Rev. sci. tech. Off. int. Epiz., 2002, **21** (2), 287-303

Classical swine fever (hog cholera) in wild boar in Europe

M. Artois [1], K.R. Depner [2], V. Guberti [3], J. Hars [4], S. Rossi [1, 5] & D. Rutili [6]

(1) Département de santé publique vétérinaire, Unité de pathologie infectieuse, École Nationale Vétérinaire de Lyon, B.P. 83, 69280 Marcy l'Étoile, France
(2) Federal Research Centre for Virus Disease of Animals, Boddenblick 5a, D-17498 Insel Riems, Germany
(3) Istituto Nazionale per la Fauna Selvatica, Via Ca' Fornacetta, 9, 40064 Ozzano, Italy
(4) Unité suivi sanitaire de la faune, Office national de la chasse et de la faune sauvage, 8, Impasse Champ Fila, 38320 Eybens, France
(5) Direction générale de l'alimentation, ministère de l'Agriculture et de la Pêche, 251 rue de Vaugirard, 75732 Paris Cedex 15, France
(6) Istituto zooprofilattico sperimentale dell'Umbria e delle Marche, Via Salvemini no. 1, 06126 Perugia, Italy

Summary

Classical swine fever (CSF) is of increasing concern in Europe where wild boar appear to play an important epidemiological role. In most parts of the continent, demographic trends are on the increase, due to improvement in game management. As a result of higher densities, populations become more susceptible to various infectious diseases, among which CSF is cause for particular concern. Wild boar do not appear to be a classic reservoir in most cases, but nevertheless may perpetuate foci of infection over the long term, constituting a real threat for the pig farming industry. Since the infection does not appear to spread easily in natural populations of free-ranging wild boars, control of the disease may be feasible. However, most of the appropriate measures, such as banning hunting, are not considered acceptable. Consequently, the expertise of wildlife disease specialists is required to help solve the problem when it occurs.

Keywords

Classical swine fever – Control – Epidemiology – Europe – Sus scrofa – Wild boar – Wildlife.

Introduction

Classical swine fever (CSF) is caused by an infection with CSF (hog cholera) virus. The disease is listed as one of the highly contagious List A diseases of *suidae* by the Office International des Epizooties (OIE). Under natural conditions, the infection occurs in domestic pigs and wild boar (*Sus scrofa*) causing major economic losses especially in countries with an industrialised pig production. The total sum of direct economic losses in The Netherlands during the CSF epidemic in domestic pigs in 1997 amounted to US$2.3 billion and more than 11 million pigs had to be destroyed (57). Large-scale culling of pigs due to CSF was also conducted in other countries of Europe (Austria, Belgium, Czech Republic, Germany, Italy and Spain) between 1991 and 2001. As a result of the ethical dimensions of the disease and the economic losses incurred, CSF is rated as one of the most important diseases of domestic animals in Europe.

In many instances, the wild boar is regarded as a reservoir for CSF virus and as possible source of infection for domestic pigs (41, 43, 76, 81, 87). The aim of this paper is to review the current knowledge of CSF in wild boar, assess the role of this species as a reservoir, examine routes of transmission to domestic pigs and discuss different strategies and methods of control.

© OIE - 2002

The disease and course of infection

The classical swine fever (hog cholera) virus

Classical swine fever is caused by a member of the *Flaviviridae* family, genus *Pestivirus* (88). This virus is antigenically related to bovine viral diarrhoea (BVD) virus and to Border disease (BD) virus; both belong to the same genus and are infectious for pigs as well as cattle and sheep. The CSF virus is a small enveloped single positive-stranded RNA virus with a diameter of 40-50 nm. The viral ribonucleic acid (RNA) codes for four structural and seven non-structural proteins (23, 58). There are no defined serotypes. Compared to BVD, CSFV strains form a relatively uniform antigenic cluster but some variation exists among isolates. Serological cross-reactions with BVD and BD viruses do occur and might hamper serological diagnosis.

The virus is relatively stable in fomites and fresh meat products. Survival can be prolonged for years. The CSF virus can survive for 90 days in frozen meat (75) and 188 days in ham (52). However, the virus is readily inactivated by detergents, lipid solvents, proteases and some disinfectants. The virus appears to be inactivated in pens and dung in a few days.

Pathogenesis and clinical course of the disease

Studies of the clinical course of CSF in wild boar have not been as extensive as for domestic pigs. However, from the few experimental studies conducted, it can be concluded that the course of the disease is similar in both species (11, 19, 44, 45).

The pathogenesis of CSF may follow two different pathways, depending on the time of infection. The animal may either be infected prenatally as a foetus when the immune system is not fully developed or the infection may take place at a post-natal stage when the immune system has already developed. The two courses differ with regard to their pathogenicity and have different consequences for the clinical disease as well as for the perpetuation of the virus within a population of susceptible hosts.

Prenatal infection

The CSF virus has the ability to cross the placental barrier and to infect the foetus *in utero*. This happens if a pregnant sow becomes transiently infected with CSF virus. Early infections result in abortions and stillbirths, whereas later infections yield persistently viraemic young. The viraemic piglets shed virus permanently until they die. They can play a key role in the spread of CSF virus within the pig population. Most viraemic piglets survive (unrecognised) for several weeks, but all eventually die. For domestic piglets, the longest period of persistent viraemia reported to date is 11 months, while for wild boar piglets viraemia lasted only 39 days (19, 85).

Only these persistently viraemic piglets develop the so-called 'late onset' form of CSF. After the prodromal period without clinical signs, persistently infected piglets may develop mild clinical signs and lesions. Growth retardation is the most common finding. Although the prenatal intra-uterine infection which leads to persistently infected offspring is rather a rare event, it is regarded as the main maintenance mechanism of CSF virus for surviving within a population (46, 80). In wild boar, the prenatal form of CSF has only been demonstrated experimentally (19) and no data are yet available from the wild.

Post-natal infection

The post-natal infection is the best known or 'classical' form of CSF. According to the clinical course, post-natal infection has been categorised as acute (including the per-acute and sub-acute courses) and chronic (16, 46, 84). The acute courses last less than four weeks and the infected animal may either recover completely (transient infection) or die (lethal infection). The mortality rate may be as high as 90%, depending on a variety of factors. The chronic CSF virus infection has been defined as disease with a duration of 30 days or more prior to death (55). However, under field conditions different disease patterns occur simultaneously.

The outcome of post-natal CSF has often been related solely to the virulence of the virus involved: strains of high virulence would induce acute and lethal disease, whereas moderately virulent strains would give rise to sub-acute or chronic infections. Mild disease or sub-clinical infection can be generated by low virulent CSFV strains (84). However, virulence on its own is a rather confusing and misleading parameter. The age of the pig at the time of exposure, its immune reactivity, the virus infecting dose as well as the breed of pig were shown amongst other factors to be of utmost importance (16, 20). In adult animals, virulent CSF virus strains often induce only transient infections with few mild clinical and pathological signs and low mortality. In sero-negative pregnant sows reproductive failure frequently occurs.

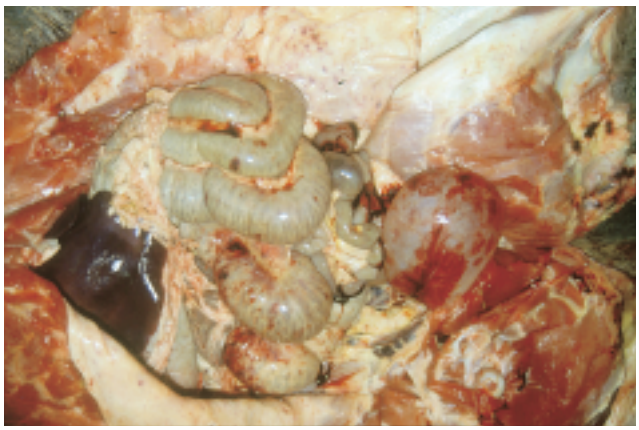
The disease in young pigs and wild boars is generally more evident, leading to high mortality rates (39). In wild boar, only the acute course of disease has been described to date. Experimental and field data are lacking for the chronic course of disease. However, this does not mean that chronic CSF in wild boar does not occur.

In acute cases, clinical signs such as high body temperature (>41°C), inappetence and apathy can be seen after an incubation period of about three to seven days. The progression of the disease is marked by conjunctivitis, nasal discharge, intermittent diarrhoea, swollen lymph nodes and muscle tremor. The terminal stage in domestic pigs is characterised by 'typical' skin cyanosis, mainly on ears, nose, tail and abdomen and skin haemorrhages of different grades predominantly over bone protuberances. In wild boar, the skin lesions are less evident and are masked by bristles. In addition, wild boar tend

© OIE - 2002

to exhibit marked behavioural changes, such as loss of natural shyness and roaming during daylight (31). Sick animals frequently show weakness of the hind legs (waving or staggering gait) which is often followed by a posterior paresis. Leucopaenia and thrombocytopaenia are common findings. The predominant post-mortem findings consist of haemorrhagic diathesis and swollen, haemorrhagic lymph nodes (Fig. 1). However, the severity of pathological lesions can vary widely.

a) Abdomen



b) Epiglottis

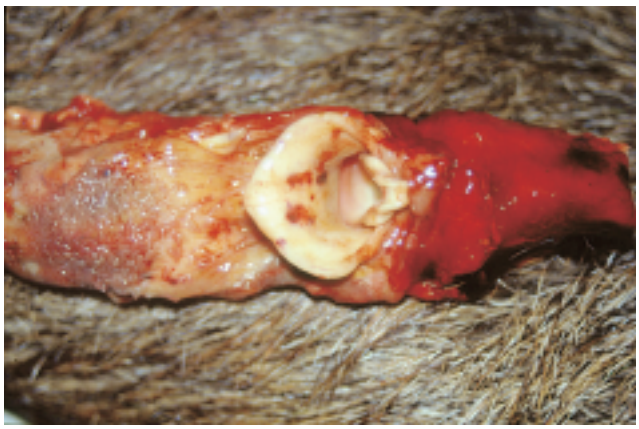


Fig. 1
Bleedings in the abdomen and epiglottis
Photos: courtesy of K. Depner

Pigs which 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. Wasting and diarrhoea are the most obvious signs. Pigs with chronic CSF may survive for more than 100 days (55). Secondary bacterial and parasitic infections are frequently involved. Thus, the clinical picture may often be uncharacteristic and misleading (according to game wardens, an important outbreak of mange was noticed in the Vosges area just prior to the 1992/1993 CSF outbreak in France). Taken together, it must be emphasised that the clinical signs of CSF in wild boar can be extremely variable depending on a range of different factors and therefore may hamper rapid recognition in the field.

Pathogenesis

Under natural conditions, the oro-nasal route is the common route of infection and the tonsils are the primary site of virus replication (virus has been demonstrated in the tonsils as early as seven hours after exposure) (71). It is interesting to note that after intra-muscular and subcutaneous injection, CSF virus was found most consistently and in a constantly high concentration in the tonsils (47, 56). From the tonsils, CSF virus is transferred via lymphatic vessels to the lymph nodes, draining the tonsil region. After replication in the regional lymph nodes, the virus reaches all other organs of the body via the blood. High titres of virus have been observed in spleen, bone marrow, visceral lymph nodes and lymphoid structures lining the small intestine. The virus probably does not invade parenchymateous organs until late in the viraemic phase. The spread of virus is usually completed within five to six days.

Immunity

The immune response of wild boar against CSF virus is analogous to the immune response of domestic pigs. As is the case with all pestiviruses, acute CSF infection affects the immune system causing immuno-suppression (83) which is mainly characterised by leukopaenia seen before the onset of fever. The virus causes severe depletion of both B cell and thymus-dependent areas. Little is known about cell-mediated immunity against CSF. In transient, acute CSF, neutralising antibodies do not appear in the blood until the leukopaenia is overcome (40). Pigs that have recovered are protected against CSF for at least six months or even for their lifetime (54). Neutralising antibodies are detectable two weeks after infection at the earliest. Pigs infected *in utero* are immuno-tolerant against the homologous CSF virus and do not produce specific antibodies (46).

Maternal antibodies have a half-life of about eleven days (21). Passive immunity generally protects piglets against mortality during the first weeks of life, but not necessarily against virus replication and shedding (21). In persistently viraemic piglets, colostrum-derived antibodies can only be detected for a short time after birth (less than one week) compared to non-viraemic litter mates (19).

Diagnosis, detection and molecular epidemiology

Clinical and pathological signs, and epidemiological determinants can be used to detect CSF in the domestic pig as well as in wild boar. Outbreaks are often suspected because an unusual number of boar carcasses are found. Laboratory diagnosis, and notably virological tests, are essential to rule out the presence of CSF. The tissues commonly used are the tonsil, head/neck lymph-nodes, spleen, ileum and kidney. Blood or

© OIE - 2002

tissue fluids are taken where serological tests are performed. The technical annexes of EU legislation (14) as well as the OIE *Manual of Standards for Diagnostic Tests and Vaccines* (63) provide useful details on the laboratory procedures for diagnosis of CSF.

Routine laboratory methods

Virus detection, either using the immunofluorescence test (IFT) on cryostat sections, the enzyme-linked immunosorbent assay (ELISA), virus isolation on cell culture or RNA detection methods, are the procedures of choice for confirmatory diagnosis of CSF. The antigen detection tests have a somewhat lower sensitivity (IFT) and lower specificity (ELISA) than virus isolation but have the great advantage of relative simplicity and rapid turnaround times. The virus isolation is the gold standard and definitive test but is labour intensive and requires at least three days before the results are available. Furthermore, in many circumstances, virus isolation may be limited because the autolysed sample material frequently obtained from wild boar can be cytotoxic to the cell culture.

The serological diagnosis of CSF is mainly used to distinguish CSF virus and BVD virus antibodies or for surveillance to determine the extent of sub-clinical spread of CSF virus infection in a population. The neutralisation test is widely used as a reference method. The ELISA is now in use world-wide for large-scale control campaigns. The ELISA for detection of CSF antibodies needs to be sensitive and specific, and as free as possible from interference by cross-reacting antibodies to BVD virus or BD virus (13). Several ELISA methods have been developed and are available commercially.

For the declaration of a primary outbreak of CSF in wild boar, positive virus isolation is essential. When the positive results arise from the IFT or polymerase chain reaction (PCR), the epidemiological circumstances should be taken into to account, e.g. animals found dead or the occurrence of CSF in a neighbouring wild boar population. For the evaluation of the trend of an epidemic and within the framework of a surveillance campaign, a positive IFT or antigen ELISA result can be considered sufficient to yield a positive diagnosis for CSF. Even the serology can be sufficient to monitor an epidemic.

Laboratory investigation for classical swine fever detection and characterisation in natural populations of wild boar

In recent years the reverse transcriptase-PCR (RT-PCR) has been used for primary diagnosis. This is both sensitive and rapid and particularly suitable for samples of material from wild animals (72); PCR can be performed within one day. This procedure may be used for the generation of genetic sequence data, which gives much more precise virus characteristics to be used for epidemiological tracing of virus (49).

In recent years, molecular typing has proved to be a very effective method to determine relationships between different outbreaks of CSF. In combination with epidemiological surveys, molecular typing has helped to trace the spread of the disease, e.g. to different geographical locations.

The detection and analysis of differences between virus isolates enables the sub-grouping of these in a phylogenetic tree (50). Over the past few years, the findings of investigations based on genetic comparison, which were performed in Italy and Germany, showed the persistence of certain viruses as well as the introduction of new viruses from different regions or countries (7, 26). Relationships between wild boar and domestic swine isolates which were obtained from a study of CSF in Tuscany (49), also provided evidence that the virus could persist in wild boar for several years whilst periodically causing outbreaks in domestic pigs.

Transmission and epidemiology

Historical situation

The history of CSF in wild boar has been associated with the persistence of the disease in domestic pigs. The disease is distributed almost world-wide (22). Historically, CSF was first described in Austria at the end of the 19th Century. Further outbreaks were documented in Germany in 1916 (17). The virus has been eradicated from North America and Australia. The first regulation attempting to control CSF in the European Union (EU) dates back to 1980 (22). Since then, the disease has occurred in Sardinia from 1983 onwards, when it became endemic (25, 43). In the past two decades, CSF in wild boar has emerged as a serious problem in Europe (42) (Table I). During this period, CSF was diagnosed and confirmed in wild boars (mainly in Austria, France, Germany and Italy) (42, 43, 69). It also appears that CSF is more or less endemic in several countries in Eastern Europe; the infection has recently (2001) been reported in Luxembourg, Slovakia and the Ukraine (64, 65, 66) (Fig. 2) (Table I).

The host: wild boar, ecology of a successful species

Ecological background

Free-ranging animal populations are regulated by several environmental constraints. The 'carrying capacity' (K) is 'the largest number of individuals of a particular species that can be maintained sustainably in a given part of their environment' (90). As a rule, every species tends to saturate the carrying capacity of the environment very rapidly. This is achieved in two ways, namely:

a) by increasing the survival of adult animals while reducing recruitment

b) by increasing fertility and thus recruitment when short life-span (i.e. average life expectancy) is imposed.

© OIE - 2002

Table I
Areas recently affected by classical swine fever among wild boars in Europe (14, 42)

Countries and regions partly infected	Year of discovery (month)	Wild boar (estimated population)	Size of the affected area (km²)	Situation in 2001
Austria				
	1990			Extinct (1992)
Lower Austria	1996			
Lower Austria	2000 (November)			(10% seropositive)
Belgium				
Liege**	2000 (May)			Extinct (November 2000)
France				
Northern Vosges*	1992 (January)	6,000	3,000	Towards extinction No virus isolation since 1997 (1.8% seropositive)
Germany				
Lower Saxony	1992	6,000	3,029	Still infected
Rhineland Palatinate*	1992			No virus isolation 1996/1997
Rhineland Palatinate**	1998	3,000	4,710	Still infected
Mecklenburg-West. Pomerania	1993	6,450	5,720	No virus isolation since 2001
Brandenburg	1995 (March)	7,000	2,900	
Baden-Wurtenburg	1998-1999	700	970	
Saxony-Anhalt	1999 (June)	2,100	625	No virus isolation since 2000
Italy				
Sardinia	1983			Enzootic
Tuscany (South)	1985/1986 (October)	8,000	3,800	Extinct 1990
Tuscany (North)	1992 (April)	305	1,000	Extinct 1992 (August)
Emilia Romagna	1995 (September)	300	75	Extinct 1996 (January)
Lombardia***	1997 (May)	1,200	370	No virus isolation since 2001
Luxembourg**	2000			
Slovakia	2001			
Switzerland				
Ticino***	1998			Extinct 2000
Ukraine				
Kiev and Tcherkassy	2001 (July)			

Asterisks: countries and regions with common borders
Nota: Several outbreaks were only detected by record of seroconverted animals and no virus isolation. In most countries, the main criterion for extinction to be considered is absence of virus isolation for more than six months
Data regarding population size shall be regarded with extreme caution and only considered as rank of order allowing rough comparison between outbreaks

The carrying capacity is determined by complex relationships between a-biotic (climate, soil, etc.) and biotic factors (food, behaviour, etc.). The latter include inter- and intra-specific competition, predation and diseases (*sensu lato*). According to the ecological theory, maximum productivity is achieved when the population is 50% of the carrying capacity.

The viability of a population might be scaled on its range (in real terms), taking into account aggregations or uniformity of its distribution, and on temporal dynamics (turn-over rate). Species that are widely and uniformly distributed and which have a high turn-over rate, are extremely viable, where viability accounts the ability of a population to survive despite environmental constraints. If large enough, the population will have a great probability to grow exponentially. Conversely, it might become extinct. To maintain a minimum viable population means to ensure the future destiny of a species. Attempts to depopulate or strongly reduce a viable population have proved to be almost impossible and sustained efforts are necessary to maintain a viable population at a desired level of density and abundance.

The natural history of the wild boar in Europe

Since the early 1950s, wild boar populations have increased both in number and distribution range throughout Europe; their population dynamics is enhanced in industrialised countries. France, Germany and Italy host several hundreds of thousands of wild boar. In France, for instance, bag records from the hunting game agency indicate that the current population is close to 800,000 individuals (Fig. 3) (33). Similar trends have been observed in the other countries of Europe, with the possible exception of Russia (74). After the Second

© OIE - 2002

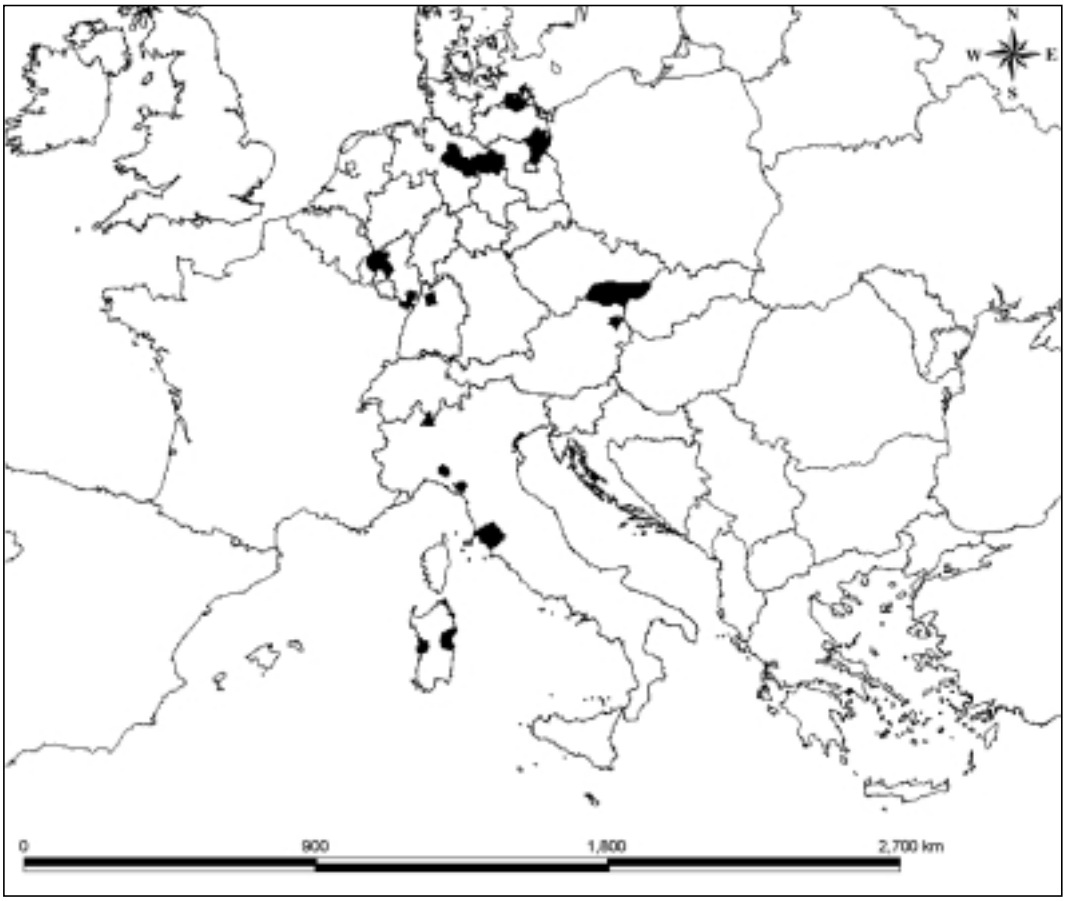


Fig. 2
Map of Europe displaying localities of recently recorded classical swine fever outbreaks among wild boars, 1990-2001
The designations employed and the presentation of material on this map do not imply the expression of any opinion whatsoever on the part of the OIE concerning the legal status of any country, territory, city or area or its authorities, or concerning the delimitation of its frontiers or boundaries

World War, many viable populations developed from the surviving scattered relic groups. The wild boar is now the most abundant larger mammal species in Europe.

Three points pertaining to ecology can explain the great abundance and broad distribution of wild boar in Europe.

Feeding ecology

Wild boar does not rely on a peculiar food. The species is a non-selective feeder; vegetables account for about 90% of the food consumption and only a small part of the diet is composed of animal proteins, mainly insects or molluscs (30). The fragmented landscapes composed of a mosaic of woodlands, arable and pastoral lands have increased the food availability and negated seasonal food shortages. Only climatic extremes still interfere with the ability of wild boar to obtain food. Deep snow (>70 cm) or dryness of the soil limit the access to food, especially for the so-called under-soil portion of the diet, which is a principal source of animal proteins (67). Management for hunting purposes, frequently provides artificial foodstuffs, thus reducing the negative effect of seasonal food shortages.

Even in very bad years, or in the absence of artificial feeding, wild boar can maintain densities of two (67) to up to six (77) adult animal per km^2. These densities are high enough to restore the population during the following reproductive season.

Absence of predators and competitors

Larger predators such as the wolf (*Canis lupus*), lynx (*Lynx* sp.) and brown bear (*Ursus arctos*) disappeared from most of Europe at the beginning of the last century. The residual populations of these species are scattered in distribution and number (9, 10). Despite this, the predation impact on the demography of wild boar is still under debate (29). As a result of the low numbers and sparse distribution of predators, one can assume that the true wild boar numbers are not yet limited by predators. The space use and habitat selection of the wild boar limits competition with other herbivorous mammals. The ecological niche occupied by this species overlaps poorly with other wild species in Europe, thereby avoiding competition to a large extent.

© OIE - 2002

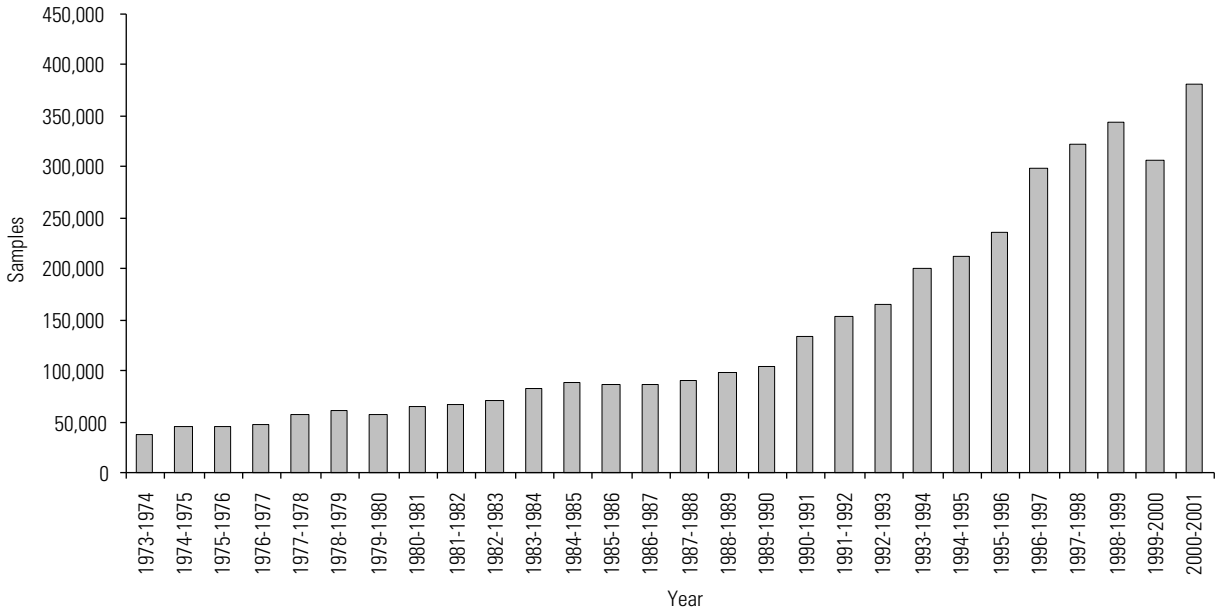


Fig. 3
Wild boar hunting bag records in France, 1973-2000
Source: National game agency/cervid/wild boar network (Office national de la chasse et de la faune sauvage)

Intra-specific competition and reproductive strategy

Surprisingly, the major competitor of the wild boar is itself. The species might suffer crashes based on density-dependent processes. If wild boar can use resources without limitation, habitat depletion is often observed. The sensitivity of the wild boar in responding to these environmental stresses has not been observed in other large mammals.

The reproductive strategy represents the principal mechanism by which the species can easily restore population. Sow heat is not seasonal but it is dependent on body condition (Fig. 4). An adult female can delay heat and mating for several months until conditions become favourable. The onset of the first pregnancy and fertility are weight-dependent, and only partially age-dependent. A sow has her first pregnancy at approximately 30 kg; this body mass can be reached at between 8 or 24 months of age, depending on food availability. The number of new-born is also positively correlated to the weight of sows. The reproductive season of the species lasts for 6 to 8 months, although annual peaks are observed (Fig. 5).

A strong compensatory mechanism allows wild boar to maintain the highest possible density in respect to the resources available in the environment. Once again, the number and density of this species is dependent on the environment and because of the ability of the wild boar to exploit resources, numbers and densities are often (if not always) high.

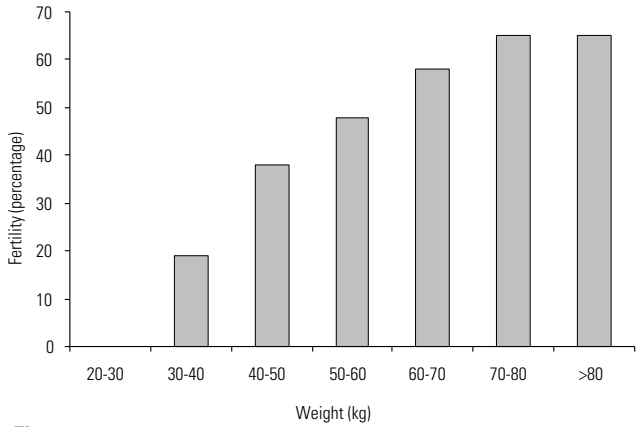


Fig. 4
Frequency of breeding sows according to weight
Source: Pedrotti *et al.* (70)

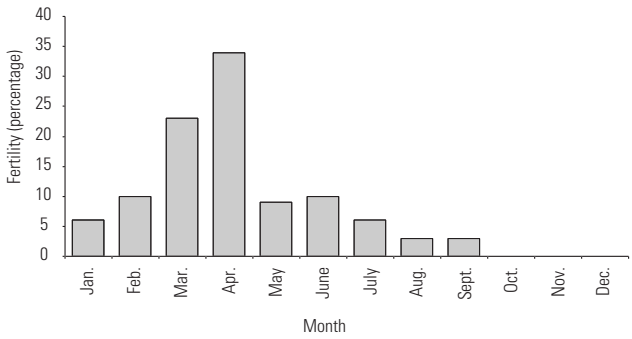


Fig. 5
Frequency of breeding sows according to the seasons of the year
Source: Pedrotti *et al.* (70)

© OIE - 2002

Origin/source of infection in wild boar populations

According to various serological surveys, most of the western populations of wild boars have remained free of CSF, and in continental Europe the disease is prevalent only in a limited number of foci (Table I) (Fig. 2) (1, 8, 17, 18, 51, 68, 78). The virus is thought to circulate naturally in some countries of the world, the extent of which is not appropriately documented.

Where a wild boar population is free of the infection, the virus can only be introduced from external sources, e.g. by artificially contaminated food (imported scraps of meat, kitchen waste, swill, diseased carcass of a buried pig) (24) or by close contact with infected domestic pigs.

Description of outbreaks

The epidemiology of CSF outbreaks in wild boar populations has been reviewed by Aubert *et al.* (6), Laddomada (42) and Froelich *et al.* (27). Many descriptions have been made of CSF introductions into susceptible populations of wild boars. These introductions of infection, whether natural or otherwise, generally proceed to a spontaneous extinction of the virus within few years or more. However, during an epizootic, mortality involving a variable proportion of the wild boar population is recorded (Santa-Cruz Island, California [60]; Pakistan [37], North Toscana or Emilia Romagna, Italy [73], Ticino, Switzerland [36], France [34]). Nevertheless, many of the recent outbreaks observed in France, Germany and Italy were long-lasting, characterised by a mortality peak after the initial virus intrusion, followed by a slow progressive decrease in the infection rate over a long period of time. This period of progressive decrease in infection rate was characterised by significant sero-conversion in an important proportion of adults. After a couple of years, the number of viral isolations decreases and may cease, but if a proportion of young (older than three months) has serum neutralising antibodies, an ongoing circulation of the virus should be suspected. Re-occurrences can be observed when surveillance is not adequate to detect the low-grade infection.

The disease is still endemic in Sardinia due to the spread of CSF infection among wild boar and free-roaming pig populations.

Analytical epidemiology

An important question when addressing the subject of wild boars and the epidemiology of CSF is to examine the way in which infection can be maintained in foci at a relatively low rate.

No seasonal or temporal trends have been observed in outbreaks of CSF in wild boar. Most, if not all, the foci of outbreaks of CSF in wild boar are limited in size (ranging from few square km to some ten thousand hectares) (Table I). Even in Sardinia, after many years, the disease is now localised in two well-defined areas (called 'risk areas') characterised by the presence of free-ranging pigs. On the rest of the island, wild boar positive for CSF have not yet been detected.

Foci of CSF in wild boar have never been reported to expand into an epidemic wave of infection over large areas, as was the case with rabies in foxes in Europe, for example. It has been claimed that this failure to spread is the result of natural barriers which limit boar movements; such borders are actually difficult to delineate with accuracy. Boars are generally restricted to wooded habitats which are by definition bordered by open areas; the latter are less favourable to movements of the animals.

The size of an affected population appears to be linked to the duration of an outbreak. When CSF died out within months, the affected foci were amongst the smallest recorded (Table I) (Ticino [36]; North Tuscany [73]). In addition to population size, host density should play a role in the number of cases recorded, and the proportion of immune individuals, but it is questionable to link host density to the duration of an outbreak. Nevertheless, a dense population will have an important turn-over which allows for a renewal of susceptible animals, thereby increasing the chances for the virus to persist.

As mentioned above, the role of strain virulence is puzzling (20). Early reports of the CSF outbreak in Europe mentioned heavy losses, according to the number of boar carcasses collected by hunters and by a decrease in successive hunting records (17). Nowadays, mass mortality has rarely been reported in recent CSF outbreaks in wild boar. Current observations made in Europe have involved a limited number of circulating strains (35, 50, 89). It could be hypothesised that most of these more recent strains are of a relatively moderate virulence, possibly leading to mild and transient forms of infection, at least in adults. The viral persistence can be explained by several factors, namely: persistent infection of gruntlings (piglets) acquired *in utero* (19) and transient infection of gruntlings partially protected by maternal antibodies (21). The young born of the year are more frequently found infected during an outbreak (39). It is obvious that infection mainly affects the younger population. As explained above, the wild boar breeding season can last for months when enough food is available and the climate is not too harsh. When the virus is introduced among susceptible boars, the virus spreads relatively rapidly, firstly among all age groups since they all are susceptible and when the population is large enough, new-born animals can sustain the infection for a long time.

Modelling

Mathematical models for CSF have recently been developed so as to highlight key points that may modify the course of the infection. Models provide the opportunity to explore the influence of many epidemiological and demographic parameters which alter the evolution of the infection (estimate transmission rate and test control strategies) (32, 37). The disease component of any model can be linked to the host population dynamics in such a way that the outcome of control measures can be explored, such as mass depopulation, specific age class culling or absence of intervention. Spatially structured models offer a better understanding of the influence of factors

© OIE - 2002

in enlarging or reducing the infected areas, such as population density, habitat, artificial or natural barriers (Figs 6 and 7) (12). Models currently available on CSF in wild boar are mainly based on density-dependent processes even if the stochastic effect has been taken into account, resulting in the following:

– the ability to spread can be summarised by a basic reproductive rate (R_o), representing the number of newly infected individuals produced in the lifetime of an infected host, when introduced to a susceptible population of a specified density (R_o >1 means that each infected animal transmits the infection to more than one susceptible animal)

– the evolution in time and the spread in space (not the emergence) are dependent on the number of susceptible animals rather than the number of infected animals; a specific threshold density (N_T) of susceptible animals leads to the infection fade-out; through density-dependent processes, R_o becomes less than 1, at this stage population density does not allow an infected animal to pass infection to a susceptible animal

– reaching this threshold density is the goal of any control; both vaccination and depopulation (even if using different mechanisms) should reduce the number of susceptible animals until the threshold density is attained.

For example, using both a deterministic and a stochastic technique developed from the occurrence of the infection in a wild boar population in Pakistan, Hone *et al.* estimated the R_o and the N_T of a CSF epidemic (37). This deterministic model assessed an R_o equal to 1.7 and a threshold density of about 270 susceptible animals over the area, i.e. 6.1 wild boars/km^2, to allow the infection fade-out. The stochastic techniques gave similar results with an R_o ranging from 0.9 (no epidemic) to 2.3 and the threshold density ranged from 424 to 221 susceptible animals. In addition, Guberti *et al.* calculated similar parameters for Eastern Sardinia based on an age stratified serological survey (32). The R_o ranged from 1.8 and 2.8, leading to a threshold density of 133 to 207 susceptible animals (corresponding to 0.6-1.1 wild boars/km^2).

Both models have similar N_T and R_o values, even if host densities are different. The scale of R_o expressed in units per disease/generation and subsequently converted into wild boars/km^2 primarily determines such a difference.

The figures reported do not have the meaning of absolute values but they should be considered as estimates. Both the calculated threshold densities are very low, and it appears clear that only a sustained depopulation effort can achieve eradication through density-dependent processes. Management of the infection should also consider the reproduction rate and thus rapid turn-over that might counteract control efforts. Models clearly indicate that the unstable equilibrium between depopulation and new-born recruitment might create a susceptible population large enough to maintain the infection (79).

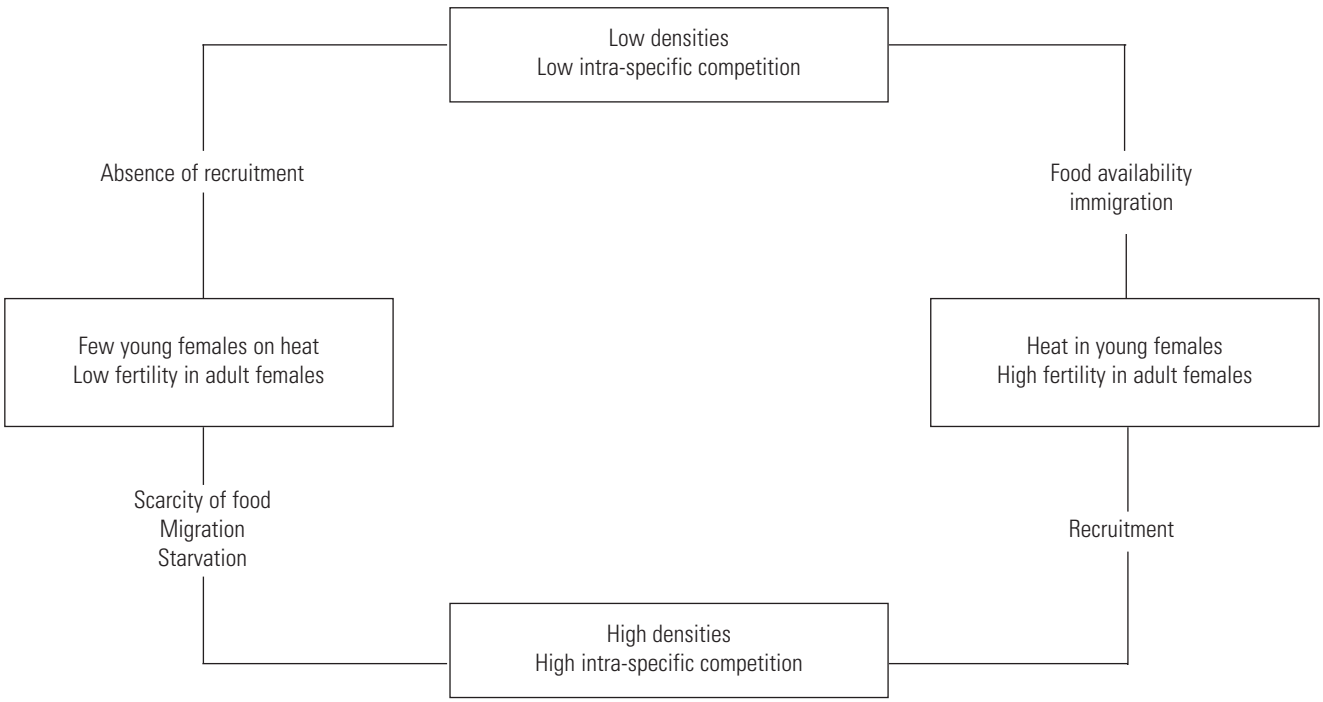


Fig. 6
Flow chart summarising population dynamics of wild boar populations

© OIE - 2002

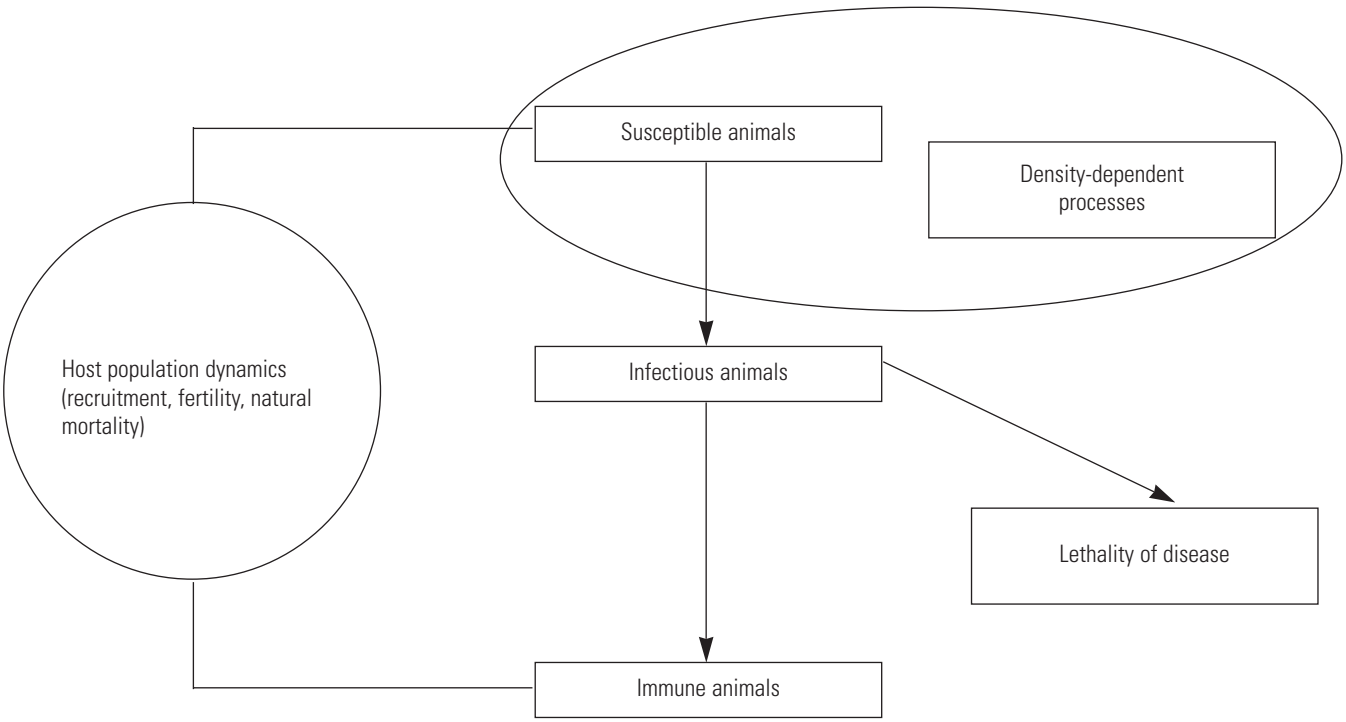


Fig. 7
Flow chart of a deterministic compartment model of classical swine fever in wild boar
Source: Guberti *et al.* (32)

Wild-boar management and control of classical swine fever

Culling is the tool most frequently used to manage wildlife which has been affected by a contagious disease (5). In the specific case of CSF in wild boar, the principal goals and scopes of such control are to be defined.

Population limitation does not represent the main goal of sports hunting; on the contrary, the aim is to achieve a viable, and possibly healthy, and well-structured population in terms of age and gender. Managed populations can increase in number each year; sports hunting techniques tend to maintain populations close to 50% of the level of the carrying capacity (notably in increasing such capacity by artificial feeding); at this level, the productivity of new-born animals is maximised as is the hunting bag.

Given the ecological elasticity of the wild boar, hunting pressure might be very ineffective in attempting to control population densities; the main effect of hunting can even be counter-productive since a consequence may be to reduce intra-specific food competition. As a result, the proportion of pregnant young females and the fertility of the adults will grow. Such hunted populations experience a reduction of the life-span of the animals and an increased turn-over, both of which lead to a younger population structure.

Side-effects of sports hunting are an increased home range size and frequent long-distance movements during dispersal which both result from disturbance, notably linked to the use of dogs (53, 86). Wild boar hunting bag records throughout Europe have tended to increase, confirming that current hunting management does not reduce the population of the species (33).

Population reduction efforts should be strictly related to specific goals; their effects must result in a reliable reduction of the population over the long-term. Population estimates are often lacking or are very vague, the desired recruitment rate and achievable density can be uncertain, so that the exact number of animals to be removed remains unknown. In practice, population management is largely unpredictable due to the limitation of available tools (even now shooting is the only method since trapping and poisoning are considered unfair or detrimental to the welfare of the animals). Population reduction can, however, be relatively efficient when the target is to reduce damage to agriculture and forestry, rather than eradicate the animals (4).

Feeding

Supplementary feeding may have positive as well as negative effects on the spread of the CSF virus. Feeding places attract the wild boars, thereby increasing the opportunity for close contact. This then facilitates transmission of CSF virus from infected to susceptible animals. In this way, CSF virus might be

© OIE - 2002

exported out of the original foci of infection (48). However, artificial feeding on a limited scale has the potential to maintain the boars within a well-defined area and to limit dispersal and slow transmission rate.

Vaccination

Oral immunisation of wild boar against CSF virus infection has been proposed as a prophylactic measure and as an additional tool for eradication. During the last decade, a conventional live virus-vaccine based on the attenuated CSF virus strain 'C' was used in several regions in Germany where CSF was endemic in wild boar (38). The vaccine used in Germany was encapsulated in blisters which were incorporated into cereal-based baits measuring 4 × 4 × 1.5 cm. The baits, which were distributed either by hand or by aeroplane, were well accepted by adult animals but not by wild boar younger than one year. Oral vaccination using baits is intended to further reduce the number of susceptible animals. The success of vaccination campaigns largely depends on the mass immunity conferred by vaccine baits which is dependent on the efficacy of the vaccine, together with the success of the bait delivery strategy. In a population which has already been infected, young animals are the principal target group for vaccination. However, to date, sero-conversion rates obtained by oral bait vaccines in young animals are inadequate, possibly because older animals are more likely to pick up the baits (38). The results of vaccination trials are still matter of discussion (42), mainly because it is difficult to differentiate the actual effect of the vaccination itself with other factors, such as naturally acquired immunity. Under certain circumstances, oral vaccination might provide an additional tool to existing CSF control measures.

Discussion

Several aspects of the difficulties which arise when CSF is present in a wild boar population have been addressed in this review. If it becomes clear that wild and domestic swine share the same strains of CSF virus, then the circumstances which allow the wild boar to act as a reservoir of infection and the appropriate measures to avoid this situation should be reviewed as proposed below.

Wild boars as reservoirs

The potential for wild boar to be reservoirs of CSF have been discussed by Aubert *et al.* (6). According to these authors, three main arguments which do not support the boar reservoir theory are put forward, as follows:

– where both *Suid* species co-exist in the wild and on farms, eradication of the infection in the domestic population has resulted in the disappearance of infection in the wild suids

– when CSF was released voluntarily in free-ranging boar populations, it was not self-sustaining

– in most circumstances, when appropriate information was collected about the origin of an outbreak among boars, human interference or errors were emphasised (mainly artificial feeding or burying of contaminated carcasses).

These considerations might be mitigated. In Sardinia, for instance, Laddomada *et al.* (43) consider that wild boar to wild boar transmission seems to play an important role in virus persistence. In contrast, African swine fever is unable to persist in the absence of cohabitation with free-roaming domestic swine.

Persistence of the infection has various mechanisms following an outbreak (39, 42), disappearance being simply one issue among others. Hypotheses suggested after observations made in Europe are that the size of the affected population, density and age structure, all play a crucial role in the shift from an epizootic to a persistent endemic situation. The larger the population size, the higher the density and the younger the age structure, the greater the chances of virus persistence.

Susceptible turn-over, mostly influenced by population regeneration with naïve boars born during the year, allows the infection reproductive rate, R_o to remain above 1. Analytical studies and modelling suggest that direct transmission, supported by persistent infection of juveniles, is the principal method of virus spread. Dispersal of adult virus shedders plays a limited role in diffusing the virus to neighbouring herds. Nevertheless, the shooting of the guiding sows can favour dispersal of their juveniles with resultant virus spread (28).

Transmission to domestic pigs from infected wild boars has never convincingly been proved to occur by aerosol or by contaminated natural products (earth, grass). Only the introduction of infected carcasses or feed, or close contact between free-ranging or inappropriately restricted domestic swine have been the cause of outbreaks on pig farms. As recently stated by the OIE Working Group on Wildlife Diseases (62), appropriate compartmentalisation can efficiently avoid cross-contamination of domestic swine, provided that effective measures are used to avoid introduction of contaminated material in pig settlements. Isolation of pig settlements is then the first safety measure for a prophylactic programme.

Nevertheless, the fact remains that when infection is present among free-ranging wild boars, the danger of virus introduction by ignorance or spite is increased; the longer the period that infection has persisted, the greater the risk of introduction onto farms. Consequently active CSF control remains an important option.

Management

Taking into account the ecological and epidemiological factors discussed above, the management of CSF among wild boars should follow a logical progression (surveillance and

© OIE - 2002

monitoring of the infection at first, followed by control and eradication of the disease) (14).

Sample collection

As wild boar populations are, by definition, elusive and difficult to assess, disease surveillance and monitoring pose difficulties. It is therefore essential to define the purpose of sampling with precision; surveillance should be organised in any area in which boars are abundant (59). According to European Union recommendations, it would be sensible to screen for CSF virus in any boar found dead for unknown reasons (14).

When CSF poses a threat, or in areas in which an outbreak is present, the infected area needs to be delineated and an estimate needs to be made of the size of the targeted population, not only simply as a function of animals that have been shot. According to European experts, natural and artificial borders (rivers, highways) are useful to delimit management units (3). In these units, estimates of the size of the wild boar population should be based on bag records and various direct and indirect factors, such as the hunting indicator of population density (HIPD). A rough estimation, or the likely range are better than no assessment at all.

If detection of the presence/absence of the infection is targeted, an infection rate threshold of 2.5% is a minimum which can be achieved by available tests (91). At this level, infection can be detected with a 95% confidence level in a population of at least one thousand individuals by sampling some 60 animals at random. As sports hunters have a preference for juvenile animals, the very young and adult members of the population are under-represented if this method is used for sampling. Therefore, sampling for epidemiological studies must be structured in terms of age and gender to avoid a bias linked to hunter preference for certain age classes. Ideally, the sample will be made up of 50% of animals belonging to the 3-month to 1-year age category, 35% to the 1- to 2-year category and 15% to those animals over 2 years of age (15).

When infection is detected, prevalence can only be estimated with precision by pooling the specificity and sensitivity of tests, population size and an hypothesis on the actual prevalence rate. For instance, using a specific and sensitive test, estimation of a prevalence close to 20% ± 8% (rank of order when CSF infection commences in a susceptible wild boar population) will be possible by examining about 100 wild boar tissue or blood samples drawn at random (82).

Special precautions must be taken not to lose any samples during transportation or storage as a result of haemolysis, putrefaction or virus destruction.

Disease surveillance requires close co-operation between the authorities in the affected areas (provinces, states or countries) to build confidence in the transfer of information. The OIE Working Group on Wildlife Diseases has underlined that a country should be encouraged to report diseases and infection among its wild animal population, and should not be penalised with regard to agricultural trade, if the same disease does not occur in domestic animals simultaneously (61).

Sampling should be designed to avoid errors in the estimation of infection prevalence: representativeness of individuals included in the sample and sufficient precision of prevalence rates to detect the infection at low levels are essential. The quality of test is of vital importance. When infection rates are low, sensitive tests are essential. Specificity can be checked afterwards if a sample gives a positive test result. Even if actual population size is difficult to assess, evaluation should be attempted and can be improved over time. Transport and storage of good blood or tissue samples poses specific difficulties; according to the experience of the authors, up to 30%, and even exceptionally 50%, of field samples can be of no value for laboratory tests due to damage (poor handling practices transport and duration of storage).

Control

Firstly, all programmes designed to control CSF in wild boar populations should be evaluated and monitored by a group of experts, including wildlife epidemiologists. Infected areas have to be marked, clusters delineated and the number of sub-populations that inhabit the area must be recorded. Among the direct control measures, a temporary hunting ban should be considered immediately after the initial declaration of CSF infection has been made. During the following breeding season, a hunting plan could be instituted which might involve the preservation of adults (considered to be naturally immunised), together with hunter targeting of gruntlings, as well as juveniles born during or after the beginning of the outbreak. At this stage, artificial feeding should be prohibited, except linear distribution of corn grains aimed at limiting movement of foraging boars (feed available to boars should be not sufficient to increase the productivity of the sows). The use of running hunting dogs should be banned to avoid dispersing the boar outside the infected area during sports hunting sessions.

If the expected decrease in the infection rate is not achieved within the expected time, it may be necessary to delineate the foci of infection more precisely, which can be restricted in space (S. Rossi and colleagues, unpublished data). Within these foci, depopulation using a combination of techniques might be considered a temporary measure, if it is more effective than shooting alone. Although emergency vaccination has not been extensively evaluated at this time, such programmes may be instituted to target specifically identified sub-populations that are maintaining the infection, so as to reduce the number of susceptible animals below the threshold of transmission. It is supposed that this threshold is reached by a number of about 200 susceptible animals in an area of approximately 220 km^2. The required efficacy of vaccination is related to the estimated population. If 1,000 wild boar are present, a sero-conversion rate of 80%, should be reached. If only 500 wild boar are

© OIE - 2002

present, the rate should be at least about 60% (2). Even if available CSF vaccines do not pose problems for human health and do not present any known danger for non-target species, environmental risk assessment analysis should be applied before proceeding.

Control programmes should last at least two years. Efficacy can be investigated using rigorous criteria. In the 'treated' areas, hunting should be reduced or forbidden in order to maintain a stable population in which the majority of animals is immune for as long as possible.

Difficulties can be anticipated with the co-operation of hunters in areas where sports hunting is popular. For most hunters, CSF is not regarded as a concern, but a hunting ban or depopulation can have a severe impact on their expected income. Information should be released on the potential impact of CSF on the economy, encouraging the responsibility of hunters, and it should be explained that the negative effects of controls on sports hunting will be temporary. Compensatory measures should be considered to stimulate the co-operation of hunters.

Wild boars are highly prized by the hunting fraternity, but are frequently regarded as pest or problem animals for the following reasons:

– agriculture (crop damage)

– territory management (road casualties)

– pig farming industry (disease transmission: swine brucellosis, encephalomyocarditis, porcine reproductive and respiratory syndrome, Aujeszky's disease)

– public health (zoonotic infections could include influenza, *Mycobacterium bovis* infection and trichinellosis; fatalities due to *Streptococcus suis* septicaemia has occurred recently in hunters).

For the reasons discussed above, wild boar management should not be limited to satisfy sports hunting. Reduction of damage to crops and veterinary and human health problems should be addressed in the management programmes. Artificial feeding and hunting management should target a population level below the threshold at which agricultural damage or disease become problematic. Translocation and release of wild boars should be strictly controlled, and cross-breeding with any breed of domestic swine should be forbidden.

Acknowledgements

The authors and co-authors are grateful to the French national game agency (ONCFS) for providing accurate data on wild boar bag records and to their numerous colleagues from the Agence française de sécurité sanitaire des aliments (AFSSA) and Veterinary Services who provided support to their studies; Ms M.-J. Duchêne, from AFSSA Nancy, was particularly helpful in providing many useful references. Comments received on the manuscript from Dr R.G. Bengis and Dr A. Laddomada were greatly appreciated.

■

Peste porcine classique chez les sangliers sauvages en Europe

M. Artois, K.R. Depner, V. Guberti, J. Hars, S. Rossi & D. Rutili

Résumé

La peste porcine classique devient un sujet de préoccupation croissant en Europe, où les sangliers sauvages semblent jouer un rôle épidémiologique majeur. Sur une bonne partie du continent, leurs populations augmentent, grâce aux progrès enregistrés en matière de gestion de la chasse. Avec l'augmentation des densités, les populations deviennent plus sensibles à diverses maladies infectieuses, dont la peste porcine classique qui suscite une inquiétude particulière. Les sangliers sauvages ne sont généralement pas des réservoirs classiques de la maladie, mais ils peuvent entretenir des foyers d'infection sur de longues durées, faisant peser une menace réelle sur la filière porcine. Étant donné que l'infection ne semble pas se propager rapidement dans les populations

© OIE - 2002

de sangliers sauvages vivant en liberté, la lutte contre la maladie paraît tout à fait envisageable. Toutefois, la plupart des mesures qui pourraient être appliquées, telles que l'interdiction de la chasse, ne sont pas jugées acceptables. Il convient, par conséquent, de recourir aux compétences de spécialistes des maladies de la faune sauvage pour aider à résoudre le problème lorsqu'il se présente.

Mots-clés
Épidémiologie – Europe – Faune sauvage – Peste porcine classique – Prophylaxie – Sanglier sauvage – Sus scrofa.

Peste porcina clásica en jabalíes salvajes europeos

M. Artois, K.R. Depner, V. Guberti, J. Hars, S. Rossi & D. Rutili

Resumen
La peste porcina clásica despierta cada vez más preocupación en Europa, donde el jabalí parece desempeñar un papel importante en la epidemiología de la enfermedad. En la mayor parte del continente las tendencias demográficas de las poblaciones de jabalíes apuntan al alza, debido a las mejoras en la gestión de la caza. Las elevadas densidades incrementan la sensibilidad de las poblaciones a diversas enfermedades infecciosas, entre las cuales suscita especial inquietud la peste porcina clásica. Aunque el jabalí salvaje no parece ser casi nunca un reservorio clásico, a la larga sus poblaciones pueden perpetuar los focos de infección, lo que supone una real amenaza para el sector de la producción porcina. Dado que la infección no parece propagarse con facilidad en poblaciones de jabalíes en libertad, debería resultar factible controlarla. Sin embargo, muchas de las medidas adecuadas para ello, como la prohibición de la caza, no se consideran aceptables. De ahí la necesidad de contar con especialistas en enfermedades de la fauna salvaje que ayuden a resolver el problema en cuanto se presenta.

Palabras clave
Control – Epidemiología – Europa – Fauna salvaje – Jabalí – Peste porcina clásica – Sus scrofa.

References

1. Albina E., Mesplède A., Chenut G., Le Potier M.F., Bourbao G., Le Gal S. & Leforban Y. (2000). – A serological survey on classical swine fever (CSF), Aujeszky's disease (AD) and porcine reproductive and respiratory syndrome (PRRS) virus infections in French wild boars from 1991 to 1998. *Vet. Microbiol.*, **77** (1-2), 43-57.

2. Anderson R.M. & May R.M. (1991). – Infectious diseases of humans. Dynamics and control. Oxford University Press, Oxford, 757 pp.

3. Anon. (1999). – Classical swine fever in wild boar. Scientific committee on animal health and animal welfare. Report of the European Commission (EC). Document XXIV/B3/R09/1999, EC, Brussels, 46 pp.

4. Artois M. (1997). – Managing problem wildlife in the Old World: a veterinary perspective. *Reprod. Fertil. Dev.*, **9**, 17-25.

5. Artois M., Delahay R., Guberti V. & Cheeseman C. (2001). – Control of infectious diseases of wildlife in Europe. *Vet. J.*, **162**, 141-152.

© OIE - 2002

6. Aubert M., Picard M., Fouquet E., Conde J., Crucière C., Ferry R., Albina E., Barrat J. & Vedeau F. (1994). – La peste porcine classique du sanglier en Europe. *Ann. Méd. vét.*, **138**, 239-247.

7. Biagetti M., Greiser-Wilke I. & Rutili D. (2001). – Molecular epidemiology of classical swine fever in Italy. *Vet. Microbiol.*, **83** (3), 205-215.

8. Blancou J., Barrat J., Devaud J.P., Vannier Ph. & Gourreau J.M. (1987). – Pathologie du sanglier (*Sus scrofa* L.) : connaissances actuelles en France. *Gibier Faune sauv.*, **4**, 279-294.

9. Boitani L. (2000). – Action plan for the conservation of wolves in Europe. Nature and Environment, No. 113. Council of Europe Publishing, Strasbourg, 86 pp.

10. Breitenmoser U., Breitenmoser-Wursten C., Okarma Kaphegyi H., Kaphegyi-Wallmann U. & Muller U.M. (2000). – Action plan for the conservation of the Eurasian lynx in Europe. Nature and Environment, No. 112. Council of Europe Publishing, Strasbourg, 70 pp.

11. Brugh M., Foster J.W. & 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.

12. Cliff A. (1995). – Incorporating spatial components into models of epidemic spread. *In* Epidemic models: their structure and relation to data (D. Mollison, ed.). Publications of the Newton Institute, Cambridge University Press, 119-149.

13. Colijn E.O., Blomraad M. & Wensvoort G. (1997). – An improved ELISA for the detection of serum antibodies directed against classical swine fever virus. *Vet. Microbiol.*, **59**, 15-25.

14. Commission of the European Communities (2001). – Council Directive 2001/89/EC of 23 October 2001 on Community measures for the control of classical swine fever. *Off. J. Eur. Communities*, No. **L 316** of 1.12.2001, 5-35.

15. Commission of the European Communities (CEC) (2002). – Diagnostic manual for the confirmation of classical swine fever. CEC, Brussels, 30 pp.

16. Dahle J. & Liess B. (1992). – A review on classical swine fever infections in pigs: epizootiology, clinical disease, and pathology. *Comp. Immunol. Microbiol. infect. Dis.*, **15**, 203-211.

17. Dahle J., Patzelt Th., Schgemann G. & Liess B. (1993). – Antibody prevalence of hog cholera, bovine viral diarrhoea and Aujeszky's disease virus in wild boars in Northern Germany. *Dtsch. tierärztl. Wochenschr.*, **100** (8), 301-340.

18. Dedek J., Loepelmann H. & Kokles R. (1989). – Ehrgebnisse Flächendeckender serologischer untersuchungen beim Schwarzwild (*Sus scrofa*) in einem Bezirk des DDR. *In* Verhandlungsbericht des 31. Internationalen Symposiums über die Erkrankungen der Zoo- und Wildtiere, Dortmund.

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

20. Depner K.R., Moennig V. & Liess B. (1997). – Epidemiologische Aspekte der Infektionbiologie der klassichen Schweinpest. *Prakt. Tierarzt Coll. vet.*, **XXVII**, 63-67.

21. Depner K.R., Muller T., Lange E., Staubach C. & Teuffert J. (2000). – Transient classical swine fever virus infection in wild boar piglets partially protected by maternal antibodies. *Dtsch. tierärztl. Wochenschr.*, **107** (2), 41-80.

22. Edwards S., Fukusho A., Lefèvre P.-C., Lipowski A., Pejsak Z., Roehe P. & Westergaard J. (2000). – Classical swine fever: the global situation. *Vet. Microbiol.*, **73**, 93-102.

23. Elbers K., Tautz N., Becher P., Stoll D., Rumenapf T. & Thiel H.J. (1996). – Processing in the pestivirus E2-NS2 region: identification of protein p7 and E2pt. *J. Virol.*, **70**, 4131-4135.

24. Englert H.K. (1953). – Enzootische Schweinpest beim Schwartzwild im Odenwald. *Tierärztl. Umsch.*, **8**, 124-127.

25. Firinu A. & Scarano C. (1988). – La peste porcine africaine et la peste porcine classique chez le sanglier en Sardaigne. *In* Wildlife diseases. *Rev. sci. tech. Off. int. Epiz.*, **7** (4), 901-908.

26. Fritzemeier J., Greiser-Wilke I., Depner K.R. & 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, 6-7 April, Perugia, Italy. Commission of the European Community, Doc. VI/7196/98 AL, Brussels, 107-109.

27. Froelich K., Van Campen H. & Hofmann M. (2001). – Classical swine fever virus (hog cholera virus) infections, Chapter 12: Pestivirus infections. *In* Infectious diseases of wild mammals, 3rd Ed. (E.S. Williams & I.K. Barker, eds). Iowa State University Press, Ames, 237-240.

28. Genov P. & Ferrari G. (1998). – Effect of hunting on the use of space and habitat of wild boars. *In* Report on measures to control classical swine fever in European wild boar, 6-7 April, Perugia, Italy. Commission of the European Community, Document VI/7196/98 AL, Brussels, 32-36.

29. Glowacinski Z. & Profus P. (1997). – Potential impact of wolves *Canis lupus* on prey populations in Eastern Poland. *Biol. Conserv.*, **80**, 99-106.

30. Graves H.B. (1984). – Behaviour and ecology of wild and feral swine (*Sus scrofa*). *J. anim. Sci.*, **58**, 482-492.

31. Griot C., Thür B., Vanzetti T., Schmidt J. & Hofmann M.A. (1999). – Classical swine fewer in wild boars: a challenge for any veterinary service. *In* Proc. 103rd Annual Meeting of the United States Animal Health Association (USAHA), 7-14 October, San Diego. USAHA, Richmond, Virginia (http://www.usaha.org/speeches/speech99/s99griot.html, accessed on 18 January 2002).

32. Guberti V., Rutili D., Ferrari G., Patta C. & Oggiano A. (1998). – Estimate the threshold abundance for the persistence of the classical swine fever in the wild boar population of the Eastern Sardinia. *In* Report on measures to control classical swine fever in European wild boar, 6-7 April, Perugia, Italy. Commission of the European Community, Doc. VI/7196/98 AL, Brussels, 54-61.

© OIE - 2002

33. Hars J., Albina E., Artois M., Boireau P., Crucière C., Garin-Bastuji B., Gauthier D., Hatier C., Lamarque F., Mesplede A. & Rossi S. (2000). – Epidémiosurveillance des maladies du sanglier transmissibles aux animaux domestiques et à l'homme. *Epidémiol. Santé anim.*, **37**, 31-43.

34. Hars J., Rossi S. & Pacholek X. (2001). – Peste porcine classique du sanglier. Bilan du suivi épidémiologique du foyer des Vosges du Nord entre 1992 et 2001. Rapport interne DGAl/ONCFS. Ministère de l'agriculture et de la pêche, Paris, 23 pp.

35. Hofmann M.A. & Bossy S. (1998). – Klassische Schweinpest 1993 in der Schweiz: molecular-epidemiologische Charakterisierung der Virusisolate. *Schweizer Arch. Tierheilkd.*, **140**, 365-370.

36. Hofmann M.A., Thür B., Vanzetti T., Schleiss W., Schmidt J. & Griot C. (1999). – Klassische Schweinepest beim Wildschwein in der Schweiz. *Schweizer Arch. Tierheilkd.*, **141**, 185-190.

37. Hone J., Pech R. & Yip P. (1992). – Evolution of the dynamics and rate of transmission of classical swine fever (hog cholera) in wild pigs. *Epidemiol. Infect.*, **108**, 377-386.

38. Kaden V., Lange E., Fisher U. & Strebelow G. (2000). – Oral immunisation of wild boar against classical swine fever: evaluation of the first field study in Germany. *Vet. Microbiol.*, **73**, 239-252.

39. Kern B., Depner K.R., Letz W., Rott M., Thalheim S., Nitschke B., Plagemann R. & 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. *J. vet. Med., B*, **46**, 63-67.

40. Korn G., Matthaeus W., Lorenz R.J. & Jakubik J. (1973). – Über eine Globulin/Transferin- und Antikörperbildungsstörung während des Leukopeniestadiums der Schweinepester-krankung. *Zentralbl. immunol. Forsch.*, **145**, 139-155.

41. Krassnig R. & Schuller W. (1993). – Schweinepest in Österreich. *Wien. tierärztl. Monatsschr.*, **80**, 229-233.

42. Laddomada A. (2000). – Incidence and control of CSF in wild boar in Europe. *Vet. Microbiol.*, **73**, 121-130.

43. Laddomada A., Patta C., Oggiano A., Caccia A., Ruiu A., Cossu P. & Firinu A. (1994). – Epidemiology of classical swine fever in Sardinia: a serological survey of wild boar and comparison with African swine fever. *Vet. Rec.*, **134**, 183-187.

44. Leforban Y. (1991). – Pathogenicity for domestic pigs of the HCV strain isolated from wild boar in Priziac (Brittany) in August 1990. *In* Report on meeting of natural swine fever laboratory within the European Community (EC), 27-28 May, Brussels. EC, Brussels, 66.

45. Leforban Y. & Cariolet R. (1992). – Characterization and pathogenicity of a hog cholera virus strain isolated from wild boars. *Ann. Rech. vét.*, **23**, 93-100.

46. Liess B. (1987). – Pathogenesis and epidemiology of hog cholera. *Ann. Rech. vét.*, **18**, 139-145.

47. Lin T.C., Shimizu Y., Kumagai T. & Sahara J. (1969). – Pathogenesis of hog cholera infection in experimentally inoculated swine. *Natl Inst. anim. Hlth Q. (Tokyo)*, **9**, 20-27.

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

49. Lowings J.P., Paton D.J., Sands J.J., De Mia G.M. & Rutili D. (1994). – Classical swine fever: genetic detection and analysis of differences between virus isolates. *J. gen. Virol.*, **75**, 3461-3468.

50. Lowings P., Ibata G., De Mia G.M., Rutili D. & Paton D. (1999). – Classical swine fever in Sardinia: epidemiology of recent outbreaks. *Epidemiol. Infect.*, **122**, 553-559.

51. Lutz W. & Wurm R. (1996). – Serologische Untersuchungen zum Nachweis von Antikörpern gegen Viren des Seuchenhaften Spätaborts, des Aujeszkyschen Krankheit, des Europäischen Schweinpest und Porzine Parvovirus beim Wildschwein (*Sus scrofa*, L. 1758) in Nordrhein-Westfalen. *Zeitschr. Jagdwissensch.*, **42**, 123-133.

52. McKercher P.D., Yedloutschnig R.J., Callis J.J., Murphy R., Panina G.F., Civardi A., Bugnetti M., Foni E., Laddomada A., Scarano C. & Scatozza F. (1987). – Survival of viruses in 'Prosciutto di Parma' (Parma ham). *Can. Inst. Food Sci. Technol. J.*, **20** (4), 267-272.

53. Maillard D. & Fournier P. (1995). – Effects of shooting with hounds on size and resting range of wild boar (*Sus scrofa*) in Mediterranean costal area. *Ibex, J. Mount. Ecol.*, **3**, 102-107.

54. Matthaeus W. & Korn G. (1967). – Die neutralisierenden Antikörper im Schwein nach experimenteller Infektion mit Schweinepestvirus. *Zentralbl. Bakteriol.*, **204**, 173-180.

55. Mengeling W.L. & Cheville N.F. (1968). – Host response to persistent infection with hog cholera virus. *In* Proc. 72nd Annual Meeting of the United States Animal Health Association (USAHA). USAHA, Richmond, Virginia, 283-296.

56. Mengeling W.L. & Packer R.A. (1969). – Pathogenesis of chronic hog cholera: host response. *Am. J. vet. Res.*, **30**, 409-417.

57. Meuwissen M.P.M., Horst H.S., Huirne R.B.M. & Dijkhuizen A.A. (1999). – A model to estimate the financial consequences of classical swine fever outbreaks: principals and outcomes. *Prev. vet. Med.*, **42**, 249-270.

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

59. Mörner T., Obendorf D.L., Artois M. & Woodford M.H. (2002). – Surveillance and monitoring of wildlife diseases. *In* Infectious diseases of wildlife: detection, diagnosis and management, Part One (R.G. Bengis, ed.). *Rev. sci. tech. Off. int. Epiz.*, **21** (1), 67-76.

60. Nettles V.F., Corn J.L., Erickson G.A. & Jessup D.A. (1989). – A survey of wild swine in the United States for evidence of hog cholera. *J. Wildl. Dis.*, **25** (1), 61-65.

61. Office International des Epizooties (OIE) (1999). – Report of the Working Group on Wildlife Diseases, 6-8 October 1998, Teramo, Italy. *In* 67th General Session of the International Committee, Document 67 SG/13/GT 2. OIE, Paris, 12 pp.

62. Office International des Epizooties (OIE) (2000). – Report of the Working Group on Wildlife Diseases, 19-21 October 1999, OIE, Paris. *In* 68th General Session of the International Committee, Document 68 SG/13/GT 1. OIE, Paris, 17 pp.

63. Office International des Epizooties (OIE) (2001). – Manual of standards for diagnostic tests and vaccines, 4th Ed, 2000. OIE, Paris, 957 pp.

© OIE - 2002

64. Office International des Epizooties (OIE) (2001). – Classical swine fever in Luxembourg: in wild boar. *Dis. Info.*, **14** (44), 2 November.

65. Office International des Epizooties (OIE) (2001). – Classical swine fever in Slovakia. *Dis. Info.*, **14** (27), 6 July.

66. Office International des Epizooties (OIE) (2001). – Classical swine fever in Ukraine. In wild boar. *Dis. Info.*, **14** (29), 20 July.

67. Okarma H., Jedrezejewska B., Jedrezejewski W., Krasinski Z.A. & Milkowski L. (1995). – The roles of predation, snow cover, acorn crop, and man-related factors on ungulate mortality in Bialowieza Primeval Forest, Poland. *Acta theriol.*, **40** (2), 197-217.

68. Oslage U. von, Dahle J., Muller Th., Kramer M., Beier D. & Liess B. (1994). – Prävalenz von Antikörpen gegen die Viren des Europäischen Schweinpest, des Aujeszky'schen Krankheit und des 'Porcine reproductive and respiratory syndrome' (PRRS) bei Wildschweinen in des Bundesländern Sachsen-Anhalt und Brandenburg. *Dtsch. tierärztl. Wochenschr.*, **101** (1), 1-40.

69. Patta C., Oggiano A., Cattina A., Vargiu M.P., Pala G., Melis P. & Ladu A. (1998). – Control of CSF in wild boar in Sardinia. *In* Report on measures to control classical swine fever in European wild boar, 6-7 April, Perugia, Italy. Commission of the European Community, Doc. VI/7196/98 AL, Brussels, 91-92.

70. Pedrotti L., Monaco A. & Franzetti B. (1999). – Testing a wild boar management strategy in an area of Northern Apennines (Bologna, Italy). *In* Abstract Booklet, XXIV Congress of the International Union of the Game Biologists (IUGB), 20-24 September, Thessaloniki. IUGB, Thessaloniki, 69.

71. Ressang A.A. (1973). – Studies on the pathogenesis of hog cholera. I. demonstration of hog cholera virus subsequent to oral exposure. *Zentralbl. Veterinärmed., B*, **20**, 272-288.

72. Rutili D. & Laddomada A. (1995). – Epidemiological surveillance of CSF on wild boar in Sardinia. *In* Annual Meeting of National Swine Fever Laboratories, 27-28 September, Valdeolmos, Madrid. Document IV/1779/96. Commission of European Communities, Brussels, 25-27.

73. Rutili D., Guberti V. & Ferrari G. (1998). – Classical swine fever in wild boar. Evaluation of control measures applied in Italy and proposal for the future. *In* Report on measures to control classical swine fever in European wild boar, 6-7 April, Perugia, Italy. Commission of the European Community, Doc. VI/7196/98 AL, Brussels, 135-137.

74. Saez-Royuela C. & Telleria J.L. (1986). – The increased population of the wild boar (*Sus scrofa* L.) in Europe. *Mammal Rev.*, **16** (2), 97-101.

75. Savi P., Torlone V.Y. & Titoli F. (1965). – Recherches sur la survie du virus de la peste porcine classique dans certains produits de charcuterie. *Bull. Off. int. Epiz.*, **63**, 87-96.

76. Schlüter H., Teuffert J., Staubach C. & Kramer M. (2000). – Monitoring of wild boar for classical swine fever. *In* Report of Symposium on classical swine fever in the wild boar, 19 May, Greifswald, Germany. Commission of European Communities, Brussels, SANCO/3916/2000, 36-57.

77. Spitz F., Janeau G. & Valet G. (1984). – Eléments de démographie du sanglier (*Sus scrofa*) dans la région de Grésigne. *Acta oecol.*, **5** (1), 43-59.

78. Stubbe I. & Stubbe W. (1994). – Weitere Ergebnisse serologischer Untersuchungen an Schalenwild und Hasen aus dem Waldgebiet Hakel. *Beitr. Jagd Wildforsch.*, **19**, 135-140.

79. Swinton J., Woolhouse M.E.J., Dobson A., Ferroglio E., Guberti V., Grenfell B.T., Heesterbeek J.A.P., Hails R., Lavazza A., Roberts M. & White P.J. (2002). – Microparasite transmission and persistence. *In* Ecology of wildlife diseases (P. Hudson, A. Rizzoli, B. Grenfell & H. Heesterbeek, eds). Oxford University Press, 272 pp.

80. Terpstra C. (1988). – Epizootiology of hog cholera. *In* Classical swine fever and related viral infections (B. Liess, ed.). Martinus Nijhoff Publishing, Boston, 201.

81. Teuffert J., Schlüter H. & Kramer M. (1997). – Europäische Schweinepest. Übersicht zur internationalen (Europa) und nationalen Schweinepestsituation – ermittelte Einschleppungsursachen und Verschleppungsrisiken. *Dtsch. Tierärztebl.*, **11** (97), 1078-1080.

82. Thrusfield M. (1995). – Veterinary epidemiology, 2nd Ed. Blackwell Science, Oxford, 479 pp.

83. Trautwein G. (1988). – Pathology and pathogenesis of the disease. *In* Classical swine fever and related viral infections (B. Liess, ed.). Martinus Nijhoff Publishing, Dordrecht, 27-54.

84. Van Oirshot J.T. (1988). – Description of the virus infection. *In* Classical swine fever and related viral infections (B. Liess, ed.). Boston, 1-25.

85. Van Oirshot J.T. & Terpstra C. (1977). – A congenital persistent swine fever infection. I. Clinical and virological observations. *Vet. Microbiol.*, **2**, 121-132

86. Veys J. (2001). – Contribution à l'étude de l'épidémiologie de la peste porcine classique du sanglier. Étude sérologique sur des marcassins. Thèse Méd. vét., Lyons, 105 pp.

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

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

89. Widjojoatmoto M.N., Van Gennip H.G.P., de Smit A.J. & Moormann R.J.M. (1999). – Comparative sequence analysis of classical swine fever virus isolates from the epizootic in the Netherlands in 1997-1998. *Vet. Microbiol.*, **66**, 291-299.

90. Wilson E.O. (1975). – Sociobiology. The new synthesis. Harvard University Press, Cambridge, 697 pp.

91. Wobeser G. (1994). – Investigation and management of disease in wild animals. Plenum Press, New York & London, 265 pp.

© OIE - 2002