A review of viral diseases of the European wild boar: Effects of population dynamics and reservoir rôle

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Abstract

There has been a worldwide increase in the number and geographical spread of wild boar populations in recent decades leading to an increase in both the circulation of disease agents and greater contact with domestic animals and humans. Diseases affect the population dynamics of wildlife but the effects of most viral diseases on the European wild boar are largely unknown. Many viral diseases present in domestic pig populations are also present in wild boars where they can provide a disease reservoir, as is clearly the case with classical swine fever, but little is known about other viral diseases such as porcine circovirus diseases or hepatitis E. This review considers the current scientific knowledge of the effects of viral diseases on wild boar populations and their rôle as potential disease reservoirs. The focus is on those viral diseases of domestic swine and wild boars that are included as notifiable by the Office International des Epizooties (OIE).

Keywords: Domestic pig; Population dynamics; Reservoir; Viral diseases; Wild boar

1. Introduction

Worldwide, the population density of the wild boar seems to be increasing (Saez-Royuela and Telleria, 1986; Gortazar et al., 2000; Acevedo et al., 2006), which not only means a larger number of hosts available for the transmission of disease, but also a higher contact rate between hosts (Acevedo et al., 2007). Knowledge of diseases circulating in wildlife populations can be important not only for conservation and livestock production but also for public health, and in this article we review current knowledge of viral diseases of the wild boar, emphasizing the effect on populations and the rôle that the wild boar may play as a viral disease reservoir for domestic pigs. Special attention will be paid to diseases listed as notifiable by the Office International des Epizooties (OIE). The distribution and ecological features that are of importance for a better understanding of the effects of viral diseases on wild boar population dynamics and pathogen circulation among wild populations are also addressed.

2. Wild boar distribution and population dynamics

Wild boars naturally inhabit vast areas of Europe and North Africa, extending to Sri Lanka, Indonesia, Japan, Taiwan and Korea. As a result of introductions, they are also found in areas far from their original distribution (Lever, 1994). In most areas where the wild boar has been introduced, hybridization with free-roaming domestic pigs has led to crossbreeding, producing what is often referred to as a feral pig, feral swine or feral hog. Feral pigs are common in the Southern USA, Australia and New Zealand (Mayer and Brisbin, 1991; Oliver and Brisbin, 1993; Waithman et al., 1999; Woodal, 1983). In this article, the term “wild boar” will be used both for the European wild boar...
and for feral pigs; in specific cases, the European wild boar and the feral pig will be distinguished as a consequence of their areas of distribution.

Although the wild boar disappeared from many parts of Europe by the end of the 17th century (Harting, 1880; Tisdell, 1982), their numbers increased again during the latter half of the 20th century (Saez-Royuela and Telleria, 1986; Gortazar et al., 2000; Acevedo et al., 2006). Wild boar densities from some studied populations are shown in Table 1. The increase in population density of the wild boar raises concerns regarding individual fitness and welfare, vegetation damage and an increasing prevalence of infectious diseases and parasites (Gortazar et al., 2006; Ruiz-Fons et al., 2006a).

3. Wild boar spatial ecology and social structure

Wild boars are present in a wide variety of environments across their distribution area, although a preference for forest habitat exists (Abaigar et al., 1994). They are gregarious animals, living in groups of variable sizes. Females with their offspring are the most frequent group pattern observed under natural conditions (Teillaud, 1986; Ahrens, 1984; Rosell et al., 2004). Adult males are seen to form groups in autumn and winter, although males usually display solitary behaviour (Fernández-Llario et al., 1996; Rosell et al., 2004). They tend to aggregate spatially due to social behaviour and irregular food availability, especially in the autumn months (Dardaillon, 1984; Rosell, 1998).

High densities and the scarcity of water in Mediterranean countries during the summer also contribute to wild boar aggregation, and it has been suggested that social behaviour differences play a role in the epidemiology of some viral diseases such as Aujeszky’s disease (Vicente et al., 2005). The social structure should therefore be taken into account in implementing disease control programs.

4. Significant viral diseases in wild boar populations

The wild boar and the domestic pig share pathogens (Lipowski, 2003). When a particular pathogen establishes a long-life cycle among a wild species, it becomes a reservoir. Although a disease can be sometimes controlled and eradicated in livestock despite the presence of a wildlife reservoir (see, for example, Lutz et al., 2003), the risk of pathogen transmission from the remaining wildlife reservoir inevitably poses a threat to the success of the campaigns.

In this review, the viral diseases we shall consider as important are those that have a direct effect on wild boars and an economic impact on domestic pig production systems. Current knowledge of viral infection status in domestic pigs and wild boars as well as the clinical manifestations of these diseases are summarised in Tables 2 and 3.

4.1. Aujeszky’s disease (pseudorabies)

Aujeszky’s disease (AD) is caused by suid alphaherpesvirus 1, also called pseudorabies virus. The domestic pig and the wild boar are natural hosts but the virus can also infect other mammals causing a fatal nervous disease (Pejsak and Truszczynski, 2006). AD remains one of the most important diseases of domestic pigs worldwide and is included as an OIE notifiable disease, although many countries have successfully eradicated it at least in domestic pig herds (Moynahg, 1997).

Effects of ADV in wild boar populations. European wild boar and feral pig populations have been reported to be infected by ADV almost worldwide in a variable proportion (Müller et al., 2000; Lipowski, 2003; Lutz et al., 2003; Vengust et al., 2005; Vicente et al., 2005). In the domestic pig, ADV infection causes respiratory, reproductive and central nervous clinical signs (Pejsak and Truszczynski, 2006).

Gortazar et al. (2002) reported an outbreak of AD in wild boars in South-central Spain, where nervous clinical signs were observed. Mortality was 14% of the juveniles and 7% of the adults affected. Müller et al. (2001) observed only mild temperature increases, some sneezing, slight nasal discharge and conjunctivitis in wild boars experimentally infected with an ADV strain of wild boar origin although after immunosuppressive treatment the animals showed severe respiratory signs and died or were euthanased. Hahn et al. (1997) concluded that ADV strains of feral pig origin were attenuated when compared to those of domestic pigs. Obviously, new and more virulent ADV strains could have consequences in the population dynamics of wild boars, especially in dense populations.

The wild boar as an ADV reservoir for the domestic pig. As ADV is widespread in wild boar populations, it is important to consider their possible role as reservoir for the domestic pig. Infection spread from the domestic pig

<table>
<thead>
<tr>
<th>Country (region)</th>
<th>Wild boars (km²)</th>
<th>References</th>
</tr>
</thead>
<tbody>
<tr>
<td>Aragón (Spain)</td>
<td>2.8–4.2</td>
<td>Herrero et al. (1995)</td>
</tr>
<tr>
<td>Burgos (Spain)</td>
<td>1.9–4.2</td>
<td>Telleria and Saez-Royuela (1986)</td>
</tr>
<tr>
<td>León (Spain)</td>
<td>1.7–11.4</td>
<td>Purroy et al. (1988)</td>
</tr>
<tr>
<td>Cataluña (Spain)</td>
<td>3.6–8.5</td>
<td>Rosell (1998)</td>
</tr>
<tr>
<td>Extremadura (Spain)</td>
<td>3</td>
<td>Garzón (1991)</td>
</tr>
<tr>
<td>Castilla-La Mancha (Spain)</td>
<td>1.2–90.9</td>
<td>Acevedo et al. (2007)</td>
</tr>
<tr>
<td>France</td>
<td>1–2.9</td>
<td>Dardaillon (1984), Spitz et al. (1984)</td>
</tr>
<tr>
<td>Italy</td>
<td>1.4–1.7</td>
<td>Marsan et al. (1995)</td>
</tr>
<tr>
<td>Byelorussia</td>
<td>1.8</td>
<td>Okarma (1995)</td>
</tr>
<tr>
<td>Poland</td>
<td>3.5</td>
<td>Jedrzejewski et al. (1997)</td>
</tr>
<tr>
<td>Germany</td>
<td>5.6</td>
<td>Reported by Howells and Edwards-Jones (1997)</td>
</tr>
<tr>
<td>Russia</td>
<td>1.2–1.9</td>
<td>Reported by Howells and Edwards-Jones (1997)</td>
</tr>
<tr>
<td>California (USA)</td>
<td>5.8</td>
<td>Reported by Howells and Edwards-Jones (1997)</td>
</tr>
</tbody>
</table>
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Table 2
Viral pathogen status in domestic pig and wild boar populations

<table>
<thead>
<tr>
<th>Pathogen</th>
<th>Domestic pig status</th>
<th>Wild boar status</th>
<th>Wild boar reservoir rôle for the domestic pig</th>
</tr>
</thead>
<tbody>
<tr>
<td>ADV</td>
<td>Worldwide spread. Eradicated in many Northern European countries and parts of North America. Under eradication in Mediterranean countries.</td>
<td>Widespread. Low prevalences in Central Europe. Medium-high prevalences in Mediterranean countries and feral pig populations in USA</td>
<td>Eradicated in Germany and in a Spanish region on a large scale. Local cases due to contacts caused by inadequate pig restriction methods</td>
</tr>
<tr>
<td>CSFV</td>
<td>Present in many Central and Eastern European countries. Absent in most of Western Europe.</td>
<td>Prevalent in a limited number of areas in different Central and Eastern European countries</td>
<td>Self-limiting infection in some populations and self-maintained infection in others. Persistence of the infection associated with population size, reproductive rate and density</td>
</tr>
<tr>
<td>ASFV</td>
<td>Present in most of Africa</td>
<td>Eradication reported in many American countries and the Iberian peninsula</td>
<td>Reported as self-limiting infection in the absence of infected domestic pigs.</td>
</tr>
<tr>
<td>PCV2</td>
<td>Widespread in domestic pig herds worldwide</td>
<td>Only reported in Europe and Canada, with medium seroprevalences in Belgium and Spain</td>
<td>Unexpected due to high prevalences in domestic pig herds</td>
</tr>
<tr>
<td>PPV</td>
<td>Widespread in domestic pigs with very high prevalences</td>
<td>Medium-high seroprevalences in European wild boars and feral pigs</td>
<td>Unexpected due to high prevalences in domestic pigs</td>
</tr>
<tr>
<td>PRRSV</td>
<td>Worldwide in domestic pig herds</td>
<td>Only serological evidence in wild boars from France and possibly USA</td>
<td>Unexpected</td>
</tr>
<tr>
<td>SIV</td>
<td>Considered a major reservoir of H1N1, H1N2 and H3N2. Can be experimentally infected by high lethal H5N1 virus.</td>
<td>Serological evidence of H1N1 virus in feral pigs and in European wild boars in Spain. Serological evidence of H1N1, H3N2, and H1N2 in European wild boars in Poland</td>
<td>Unknown</td>
</tr>
<tr>
<td>TGEV and PRCV</td>
<td>Widely present in domestic pig herds worldwide; TGEV present worldwide but sporadic</td>
<td>Limited information. TGEV absent in wild boars. Three percent PRCV seroprevalence in Slovenian wild boars</td>
<td>Not expected</td>
</tr>
<tr>
<td>BVDV and BDV</td>
<td>Sporadic and mainly due to contacts or shared habitat with domestic ruminants</td>
<td>BVDV antibodies reported only in France as differential diagnosis with CSFV antibodies</td>
<td>Not expected</td>
</tr>
<tr>
<td>FMDV</td>
<td>Enzootic in most areas of Africa, Asia and South America.</td>
<td>Without evidence</td>
<td>Unexpected</td>
</tr>
<tr>
<td>VSV and VSDV</td>
<td>VSV endemic in North America. VSDV present in European domestic pig herds</td>
<td>Unknown</td>
<td>Unknown</td>
</tr>
<tr>
<td>HEV</td>
<td>Worldwide distribution</td>
<td>Serological and molecular evidence in Japanese wild boars</td>
<td>Unknown. Possible source for humans due to consumption of raw or uncooked wild boar meat</td>
</tr>
<tr>
<td>TTV</td>
<td>Widespread in domestic pigs</td>
<td>Widely present in Spanish wild boar populations</td>
<td>Unknown</td>
</tr>
</tbody>
</table>


4.2. Classical swine fever

Classical swine fever (CSF) is caused by a *Pestivirus* closely related to bovine viral diarrhoea virus (BVDV) and border disease virus (BDV) (Wengler et al., 1995) and is widespread in domestic pigs but in Western Europe and North America (Artois et al., 2002). As a highly contagious disease, CSF is included within the single list of diseases notifiable to the OIE. It is a major disease of pigs and causes high economic losses due to preventive culling of pigs, restrictions in the trade of animals in infected areas and compensation to farmers (Terpstra and de Smit, 2000).

Effects of CSFV in wild boar populations. CSFV circulates among the wild boar populations of Central and Eastern Europe (Moennig et al., 1999) but most of Western Europe is considered CSF-free. In those countries where the wild boar and vice versa is possible, and has been demonstrated in experimental infections (Tozzini et al., 1982; Müller et al., 2001). Contact between infected and susceptible animals can lead to virus transmission but Müller et al. (1997) rejected the rôle of German wild boar as ADV reservoirs for the domestic pig on the basis of molecular differences between virus strains in the two species.

In a recent study carried out in South-central Spain, it was concluded that there was no evidence of ADV interaction between the domestic pig and the wild boar (F. Ruiz-Fons et al., unpublished data). However, in one report it was suggested that the wild boar was responsible for AD outbreaks in outdoor domestic pig herds (Hars and Rossi, 2005), which represent the most serious risk for the transmission of ADV and other pathogens between both suids.
Table 3
Main clinical signs of viral diseases in domestic pigs and wild boars

<table>
<thead>
<tr>
<th>Virus</th>
<th>Clinical signs in domestic pigs</th>
<th>Clinical signs in wild boars</th>
</tr>
</thead>
<tbody>
<tr>
<td>ADV</td>
<td>Dependent on age, infective dose and strain virulence. Nervous (piglets), respiratory (mainly in growing) and reproductive (sows) signs</td>
<td>Only evidence of nervous signs in naturally infected animals. Severe respiratory signs after immunosuppressive treatment of experimentally infected animals</td>
</tr>
<tr>
<td>CSFV</td>
<td>Depending on the clinical course of the infection; more severe in acute than in chronic disease. Anorexia, fever, conjunctivitis, constipation, diarrhoea, hyperaemia of the skin, posterior paresis, purplish discoloration in abdomen, snout, ears and medial sides of the legs, convulsions</td>
<td>Clinical signs similar to the domestic pig. High mortality rates in young wild boars</td>
</tr>
<tr>
<td>ASFV</td>
<td>Severe haemorrhagic disease in all age classes</td>
<td>Clinical course identical to domestic pigs</td>
</tr>
<tr>
<td>PCV2</td>
<td>Cause of PMWS: wasting, unthriftiness, pallor of the skin, respiratory distress, diarrhoea, and occasionally icterus. PCV2 is also implicated in other porcine circovirus diseases</td>
<td>Few reports of PMWS affected farmed and free-living wild boars, with same clinical signs as the domestic pig. Unknown if other PCVD occur in wild boars</td>
</tr>
<tr>
<td>PPV</td>
<td>Reproductive failure in females. Associated with PMWS triggering in some cases</td>
<td>Associated to lower ovulation rate. Expected to be similar to domestic pigs</td>
</tr>
<tr>
<td>PRRSV</td>
<td>Respiratory and reproductive signs. Associated with PMWS triggering in some cases</td>
<td>Unknown</td>
</tr>
<tr>
<td>SIV</td>
<td>Fever, cough, dyspnoea and prostration, generally rapid recovery</td>
<td>Unknown</td>
</tr>
<tr>
<td>TGEV</td>
<td>Transient vomiting, yellowish diarrhoea, weight loss, dehydration</td>
<td>Unknown</td>
</tr>
<tr>
<td>PCV2</td>
<td>Respiratory signs of severity dependent on strain. Severity greater in co-infection with PRRSV</td>
<td>Unknown</td>
</tr>
<tr>
<td>BVDV</td>
<td>Commonly sub-clinical</td>
<td>Unknown</td>
</tr>
<tr>
<td>BDV</td>
<td></td>
<td></td>
</tr>
<tr>
<td>FMD, VSV, and VSDV</td>
<td>Clinical signs cannot be distinguished between FMD, VSV and VSDV. They consist of fever, formation of vesicles and erosions on snout, lips, tongue, hard and soft palate and coronary band of the feet</td>
<td>Unknown</td>
</tr>
<tr>
<td>HEV</td>
<td>No clinical signs reported. Slight hepatic inflammation only seen histopathologically</td>
<td>Unknown</td>
</tr>
<tr>
<td>TTV</td>
<td>Currently considered to be non-pathogenic</td>
<td>Unknown</td>
</tr>
</tbody>
</table>


the disease has been described, it is prevalent in only a limited number of areas (Artois et al., 2002).

Susceptibility, clinical manifestation and lesions of CSF are similar in both the wild boar and in domestic pigs following experimental infection (Brugh et al., 1964; Aubert et al., 1994; Depner et al., 1995). Mortality rates vary with the clinical course of the disease, with higher values reported in acute rather than in sub-acute and chronic cases. High mortality rates are frequently observed in piglets both in domestic pigs and wild boars (Kern et al., 1999), especially at the onset of an outbreak.

Excretion of the virus by different routes, such as saliva, nasal and lachrymal secretions (Aubert et al., 1994), could lead to horizontal transmission via direct contact. CSF virus may survive for a considerable time in protein-rich environments (Edgar et al., 1952; Helwig and Keast, 1966), possibly leading to indirect transmission through carcass consumption. Transplacental transmission can lead to persistently infected animals (late-onset CSF) with no immune reaction against the virus (Meyer et al., 1980; Depner et al., 1995). Late onset infection caused death in a wild boar piglet in 39 days (Depner et al., 1995), although environmental conditions to which wild boar are subjected suggest that the expected half-life for persistently infected wild boar piglets should be shorter than this. High mortality rates in young animals after an outbreak can lead to changes in the population dynamics of wild boars.

Wild boar as CSF virus reservoir for the domestic pig. Outbreaks are generally self-limiting in most wild boar populations (Ferrari et al., 1998; Fritzmeier et al., 1998; Rossi et al., 2005) but, in other cases, CSF virus circulates for years (Laddomada et al., 1994; Kern et al., 1999). The role of the wild boar as a CSF virus reservoir and possible source of infection for the domestic pig is well known. Moreover, epidemiological links between CSF virus infections in wild boars and domestic pigs have been repeatedly reported, mainly in Germany (Wachendorfer et al., 1978; Krassnig and Schuller, 1993; Laddomada et al., 1994; Teuffert et al., 1997).

Aubert et al. (1994) proposed three reasons why wild boars should not be considered as CSF virus reservoirs and a risk to the domestic pig. Firstly, when the domestic pig and wild boar coexist and CSF is then eradicated from the domestic pig population, the disease is not maintained in the wild boar. Secondly, when CSF virus has been intentionally introduced in feral pig populations, the disease is not self-maintained. Thirdly, when information about the origin of a CSF outbreak in wild boars has been correctly
collected, human interference was apparent. Notwithstanding this thesis, all CSF virus strains isolated from wild boars in Germany in the 1990s were also isolated in domestic pigs from the same locations. Moreover, 92% of the primary outbreaks in domestic pigs were located in regions where CSF was endemic among the wild boar populations, and it was considered that 60% of the outbreaks were due to direct or indirect contacts with wild boars (Moennig et al., 1999). Similar observations have also been reported in Italy (Rutili, 1997; Ferrari et al., 1998).

The role of wild boar density in the persistence of CSF virus among wild populations after the onset of an epizootic outbreak may have an influence together with age structure and the size of the affected population (Artois et al., 2002). CSF could persist in dense wild boar populations where there are no barrier restrictions (such as highways), due to a high recruitment rate and an increased availability of young animals. This would impede control and eradication schemes due to the increased risk of transmission from wild boar to domestic pigs.

4.3. African swine fever (ASF)

African swine fever virus (ASFV) is able to infect both domestic and wild suids (Sánchez-Vizcaíno, 2006) and can also replicate in soft ticks of the genus Ornithodoros (Sanchez Botija, 1963; Plowright et al., 1970; Mellor and Wilkinson, 1985). ASFV first appeared in domestic pigs in Kenya in 1921 as a consequence of transmission from wild African suids (Sánchez-Vizcaíno, 2006). The infection spread to the Iberian Peninsula, first in Portugal (Manso et al., 1998) and later into Spain (Polo Jover and Sanchez Botija, 1961), and since then it has appeared in several South and Central American countries and Sardinia. ASF is included within the single list of diseases notifiable to the OIE. High morbidity and mortality is reported during ASF outbreaks in domestic swine, leading to economic losses not only due to high animal mortality but also to the restrictions in trade of animals and their products.

Effects of ASFV on wild boar populations. After the appearance of ASFV in the Iberian Peninsula, the virus appears to have spread from domestic pigs to the European wild boar and evidence of ASFV infection in wild boars has been reported in Spain (Ordas et al., 1981; Pérez et al., 1998), Portugal (Da Cruz Braço-Forte, 1980) and Sardinia (Firinu and Scarano, 1988; Laddomada et al., 1993). During the first reported ASF outbreaks, clinical signs of the purpura and acute course of the disease were observed in wild boars (including fever, lethargy, purplish discoloration of the skin of ventral areas and sudden death). Later, the clinical course of the disease became subacute (Pérez et al., 1998).

Reports of experimental and natural infection in European wild boars and feral pigs agree that gross and microscopic lesions were identical to those seen in the domestic pig (Ravaioli et al., 1967; McVicar et al., 1981). Thus, the onset of an ASF epizootic outbreak in wild boars would have a great impact as a consequence of high mortality.

ASFV is able to persist for at least 1 year in Ornithodoros spp. ticks (Endris et al., 1987; Hess et al., 1989). Nevertheless, Hess et al. (1989) concluded that mortality is higher in ASFV infected ticks than in non-infected ones, and suggested that this could contribute to ASFV clearance from tick populations that are not subjected to reinfection. To our knowledge, there are no reports of O. erraticus parasitizing wild boars. Moreover, soft ticks do not stay attached to their hosts for a long time, and so are rarely detected in post-mortem inspections of hunter-harvested animals.

Wild boar as an ASFV reservoir for the domestic pig. No seropositive wild boars have been reported in areas where the domestic pig is free of the disease (Firinu and Scarano, 1988; Pérez et al., 1998) or, when reported, the virus circulated at very low levels among the wild population (Laddomada et al., 1993). Indeed, Laddomada et al. (1994) suggested that the virus is unable to persist in wild boar populations without contact with infected domestic pigs.

4.4. Porcine circovirus diseases

Porcine circoviruses (PCV) are small viruses of the family Circoviridae. Two PCV genotypes have been described: porcine circovirus type 1 (PCV1), which is considered non-pathogenic for swine, and porcine circovirus type 2 (PCV2), which was firstly isolated from pigs in Canada in association with a novel disease called postweaning multisystemic wasting syndrome (PMWS) (Harding, 1996).

PCV2 infection in domestic pigs has been further linked to other diseases or conditions which are now included under the term porcine circovirus diseases (PCVD). Of these, PMWS is considered the most significant due to its high economic impact on the pig industry (Segalés et al., 2005). PMWS has been experimentally reproduced with only PCV2 virus in the inoculum (Bolin et al., 2001), although only in a small number of experiments. PMWS is currently considered to be a multifactorial disease in which PCV2 in necessary but is not sufficient to trigger the clinical outcome (Segalés et al., 2005).

Effects of PCV2 on wild boar populations. PCV2 seroprevalence in Belgian and Spanish wild boars has been reported to be around 30–40% (Sánchez et al., 2001; Vicente et al., 2004). PCV2 infection has been detected in about 20% of Hungarian wild boars using polymerase chain reaction (PCR) (Cságora et al., 2006). These reports indicate that PCV2 circulates at a high rate among the wild boar populations in Europe.

PMWS typically affects nursery and fattening domestic pigs (2–4 months of age) causing wasting, pallor of the skin, unthriftiness, respiratory distress, diarrhoea and sometimes icterus (Segalés and Domingo, 2002). Although PMWS reports in wild boars are scarce, they have been described in North America and Europe (Ellis et al., 2003; Schulze et al., 2003; Vicente et al., 2004). In all clinical cases, the affected animals’ ages ranged from 4 to 10
months old, except for 6-week-old, farm-bred feral pigs from Canada. Clinical symptoms (when observed) and the gross and microscopic lesions resembled those reported for the domestic pig. Clinical reports of PMWS in wild boars also refer to increased piglet mortality either within the herd or the hunting estate where the clinical cases were found (VLA, 2003; Vicente et al., 2004).

Multiple infections are common in free-living wild boars (Ruiz-Fons et al., 2006b), and under immunosuppressed conditions the risk of disease development could be higher. If we take into account the fact that other pathogens are also widely present in European wild boar populations, PCV2 and its most significant associated disease, PMWS, could have a significant role in affecting mortality rates in association with other concurrent infections.

Wild boar as PCV2 reservoir for the domestic pig. At the present time it is premature to assign a role to the wild boar as a PCV2 reservoir for the domestic pig. PCV2 isolates from wild boars have been found to be identical to those from domestic pigs in the same or in distant regions (Knell et al., 2005; Csálgóta et al., 2006). It is however likely that the origin of PCV2 infection in wild boar populations could be through contacts with domestic pigs, not least because of the high PCV2 infection rates (close to 100%) in pig herds. Nevertheless, there is no current knowledge about the direction of transmission from one species to the other.

4.5. Porcine parvovirus

Porcine parvovirus (PPV) is classified in the genus Parvovirus and is distributed worldwide in the domestic pig (Mengeling, 2006). All PPV isolates present similar if not identical antigenic characteristics (Johnson et al., 1976; Ruckerbauer et al., 1978). PPV has only been associated with reproductive failure in females, while acute infection of post-natal pigs is usually subclinical (reviewed by Mengeling, 2006). Thus, the major and (usually) only clinical effect of PPV infection is reproductive failure.

Effects of PPV on wild boar populations. PPV is widely distributed in European wild boar and feral pig populations, with seroprevalences ranging from 14% to 77% (Liebermann et al., 1986; Payeur et al., 1989; New et al., 1994; Lutz and Wurm, 1996; Saliki et al., 1998; Gipson et al., 1999; Roic et al., 2005; Ruiz-Fons et al., 2006b; Vengust et al., 2006). Despite serological evidence of PPV antibodies in wild boar populations, no direct effects of PPV have been described although PPV seroprevalence in female wild boars has a negative influence on ovulation rate (Ruiz-Fons et al., 2006b). The effect of PPV on the first stages of the oocyte is unknown, but based on these results there could be a direct effect on ovulation rate.

Reproductive failure depends on the timing of the infection during gestation. When transplacental infection occurs during the second half of gestation (>70 days), fetuses are able to develop an immunological response and will survive in utero (Redman et al., 1974; Bachmann et al., 1975). Reproductive failure could occur in wild boar gilts that are pregnant for the first time without previous contact with the virus. PPV infection before mid-gestation could then lead to reproductive failure. Although little information is available regarding the direct effects of PPV on female wild boars, the high seroprevalence rates found suggest that reproductive performance could be partially restricted by PPV.

Wild boar as PPV reservoir for the domestic pig. As PPV seroprevalences are higher in domestic pig herds than in European wild boar populations (with some exceptions, see Lutz and Wurm, 1996), it is unlikely that the wild boar could act as a PPV reservoir for domestic pigs. Nevertheless, transmission between both could take place in both directions if contact between pigs and wild boars occurs.

4.6. Porcine reproductive and respiratory syndrome (PRRS)

PRRS virus (PRRSV) is an Arterivirus closely related to lactate dehydrogenase-elevating virus (LDV) of mice and other viruses of the family Arteriviridae. Two different PRRSV genotypes are recognized at the present time, one from North America and the other from Europe, and these show approximately 60% nucleotide homology (Nelsen et al., 1999). The origin of PRRSV is unclear, although it has recently been suggested that it may be LDV infected wild boar in central Europe (Plagemann, 2003). Together with PPV, PRRSV is currently considered to be one of the most common viral causes of reproductive failure in domestic pigs (Mengeling et al., 2000).

Effects of PRRSV on wild boar populations. There are limited scientific data regarding PRRSV seroprevalence in European wild boar and feral pig populations. Only 1.7% of feral pig sera tested positive in Oklahoma State, USA (Saliki et al., 1998), and 1.3% and 8.3% of free-living and farmed wild boar, respectively, in France (Albina et al., 2000). Other studies have yielded negative results (Oslage et al., 1994; Lutz and Wurm, 1996; Gipson et al., 1999; Vicente et al., 2002; Ruiz-Fons et al., 2006b; Vengust et al., 2006).

PRRSV causes a marked increase in return to oestrus, late-term abortions, stillborn and weak piglets. In many cases, severe respiratory disease in suckling and weaned pigs also occurs (see review by Zimmerman et al., 2006). No clinical cases of PRRS have been described in wild boars, for which clinical symptoms, if any, remain unknown. We could speculate that respiratory and reproductive disorders occur as in the domestic pig, but the apparent low circulating rates of the virus among free-living wild boars suggest no significant influence of PRRSV in this species.

Wild boar as PRRSV reservoir for the domestic pig. PRRSV transmission would be favoured within dense wild boar populations, but the lack of infection in many of these animal groups suggest that the initial transmission from domestic swine to wild boar does not occur, or occurs very sporadically. Currently, the transmission of PRRSV from
domestic swine to wild boar is more probable than vice versa. Current knowledge offers no evidence that the wild boar is a PRRSV reservoir.

5. Other viral infections in wild boar populations

5.1. Swine influenza

Swine influenza is caused by type A influenza viruses (Olsen et al., 2006). The domestic pig is considered to be a major reservoir of H1N1 and H3N2 influenza viruses (Brown, 2000) and clinical manifestations include fever, cough, dyspnoea and prostration, usually followed by a rapid recovery.

Swine influenza virus (SIV) serological data are available from European wild boar and feral pig populations. Antibodies to three subtypes of SIV, H1N1, H3N2 and H1N2, have been detected in wild boar populations, although in variable and, generally, low concentrations (Markowska-Daniel and Pejsak, 1999; Markowska-Daniel, 2003; Markowska-Daniel and Kowalczyk, 2005). Seroprevalence may vary from 0% to 75% depending on the country or region and SIV subtype (Saliki et al., 1998; Gipson et al., 1999; Vicente et al., 2002; Vengust et al., 2006). The H1N1 subtype seems to be the most prevalent among wild boar (Gipson et al., 1999).

Transmission of SIV is mainly by the oronasal route in domestic pig herds due to direct contact between infected and susceptible animals and via aerosol. As close contact among wild boars is density-dependent, the transmission of SIV in low-density wild boar populations will lead to the extinction of the pathogen or to very low circulating rates of the virus. However, in semi-captive or farmed and dense wild boar populations, SIV could become endemic. Moreover, the role of the wild boar in relation to the highly pathogenic avian H5N1 influenza virus should be considered.

5.2. Infection by coronaviruses

Transmissible gastroenteritis virus (TGEV) and porcine respiratory coronavirus (PRCV) are responsible for gastrointestinal and respiratory clinical signs, respectively, in domestic pigs (Saif and Sestak, 2006). TGE has been described in most countries worldwide, but its importance has decreased with time since PRCV will immunize pigs against TGEV infection (Saif and Sestak, 2006) and, as PRCV is enzootic in European domestic pigs, this has led to a significant decrease in the economic impact of TGE (Pensaert and Cox, 1989; Laude et al., 1993).

Little information is available regarding coronavirus infections in European wild boars and feral pigs. Feral pigs in the USA did not show antibodies against TGEV (Woods et al., 1990; Saliki et al., 1998) and similar findings were reported from Slovenian wild boars (Vengust et al., 2006), although 3% of the animals tested had anti-PRCV antibodies. Although more information is needed, the available data suggest that coronavirus infections are not common among wild boar populations and these are not therefore likely to be a reservoir for domestic swine.

5.3. Infection by other pestiviruses

Bovine viral diarrhoea virus (BVDV) and border disease virus (BDV) infect a wide variety of domestic and wild ungulates (Nettleton, 1990; Depner et al., 1991; Vannier and Albina, 1999). Both agents are classified in the genus Pestivirus together with CSFV.

Serological evidence of both infections has been reported in wild boar populations (Dahle et al., 1993; Albina et al., 2000). New et al. (1994) did not find BVDV antibodies in feral pigs in the USA. In the light of the available data, the wild boar is not expected to be identified as a BVDV or BDV reservoir but serological cross-reactions with CSFV false positives must be taken into account when surveys are carried out.

5.4. Infection by picornaviruses

Foot-and-mouth disease (FMD) affects wild and domestic ungulates (Thomson et al., 2001). It is a highly contagious disease with a high impact on the trade of animals and their products and it is included within the single list of diseases notifiable to the OIE. FMD has been widely reported in domestic animals in Europe and currently persists in the northern part of South America, most African countries, the Middle East, and some countries of Eastern Europe and in Asia (Thomson et al., 2001). In contrast, vesicular stomatitis virus (VSV) is endemic on the American continent (Lubroth et al., 2006). Both VSV and swine vesicular disease virus (SVDV) are important as differential diagnoses with FMDV infection due to similar clinical signs (Lubroth et al., 2006).

The domestic pig and also the wild boar are natural hosts for these picornaviruses. Information is scarce in relation to the wild boar but Pech and Hone (1988) considered the possible role of feral pigs to be highly important should FMD enter Australia.

5.5. Hepatitis E

Hepatitis E virus (HEV) is a RNA virus belonging to the Hepeviridae family (Emerson et al., 2004). Hepatitis E is an important disease of public health concern due to its zoonotic character. HEV has been widely reported to infect domestic swine herds around the world (Clayson et al., 1995; Meng et al., 1997, 1999; Chandler et al., 1999; Hsieh et al., 1999; Pina et al., 2000). HEV transmission from wild animals to humans has been reported due to the consumption of raw or under-cooked deer or wild boar meat (Matsuda et al., 2003; Tei et al., 2003; Li et al., 2005).

HEV has been found in wild boar both by serology and molecular analyses (Takahashi et al., 2004; Nakamura et al., 2006). The scarcity of knowledge of the sanitary sta-
tus of wild boar populations in regard to HEV outside Japan makes it impossible to establish either the impact of the disease among wild boar or the possible role of the wild boar as a putative HEV reservoir for the domestic pig. However, the relative widespread HEV infection in domestic pigs in Europe (Banks et al., 2004; De Deus et al., 2007) suggests that to find HEV in European wild boar populations would not be surprising. Indeed there is recent information that HEV may already have been found (N. De Deus, personal communication).

5.6. Infection by torque teno viruses

Torque teno virus (TTV) was first isolated from a human patient (Nishizawa et al., 1997) and later from domestic animals (Leary et al., 1999; Okamoto et al., 2002), although the virus is considered species-specific. The virus has been reported to be present in almost 100% of domestic pig herds in many different countries (McKeown et al., 2004). Recently, two different genogroups of swine TTV have been identified (Niel et al., 2005), and both are highly prevalent in domestic swine (Kekarainen et al., 2006). TTV is considered non-pathogenic for all species where it has been found, but swine TTV genotype 2 has been found more prevalent in PMWS affected pigs than in non-affected pigs (Kekarainen et al., 2006).

Only one survey is available on the presence of TTV in wild boars, and one or the other swine TTV genotypes were found in 84% of the tested animals (Martínez et al., 2007). This report found differences regarding management, age, sex and TTV genogroup, but their significance remains to be assessed.

6. Discussion

Viral disease poses a threat for production efficiency in industrialized pig producing countries as well as having a great impact in those developing countries where pig meat is an important food resource. Viruses infecting domestic pigs are also able to infect wild boars. These facts, together with the increasing economic relevance of wild boars for the emerging hunting industry, have led us to publish this review.

Diseases affect wildlife species in a similar manner to domestic animals. Many wildlife species become infected with a pathogen that can infect domestic animals or humans and so becomes a reservoir. Three points have to be taken into account in order to consider an animal species as a reservoir: (1) there must be a lot of them; (2) the pathogen must be able to infect the animal, and (3) the agent must be transmissible to other animals (Wobeser, 1994; Corner, 2006). On this basis, the wild boar could act as reservoir of viruses for the domestic pig, as has been shown in the case of CSFV in Central Europe.

Some viral diseases can have an immense impact on wild boar population dynamics, especially those with high mortality rates. Other diseases are more subtle, modelling survival or reproductive rates. To date, there is simply not enough information about the impact of viral and other infectious diseases on wild boar dynamics and much opinion must therefore be speculative. There is also a lack of information on pathogenesis, clinical manifestation, epidemiology and prevention and control methods of viral diseases in wild boar. Although both the domestic pig and the wild boar are considered as the same species and basic features of the viral infection could be identical, risk factors widely differ between domestic and wild species. Knowledge in this latter field should be improved.

Several characteristics make the wild boar a very interesting species for epidemiological research on wildlife diseases: (1) it has a worldwide distribution; (2) it shares common infectious and parasitic agents with the domestic pig; (3) it has a great ability to adapt to different environments and to colonize new habitats; (4) it has a fast reproductive rate and is able to recover from population declines; (5) it has a complex social behaviour, and (6) it adapts well to captivity and so offers good possibilities for controlled experimental research.

The wild boar can maintain some viral pathogens without the intervention of domestic or other wild animals and is a true reservoir of several viral pathogens that affect the domestic pig. Avoiding close contact between wild boars and domestic animals is therefore of logical importance in disease control and eradication programmes. Artificial management of wild boar populations, such as by the use of controlled fencing and feeding, causes increased density and spatial aggregation and an increased risk of disease transmission, and natural management techniques and systems are therefore to be preferred.

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