15% to more than 2.5 years age class.

In areas where hunting pressure is very low or absent, 32 animals should be sampled for each one of the three age classes.

This way of sampling takes into account the usual population structure observed in hunted and non-hunted populations (MORETTI, 1985; ZILIO and PEDROTTI, 1998).

6.5 Monitoring of the disease in infected areas

The same sample size used for the assessment of the seroprevalence should be used. In this case it is of primary importance to study the serological profile of the population to determine whether the infection persists at an endemic level or whether there is evidence of a trend that might indicate the fading out of the infection. In this case the age structure of the sampled animals is the key point for the understanding of the evolution of the disease.

A general rule is that the collection of samples should be performed in a short time, approximately 1 month, using animals shot during specific programmes of surveillance activity. It is documented that hunting carried out for short periods does not significantly affect the level of movement of individuals (MAILLARD and FOURNIER, 1995; GENOV and FERRARI, 1998). Age classes should be identified according to tooth eruption and not on the size of the animals which is subjected to larger bias (BOITANI and MATTEI, 1992). The use of a questionnaire is recommended where additional data, e.g., on the place where the single animal has been shot, etc. must be reported.

In large areas where the absence of clearly defined natural barriers does not allow a further subdivision in smaller regions, the area should be divided into areas of approximately 200 km^2. In each one of these areas, individuals must be sampled following the above mentioned criteria and according to the objective of the sampling. Results should be reported for each of the areas, avoiding large data set (i. e. prevalence of 0.3 % on 3,500 sera sampled) and maintaining age structured data according to four age classes (0-3 months; 3 months-1.5 years; 1.5-2.5 years; >2.5 years if age is observed during autumn-winter).

6.6 Monitoring in domestic pigs

Increased awareness and clinical examination of pig holdings situated in the zone infected with CSF in wild boar. Suspect animals and aborted foetuses must be subjected to the diagnostic tests for CSF virus or viral antigen. The veterinarian has to send samples for the exclusion of CSF. It is vital that sick pigs (fever) are notified to the practitioner.

The following measures should be carried out:

– domestic breeding pigs in the infected zone of wild boar situated in a CSF free country should be subjected to an increased serological survey for CSF antibodies;

– each dead pig should be notified and investigated;

– systematic investigation (or at least a representative sample) for CSF virus in rendering plants for dead pigs;

– in outdoor holdings each case of high fever and each pig that died in suspicious circumstances must be tested for virus;

– very small holdings can pose a high risk (e.g. poor biosecurity measures and lack of official control). In the case of an outbreak and in case of an endemic situation each of the small-holdings (1-2 pigs) must be reported and registered by the authorities;

– pigs from small-holdings should be investigated serologically when slaughtered.

7 AREAS OF FUTURE RESEARCH

- So far there is no proven successful strategy for the control and eradication of CSF endemic in wild boar. The above recommendations are based on current knowledge and experience. In addition, there are a number of open questions, that should be dealt with in order to improve the prospects of combating the infection in wild boar more effectively. Research on this area should be encouraged.

- Suitable control measures for different geographical ('open' flat area or 'closed' region, e.g., in mountains) and epidemiological situations are:

  - oral vaccination;

  - improvement of bait delivery with the goal of higher seroconversion rates in young animals;

  - influence on seroprevalence of different age groups on the progression of the infection;

  - improvement of vaccination technology, e.g. development of live marker vaccines;

  - effect of different hunting strategies on control of CSF;

  - effect of different hunting strategies in endemic situations;

  The main questions are:

  How does hunting influence the movement of (infected) wild boar?

  How does hunting influence the population size?

  How does hunting influence the age structure within the population?

  How does hunting influence the prevalence of CSF?

- Epidemiology

  - number of wild boar which are necessary to maintain the infection

  - factors affecting the geographical diffusion of the infection

- Optimizing monitoring schemes

- Transmission of CSF virus from wild boar to domestic pigs

8   SUMMARY

The Member States of the European Union are free from Classical Swine Fever (CSF) in domestic pigs. However, the occurrence of CSF in wild boar in some areas of the EU gives rise to concern because it poses a threat to domestic pigs. Whereas empirical data from earlier CSF epidemics in wild boar showed a tendency for the infection to die out over time, CSF outbreaks in the early 1990's apparently became endemic and have persisted for years. Possible causes of this change are the increasing density of wild boar populations and the emergence of viral strains of moderate virulence.

Research on the ethology of wild boar during the last 20 years has shown that the animals live in stable social structures of young males and females led by adult females. Indiscriminate hunting disrupts this structure causing animals to migrate, whereas selective hunting of young individuals leaves the group structure intact. Normally subadult males leave the herd, stay in their native area and remain in contact with their herd, unless they are dispersed by hunting activity. The search for food or feeding causes wild boar to migrate over long distance, i.e. having contact with other groups.

The usual way of introduction of CSF into a wild boar population is via feeding of swill. Initially all age classes are affected. However, as in domestic pigs, the infection kills primarily young animals, which are found in the field as early indicators of an epidemic. Older animals may survive the infection and develop a long lasting stable immunity. Later stages of the epidemic are characterised by a high proportion of immune older individuals and fresh infections occurring only among young non-immune animals. When the threshold level of susceptible wild boar drops below a critical value of approximately $1/km^2$ the basic reproduction ratio (R_0) is <1, i.e. the infection will die out. A higher number of susceptible animals will lead to an endemic situation. The latter situation is likely to occur in densely populated wide open areas without effective natural boundaries.

So far there are no strategies known for the efficient control of endemic CSF in wild boar. The recommendations for control strategies given in this paper, and summarised below, are not based on experimental or field trials and therefore should not be regarded as rigid guidelines. They are cumulatively derived and combined from experiences with single measures which have been applied in the field.

Recommendations

- Control measures must be implemented immediately following the detection of an outbreak.

- The area of risk must be defined taking into account the habitat of wild boar metapopulations and natural boundaries.

- Since co-operation of farmers, hunters and the public is essential for the success of a control programme, an awareness and education programme must be launched.

- In epidemic situations it is essential to take action in order to prevent the movement of animals. This involves a ban on hunting for the acute phase of the epidemic and ban on feeding the wild boar. However, where low level feeding was practised it should be continued in order to prevent local wild boar from emigrating in search of feed. The infection will be self limiting provided that the threshold level of susceptible animals is not exceeded ($R_0<1$).

- At all stages the movement of the infection should be monitored by virological investigation of animals found dead and by serological investigation of animals shot in adjacent areas.

- Since an endemic situation is likely to occur when the affected area has little or no effective boundaries to separate neighbouring wild boar populations and when population density is high, for the purpose of observation large areas should be sub-divided on a map into sub-areas of apporximately 200 km^2. The epidemiological status of each subarea should be monitored separately in order to identify possible permanent endemic areas or epidemic waves, respectively. In any case the goal is to reduce the number of susceptible wild boar to below the threshold level to reach an R_0 value of <1. This may be achieved by a combination of measures, e.g. selective hunting of young (non-immune) wild boar, reduction of the number of young animals by cage trapping and by oral immunisation of young individuals, respectively.

- Because CSF epidemics and endemics in wild boar pose a serious threat to domestic pigs, a number of restrictions must be imposed in order to prevent the infection of domestic pigs in the area at risk and to prevent the spread of the virus beyond the infected area. In rare cases outbreaks of CSF occur in domestic pigs in areas where wild boar are living. In these cases spread of the virus to the wild boar population, via e.g. manure, must be prevented.

- The success of control measures largely depends on a sound monitoring protocol. Virological and serological monitoring yields information about the epidemiological status of affected subareas and areas. Positive virological findings in young animals found dead or shot are indicative of an ongoing infection in the subarea. Likewise serologically positive samples in young wild boar (3 months-1.5 years) are evidence for recent infections. Serologically negative animals (>3 months) are an indication for the absence of CSF virus in the subarea.

So far there is no proven successful strategy for the control and eradication of CSF endemic in wild boar. The above recommendations are based on current knowledge and experience. In addition, there are a number of open questions, that should be dealt with in order to improve the prospects of combating the infection in wild boar more effectively. Research should be encouraged, e.g. iimprovement of oral vaccines (live marker vaccine), immunisation protocols (targeting young individuals), effects of hunting on wild boar populations, epidemiology of CSF in wild boar and modes of transmission of CSF from wild boar to domestic pigs.

9 REFERENCES

Ahrens M. (1984). Untersuchungen zur Reproduktion beim Schwarzwild. *Beiträge zur Jagd- und Wildforschung* **13**, 231-243

Anderson R.M. and May R.M. (1985). Vaccination and herd immunity to infectious diseases. *Nature* **318**, 323-329.

Anderson R.M. and May R.M. (1992). Infectious diseases in Humans. Dynamics and control. *Oxford University Press*. Oxford.

Aubert M., Picard M., Fouquet E., Conde J., Cruciere C., Ferry R., Albina E., Barrat J.,Vedeau F., (1994). La peste porcine classique du sanglier en Europe. *Ann. Med. Vet.* **138**, 239-247.

Bagon M., Harper J.L., Townsend C.R. (1996) Ecology. Individuals, Populations and Communities, Blackwell Science, Oxford

Baker J.A. (1946). Serial passage of hog cholera virus in rabbits. *Proc. Soc. Exp. Biol. Med.* **63**, 183-187.

Baubet E., Brandt S. and Touzeau C. (1998). Effet de la chasse sur les stratégies d'occupation de l' espace des sangliers (Sus scrofa). Analyses preliminaires. *Gibier Faune Sauvage, Game Wildl*. Vol. 15 (Hors serie Tome 2), 655-658

Boitani L. and Mattei L. 1992. Ageing wild boar (*Sus scrofa*) by tooth eruption. In: Ongulés/Ungulates 91. *Proceedings of the International Symposium*. Spitz F., Janeau G., Gonzales G., Aulagnier S. (Eds.). S.F.E.P.M. - I.R.G.M., Paris, Toulouse: 419-421.

Brugh M., Foster J. W. and Hayes F. A. (1964): Studies on the comparative susceptibility of wild European and domestic swine to hog cholera. *Am. J. Vet. Res*. **25**, 1124-1127.

Cannon R.M. and Roe R.T. (1982). Livestock disease surveys. A field manual for Veterinarians. *Australian Government Publishing Service*. Canberra.

Cochrane W.G. (1977) Sampling techniques. John Wiley and Sons, London.

Dahle J. and Liess B. (1995): Comparative study with cloned classical swine fever virus strains Alfort and Glentorf: Clinical, pathological, virological and serological findings in weaner pigs. *Wien. Tierärztl. Mschr*. **82**, 232-238.

Depner, K.R., Granzow H., Kern B., Liess B., Müller P. (1998). Schweinepest – Uneingeschränkte Jagd ist kontraproduktiv. *Wild und Hund* **15**, 34-39.

Depner K.R., Müller A., Gruber A., Rodriguez A., Bickhardt K. and Liess B. (1995). Classical swine fever in wild boar (*Sus scrofa*) - experimental infections and viral persistence. *Dtsch., Tierärztl. Wochenschr*. **102**, 381-384.

Elbers K., Tautz N., Becher P., Stoll D., Rümenapf T. and Thiel H.J. (1996). Processing in the pestivirus E2-NS2 region: identification of proteins p7 and E2p7. *J. Virol*. **70**, 4131-4135.

EU (1980). EU Council Directive 80/217/EEC of 22 January 1980 introducing Community measures for the control of Classical Swine Fever, last amended 14 June 1993.

Ferrari G., Guidoni M., Amaddeo D., Autorino G.L. and Forletta R. (1998). Epidemiology of CSF in wild boar in Toscana. In: Report on measures to control classical swine fever in European wild boar, Perugia, Italy, Commission of the European Communities, Document VI/7196/98-AL, pp. 107-109.

Fritzemeier J., Greiser-Wilke I., Depner K. R. and Moennig V. (1997): The epidemiology of CSF in Germany between 1993 and 1997. In: Report on Annual Meeting of National Swine Fever Laboratories, Vienna, Austria, 16-17 June 1997, Commission of the European Communities, Document VI/7888/97, pp. 33-35.

Fritzemeier J., Greiser-Wilke I., Depner K. and Moennig V. (1998). Characterization of CSF virus isolates originating from German wild boar. In: Report on measures to control classical swine fever in European wild boar, Perugia, Italy, Commission of the European Communities, Document VI/7196/98-AL, pp. 107-109.

Gaillard, J.M., Brandt, S. and Jullien, J.M. (1993). Body weight effect on reproduction of young wild boar (*Sus scrofa*) females: A comparative analysis. *Folia Zoologica* **42**(3), 204-212.

Genov P. and Ferrari G. (1998). Effect of hunting on the use of space and habitat of wild boars. In. "Measures to control Classical Swine Fever in European Wild Boar" Perugia, Italy, 6-7 April 1998. European Union. Commission des Communautés Européennes. Direction Générale VI Agriculture, 32-36.

Grenfell B.T. and Anderson R.M. (1985). The estimation of age-related rates of infections from case notifications and serological data. *J. Hygiene* **95**, 419-436.

Guberti V., Rutili D. Ferrari G. Patta C. and Oggiano A. (1998). Estimate of the threshold abundance for the persistence of the classical swine fever in the wild boar population of eastern Sardinia. In: Report on measures to control classical swine fever in European wild boar, Perugia, Italy, Commission of the European Communities, Document VI/7196/98-AL, pp. 54-61.

Hannover Veterinary School (1998). Untersuchungen zu Vorkommen, Ausbreitung und Bekämpfung der klassischen Schweinepest (KSP) in Deutschland 1993-1995. Abschlußbericht zum Forschungsprojekt 96HS022, Institute of Virology, Hannover Veterinary School, Bünteweg 7, D-30559 Hannover, Germany.

Hofmann M.A., Brechtbühl K. and Stäuber N. (1994). Rapid characterization of new pestivirus strains by direct sequencing of PCR-amplified cDNA from the 5' non-coding region. *Arch. Virol.* **139**, 217-219.

Hone J., Pech R.P. and Yip P. (1992). Estimation of dynamics and rate of transmission of classical swine fever (hog cholera) in wild pigs. *Epidemiology and Infection* **108**, 377-386.

Hooft van Iddekinge B.J., de Wind N., Wensvoort G., Kimman T.G., Gielkens A.L. and Moormann R.J. (1996). Comparison of the protective efficacy of recombinant pseudorabies viruses against pseudorabies and classical swine fever in pigs; influence of different promoters on gene expression and on protection. *Vaccine* **14**, 6-12.

Janeau G., Spitz F. (1984). L'espace chez le sanglier (*Sus scrofa scrofa*). L´occupation et mode d'utilisation journalier. *Gibier Faune Sauvage* **1**, 73-89

Kaden V. (1998). Zur Situation der klassischen Schweinepest beim Wildschwein in der Europäischen Gemeinschaft und zu einigen Aspekten der Seuchenverbreitung. *Berl. Münch. Tierärztl. Wschr.* **111**, 201-207.

Kern B. (1998). Experiences with CSF-Vaccination of wild boar in Brandenburg. In. "Measures to control Classical Swine Fever in European Wild Boar" Perugia, Italy, 6-7 April 1998. European Union. Commission des Communautes Européennes. Direction Generale VI Agriculture, 122-127.

Kern B., Depner K. R., Letz W., Rott M. and Liess B. (1999): Incidence of classical swine fever (CSF) in wild boar in a densely populated area indicating CSF virus persistence as a mechanism for virus perpetuation. *Vet. Med.* (in press).

Koprowski H., James T.R. and Cox H.R. (1946). Propagation of hog cholera virus in rabbits. *Proc. Soc. Exp. Biol. Med.* **63**, 178-183.

Krassnig R. and Schuller W. (1993). Schweinepest in Österreich. *Wien. Tierärztl. Mschr.* **80**, 229-233.

Laddomada A., Patta C., Oggiano A., Caccia A., Ruiu A., Cossu P. and Firinu A. (1994). Epidemiology of classical swine fever in Sardinia: A serological survey of wild boar and comparison with African swine fever. *Vet. Rec.* **19**, 183-187.

Loepelmann H.-W. and Schuster P. (1997). Ködern tabu? *Unsere Jagd* **2/97**, 12-13.

Lowings P., Ibata G., Needham J. and Paton D. (1996). Classical swine fever diversity and evolution. *J. Gen. Virol.* **77**, 1311-1321.

McKercher P.D., Hess W.R., and Hamdy F. (1978). Residual viruses in pork products. *Applied and Environmental Microbiology* **35**, 142-145.

McKercher P.D., Yedloudschnig R.J., Callis J.J., Murphy R., Panina G.F., Civardi A., Bugnetti M., Foni E., Laddomada A., Scarano C., and Scatozza F. (1987). Survival of viruses in "Prosciutto di Parma" (Parma Ham). *Can. Inst. Food Sci. Technol.* **20**, 267-272.

Maillard D. and Fournier P. (1995). Effects of shooting with hounds on size of resting range of wild boar (*Sus scrofa* L.) groups in mediterranean habitat. Ibex: *J. Mountain Ecology* **3,** 102-107.

Mauget R., Campan R., Spitz F., Dardaillon M., Janeau G. and Pepin D. (1984). Synthese des connaissance actuelles sur la biologie du sanglier, perspectives de recherche. Symposium International sur le Sanglier. Toulouse, 24-26 Avril 1984. INRA, 15-53.

Mebus C.A., House C., Ruiz Gonzalvo F., Pineda J.M., Tapiador J., Pire J.J., Bergada J., Yedloutschnig R.J., Sahu S., Becerra V., and Sanchez-Vizcaino J.M. (1993). Survival of foot-and-mouth disease, African swine fever, and hog cholera viruses in Spanish serrano and cured hams and Iberian cured hams, shoulders and loins. *Food Microbiology* **10**, 133-143.

Meyer H., Liess B., Hermanns W. and Trautwein G. (1980). Experimentelle, diaplazentare Infektion von Schweinefoeten mit dem Virus der Europäischen Schweinepest (ESP). Virologische und serologische Untersuchungen in der postnatalen Phase. Fortschritte der Veterinärmedizin. *Suppl. Zbl. Vet. Med.* **30**, 140-144.

Meyers G. and Thiel H. J. (1996). Molecular characterization of pestiviruses. *Adv. Virus Res.* **47**, 53-118.

Meyers G., Thiel H.J. and Rümenapf T. (1996). Classical swine fever virus: recovery of infectious viruses from cDNA constructs and generation of recombinant cytopathogenic defective interfering particles. *J. Virol.* **70**, 1588-1595.

Moennig V. and Plagemann G.W. (1992). The pestiviruses. *Adv. Virus Res.* **41**, 53-98.

Moormann R.J., van Gennip H.G., Miedema G.K., Hulst M.M. and van Rijn P.A. (1996). Infectious RNA transcribed from an engineered full-length cDNA template of the genome of a pestivirus. *J. Virol.* **70**, 763-770.

Moretti, M. (1985). Birth distribution, structure and dynamics of a hunted mountain population of wild boar (*Sus scrofa*), Ticino, Swizerland. Ibex: *J. Mountain Ecology* **3**, 192-196.

OIE (1996). Chapter 2.1.13. Classical swine fever (hog cholera). *In*: *OIE Manual of Standards for Diagnostic Tests and Vaccines*, 3rd ed., OIE, Paris, 145-154.

Panina G.F., Civardi A., Cordioli P., Massirio I., Scatozza F., Baldini P., and Palmia, F. (1992). Survival of hog cholera virus (HCV) in sausage meat products (Italian salami). *International Journal of Food Microbiology* **17**, 19-25.

Rümenapf T., Stark R., Meyers G. and Thiel H.J. (1991). Structural proteins of hog cholera virus expressed by vaccinia virus: further characterization and induction of protective immunity. *J. Virol.* **65**, 589-597.

Ruggli N., Tratschin J.D., Mittelholzer C. and Hofmann M.A. (1996). Nucleotide sequence of classical swine fever virus strain Alfort/187 and transcription of infectious RNA from stably cloned full-length cDNA. *J. Virol.* **70**, 3478-3487.

Rutili G. (1997). Country report on classical swine fever in Italy. In: Report on Annual Meeting of National Swine Fever Laboratories, Vienna, Austria, 16-17 June 1997, Commission of the European Communities, Document VI/7888/97, p. 13.

Rutili D., Guberti V. and Ferrari G. (1998). Classical Swine Fever in wild boar. Evaluation of control measures applied in Italy and proposal for the future. In. "Measures to control Classical Swine Fever in European Wild Boar" Perugia, Italy, 6-7 April 1998. European Union. Commission des Communautes Européennes. Direction Generale VI Agriculture, 135-137.

Savi P., Torlone V. and Titoli F. (1965). Recherches sur la survie du virus de la peste porcine classique dans certains produits de charcuterie. *Bull. OIE* **63**, 87-96.

Stubbe C. (1987). Lebensraumnutzung und Populationsumsatz des Schwarzwildes in der DDR - Ergebnisse der Wildmarkierung. Unsere Jagd **32**, 228-230.

Stubbe W. and Stubbe M., (1977). Vergleichende Beiträge zur Reproduktions- und Geburts- biologie von Wild- und Hausschwein - Sus scrofa. *Beiträge zur Jagd- und Wildforschung* **10**, 153-179.

Stubbe C., Mehlitz, S., Peukert R., Goretzki, J., Stubbe, W., and Meynhardt, H. (1989). Lebensraumnutzung und Populationsumsatz des Schwarzwildes in der DDR - Ergebnisse der Wildmarkierung. *Beiträge zur Jagd- und Wildforschung* **16**, 212-231.

Teillaud P. (1986). La vie sociale chez de sanglier. *Cahiers d'Ethologie appliquée* **6(2)**, 157-184

Teuffert J., Schlüter H. and Kramer M. (1997): Europäische Schweinepest. Übersicht zur internationalen (Europa) und nationalen Schweinepestsituation - ermittelte Einschleppungsursachen und Verschleppungsrisiken. *Dtsch. Tierärztebl.* **45**, 1078-1080.

Van Oirschot J.T. (1999). Hog cholera. *In*: *Diseases of swine, 8th ed.,* Shaw B.E.et al., (eds). Iowa State University Press, Iowa,159-172.

Van Oirschot, J.T. (1979) Experimental production of congenital persistent swine fever infections. 1.Clinical, pathological and physiological observations 2. Effect on functions of the immune system. *Vet. Microbiol*. **4**, 117-132, 133-197. Van Oirschot J.T. and Terpstra C.A. (1977). A congenital persistent swine fever infection. I. Clinical and virological observations. II. Immune response to swine fever virus and unrelated antigens. *Vet. Microbiol*. **2**, 121-142.

Van Rijn P.A., Bossers A., Wensvoort G. and Moormann R.J. (1996). Classical swine fever virus (CSFV) envelope glycoprotein E2 containing one structural

antigenic unit protects pigs from lethal CSFV challenge. *J. Gen. Virol.* **77**, 2737-2745.

Van Zijl M., Wensvoort G., de Kluyver E., Hulst M., van der Gulden H., Gielkens A., Berns A. and Moormann R. (1991). Live attenuated pseudorabies virus expressing envelope glycoprotein E1 of hog cholera virus protects swine against both pseudorabies and hog cholera. *J. Virol.* **65**, 2761-2765.

Wachendörfer G., Reinhold G. E., Dingeldein W., Berger J., Lorenz J. and Frost J. W. (1978): Analyse der Schweinepest-Epizootie in Hessen in den Jahren 1971-1974. *Dtsch. Tierärztl. Wochenschr.* **85**, 113-120.

Wengler G. (1991). Family Flaviviridae. *In*: Classification and nomenclature of viruses. *Fifth report of the International Committee on Taxonomy of Viruses.* Francki R.I.B., Fauquet C.M., Knudson D.L. and Brown F. (eds). Springer Verlag, Berlin, 223-233.

Zilio A. and Pedrotti L. (1998). Effect of hunting activity on population structure of wild boar (*Sus scrofa*). In. "Measures to control Classical Swine Fever in European Wild Boar" Perugia, Italy, 6-7 April 1998. European Union. Commission des Communautés Européennes. Direction Générale VI Agriculture, 17-31.

ADDITIONAL READING

Abaigar T. (1992) - Paramètres de la reproduction chez le sanglier (Sus scrofa) dans le sud-est de la péninsule ibérique. Mammalia. 56 : 245-250.

Baubet E. (1998) - Biologie du sanglier en montagne : biodémographie, occupation de l'espace et régime alimentaire. Thèse, Université Claude Bernard - Lyon I.

Brandt S., Vassant J., and Jullien J-M (1998) - Domaine vital diurne des sangliers en forêt de Châteauvillain - Arc en Barrois. Bulletin mensuel de l'ONC, n° 234, juin.

Boitani L., Trapanese P., Mattei L., and Nonis D. (1995) - Demography of a wild boar (Sus scrofa, L.) population in Tuscany, Italy. Gibier Faune Sauvage. 12 : 109-132.

Bulletin Mensuel De L'office National De La Chasse (1993) spécial « actes du colloque : gestion du sanglier », Bergerac, n° 191.

Loepelmann H., and Schuster P. (1997). Ködern Tabu. Unsere Jagd. 2, 12-13.

Vassant J., Brandt S., and Jullien J.M. (1992) - Des sangliers encore plus sédentaires qu'il était imaginable de le croire. Bulletin mensuel ONC 165:31-39, Février.

Vassant J. (1997) - Agrainage et gestion des populations de sangliers. Fiche technique n° 92, supplément au Bulletin mensuel de l'Office national de la chasse n° 227, novembre.

Vassant J., Brandt S., Maillard D., and Baubet E. (1998) - Wild boar ethology and behaviour. Meeting : measures to control classical swine fever in European wild boar. Perugia, Italy, 6-7 april 1998 : 6-16.

10   ACKNOWLEDGEMENTS

11 ANNEX I

A survey of the situation of CSF in wild boar

Wild boar populations in European countries and monitoring for CSF

Country	Wild boar population		Investigation and choice of samples			
	Estimated population	Distribution	Random	Sick wild boar	Proximity to outbreaks**	CSF occurrence
Andorra	n.a*	overall	no	no	No	n.i.***
Armenia	n.a.	regional	n.a.	n.a.	n.a.	n.a.
Austria	n.a.	n.a.	n.a.	n.a.	n.a.	yes
Belarus	26.643	overall	no	yes	No	n.i.
Croatia	10.200	overall	yes	yes	Yes	no
Cyprus	0					
Czech Rep.	38.000	overall	yes	yes	Yes	yes
Denmark#	n.a.	regional	no	no	No	n.i.
Estonia	10.000	overall	n.a.	n.a.	n.a.	n.a.
Finland	300	regional	yes	yes	No	n.i.
France	450.000	regional	yes	yes	No	yes

Wild boar populations in European countries and monitoring for CSF (continued)

Germany	600.000	overall	yes	yes	Yes	yes
Greece	500	regional	no	no	No	n.i.
Iceland	0					
Ireland	0					
Italy	n.a.	regional	yes	yes	Yes	yes
Latvia	17.274	overall	yes	no	No	yes
Lithuania	19.400	overall	yes	yes	Yes	no
Luxembourg	15.000	overall	yes	no	No	no
Malta	0					
Moldavia	1.137	overall	yes	no	Yes	n.i.
Netherlands	3.000	regional	yes	yes	Yes	no
Norway	0					
Portugal	60.000	n.a.	yes	no	No	no
Romania	16.800	overall	yes	yes	Yes	no
Slovakia	19.536	overall	yes	yes	Yes	yes

Wild boar populations in European countries and monitoring for CSF (continued)

Slovenia	5.000	overall	yes	yes	Yes	n.i.
Spain	n.a.	overall	no	no	No	n.i.
Sweden	10.000	regional	no	no	No	n.i.
Switzerland	8.000	regional	yes	yes	Yes	yes
United Kingdom	100	regional	no	no	No	n.i.
Ukraine	43.000	overall	no	yes	Yes	yes
Uzbekistan	n.a.	overall	no	no	No	n.i.

* data not available   ** proximity to CSF outbreaks in domestic pigs

*** not investigated

\# Since 1933 no cases of CSF have been reported in Denmark. This country has no wild free-living population of wild boar, however, a small number of managed wild boar exists under controlled conditions. Such animals are included in the continuous Danish surveillance for classical swine fever.

Source: Moennig, V.:

Strategies for the control of classical swine fever, including the application of modern vaccines.

In: 18th Conference of the OIE Regional Commission for Europe. Prague, Czech Republic, 22-25 September 1998.

12 ANNEX II

List of addresses of National Swine Fever Laboratories of EU Member States and Poland

AUSTRIA

Bundesanstalt für Virusseuchenbekämpfung bei Haustieren

Robert Koch Gasse 17   A-2340 Mödling

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BELGIUM

National Veterinary and Agrochemical Research Institute

Groeselenberg 99   B-1180 Brussels

Fax: +32 2 375 0979   Tel.: +32 2 375 4455

DENMARK

Danish Veterinary Institute for Virus Research, Lindholm

DK-4771 Kalvehave

Fax: + 45 55 860300   Tel.: + 45 55 860200

FINLAND

National Veterinary and Food Research Institute (EELA)

P.B. 368   FIN-00231 Helsinki

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AFSSA-Alfort

rue Pierre Curie, 22 - B.P. 67   F-94703 Maisons-Alfort

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E-mail: vaal30@calvacom.fr

AFSSA-Ploufragan

B.P. 53 - Zoopole des Cotes d'Armor   F-22440 Ploufragan

Fax: +33 2 9678 6861   Tel: +33 2 9676 0130

GERMANY

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D-17498 Insel Riems

Fax: +49 38351 7219   Tel.: +4938 351 70

GREECE

Veterinary Institute of Infectious and Parasitic Disease

25 Neapoleos Str.   GR-15310 Ag. Paraskevi

Fax: +30 1 6399 477   Tel.: +30 1 6010 903

Institute of infectious and parasitic diseases

Virus laboratory 80, 26th October St.   GR-54627 Thessaloniki

Fax: +30 31 252 023   Tel: +30 31 252 023

IRELAND

Department of Agriculture Veterinary Research Laboratory

Abbotstown Castleknock   Dublin 15, IRELAND

Fax: +353 1 8220363   Tel.: +353 1 6072782

ITALY

Instituto Zooprofilattico Sperimentale

Via G. Salvemini 1   I-06 100 Perugia

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Institute for Animal Science and Health (ID-DLO)

Houtribweg 39   P.O.Box 65   NL-8200 AB Lelystad

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PORTUGAL

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SWEDEN

National Veterinary Institute

Box 7073   S-750 07 Uppsala

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UNITED KINGDOM

Central Veterinary Laboratory New Haw, Weybridge

Surrey KT15 3NB, UK

Fax: +44 1932 357 239   Tel.: +44 1932 357 7474

Community Reference Laboratory for CSF

School of Veterinary Medicine   Institut of Virology

Bünteweg 17   D-30559 Hannover

Fax: +49 511 953 8898/ 8856   Tel: + 49 511 953 8841/ 8850

E-mail: moennig@viro.tiho-hannover.de

E-mail: gfloegel@viro.tiho-hannover.de

POLAND

OIE Reference Laboratory for CSF

National Veterinary Research Institute

Partyzantow 57   24 100 Pulawy, POLAND

Fax: 0048-81-8862 595   Tel.: 0048-81-8863 051