Wild boars as sources for infectious diseases in livestock and humans

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Wild boars (Sus scrofa) are indigenous in many countries in the world. These free-living swine are known reservoirs for a number of viruses, bacteria and parasites that are transmissible to domestic animals and humans. Changes of human habitation to suburban areas, increased use of lands for agricultural purposes, increased hunting activities and consumption of wild boar meat have increased the chances of exposure of wild boars to domestic animals and humans. Wild boars can act as reservoirs for many important infectious diseases in domestic animals, such as classical swine fever, brucellosis and trichinellosis, and in humans, diseases such as hepatitis E, tuberculosis, leptospirosis and trichinellosis. For examples, wild boars are reservoirs for hepatitis E virus, and cluster cases of hepatitis E have been reported in Japan of humans who consumed wild boar meat. In Canada, an outbreak of trichinellosis was linked to the consumption of wild boar meat. The incidence of tuberculosis owing to Mycobacterium bovis has increased in wild boars, thus posing a potential concern for infections in livestock and humans. It has also been documented that six hunters contracted Brucella suis infections from wild swine in Florida. This article discusses the prevalence and risk of infectious agents in wild boars and their potential transmission to livestock and humans.

Keywords: wild boar; swine; zoonosis; hepatitis E virus; trichinellosis; tuberculosis

1. INTRODUCTION

Wild boars (Sus scrofa) are indigenous in many countries in the world. These free-living swine populations pose not only ecological concerns but infectious disease concerns as well. Wild boars harbour many important infectious agents that are transmissible to domestic pigs and other animal species including humans. Changes of human habitation to suburban areas owing to growing world populations, increased use of lands for agricultural purposes, and deforestation have all increased chances of contact exposure of wild boars to humans and domestic animals. In addition, recreational hunting of wild boars and consumption of wild boar meats in some regions of the world further provided ample opportunities for direct human contacts with wild boars, and thus created an ideal environment for the transmission of pathogens between wild boars and domestic swine, and between wild boars and humans (Gibbs 1997). In some regions, the wild boar populations are on the rise in part owing to the development of a commercial hunting industry (Acevedo et al. 2006), and this could further complicate the problem.

Wild boars also pose a problem when countries are trying to eradicate zoonotic diseases such as hepatitis E, tuberculosis, brucellosis and trichinellosis in humans and important livestock infectious diseases such as pseudorabies and porcine circovirus-associated diseases (PCVADs) in pigs. This is especially relevant for disease-free populations of animals and humans not exposed previously to the agents from wild boars and with no herd or population immunity, and thus highly susceptible to such infectious agents.

This review summarizes the potential risks posed by wild boars as sources of livestock (especially domestic swine) infections and as reservoirs for zoonoses. Specific examples of viral (hepatitis E), bacterial (tuberculosis) and parasitic (trichinellosis) zoonoses transmissible from wild boars to humans are also discussed.

2. VIRUSES IN WILD BOARS THAT ARE POTENTIALLY TRANSMISSIBLE TO LIVESTOCK AND HUMANS

Wild boars are reservoirs for a number of viruses that cause important diseases in livestock and humans (Gibbs 1997; Ruiz-Fons et al. 2008). Table 1 lists the viruses that are known to be prevalent in wild boars with potential for transmission to domestic animals and humans.

(a) Wild boars as reservoirs for livestock infections

Among the agriculturally important pathogens known to be prevalent in wild boars are classical swine fever
virus (CSFV), pseudorabies virus (PRV) infection, African swine fever virus (ASFV), porcine circovirus type 2 (PCV2), porcine reproductive and respiratory syndrome virus (PRRSV) and porcine parvovirus (PPV) (table 1).

CSFV, a small single-strand positive-sense RNA virus, belongs to the genus *Pestivirus* in the family *Flaviviridae*. CSFV infects both domestic swine and wild boars and can be transmitted from wild boars to domestic pigs and vice versa (Brugh *et al.* 1964). CSFV is highly contagious and swine are the only known reservoir. CSFV is transmitted primarily via the oronasal route by direct or indirect contact with infected domestic pigs or wild boars or via the oral route by ingestion of contaminated foodstuffs (Edwards *et al.* 2000). The clinical signs of disease associated with CSFV infection vary greatly with acute and/or chronic forms of the disease (Le Potier *et al.* 2006).

Several countries, including the USA, Australia, Canada and New Zealand are free of CSFV in domestic pig populations (Paton & Greiser-Wilke 2003). CSFV is circulating in some European countries,
with prevalence rates ranging from less than 1 per cent in France (Albina et al. 2000), 31 per cent in Switzerland (Schnyder et al. 2002) and to up to 39 per cent in Croatia (Zupancic et al. 2002). CSFV infection reported in recent outbreaks in wild boars appeared to be subclinical with little mortality (Le Potier et al. 2006), although wild boars can serve as reservoirs for infection of domestic pigs. In some European countries, CSFV has been reintroduced periodically into domestic pigs via contact with infected wild boars (Le Potier et al. 2006).

PRV, an alphaherpesvirus, has a worldwide distribution and infects both wild boars and domestic swine (Ruiz-Fons et al. 2007, 2008). PRV infections in adult feral swine generally do not cause morbidity or mortality (Romero et al. 2001) but can result in latent infections. An important feature of PRV infection in wild or domestic swine is the establishment of latency in neuronal and non-neuronal cells, which has important implications for the spread of PRV. Exposure to PRV is high among wild boars and the seroprevalence rate ranges from 9 to 61 per cent in wild boars from different countries (table 1). The seroprevalence result was confirmed by the detection of PRV DNA in tissues of wild boars (Ruiz-Fons et al. 2007). The high prevalence of PRV in wild boars poses a concern for the health of both wild boars and other domestic animals and prospects for PRV control and eradication campaigns in swine. In the USA, a national pseudorabies eradication programme began in 1989 and, currently, domestic swine in the majority of states are free of PRV. However, the virus is well established in wild boar populations in the USA and other countries, and wild boars represent a potential reservoir of PRV for infection of domestic swine and native wildlife.

ASFV, an OIE (World Organization for Animal Health) List A virus, is a DNA virus in the family Asfarviridae. ASFV is highly resistant to inactivation by temperature and acidic pH and can persist in frozen or uncooked meats for weeks or months (Sanchez-Vizcaino 2006). ASFV is endemic in many African countries and also in Sardinia, Italy (Sanchez-Vizcaino 2006). In addition to domestic pigs, ASFV also infects European wild boars and African wild suids such as warthogs, bush pigs and giant forest hog (Sanchez-Vizcaino 2006). Serological evidence of ASFV prevalence in wild boars has been reported in Italy and Spain where approximately 10 per cent of the wild boars tested (Pérez et al. 1998) had antibodies to ASFV but did not show clinical signs. European wild boars are not believed to play an important role as reservoirs for ASFV.

PCV2, the causative agent of PCVAD is a small single-stranded DNA virus with a circular genome (Ramamoorthy & Meng 2008). PCVAD is characterized by wasting, enlarged lymph nodes, jaundice and weight loss in affected weanling pigs, and lymphoid depletion is the hallmark microscopic lesion. Recently, several other complex syndromes, including reproductive failure, enteritis, pneumonia and necrotizing dermatitis, have also been linked to PCV2 infection. PCVAD is further complicated by co-infections with other bacterial and viral pathogens (Ramamoorthy & Meng 2008).

Evidence of PCV2 infection in wild boars has been reported in Hungary, Spain and Belgium (table 1). Approximately 50 per cent of wild boars tested in Spain were seropositive for PCV2 antibodies and approximately 21 per cent of wild boars from Hungary were positive for PCV2 DNA (Cságola et al. 2006). PCVAD was reported in Eurasian wild boars raised under free-range conditions (Vicente et al. 2004). Affected wild boars had pneumonia and enteritis and were cachectic. These data indicate that there is a high rate of PCV2 infection in wild boars, although the exact role of wild boars in PCV2 transmission to domestic pigs is not clear.

PRRSV, a single-strand positive-sense RNA virus in the family Arteriviridae, causes arguably the most economically significant disease facing the global swine industry today (Meng 2000a). Nursery pigs infected by PRRSV develop pneumonia and other respiratory diseases, whereas infected sows develop reproductive failures. PRRSV infection can subvert the pig immune system and cause persistent infection, which is one of the major obstacles for PRRSV control. The interactions between PRRSV and other swine pathogens produce clinically and economically significant disease syndromes, such as porcine respiratory disease complex. The role of wild boar in the transmission of PRRSV to domestic pigs is unclear. Antibodies to PRRSV have been detected in wild boar in some countries (table 1), although others failed to detect PRRSV antibodies in wild boars. There is lack of convincing evidence to suggest that wild boars serve as a reservoir for PRRSV.

PPV, a small DNA virus in the family Parvoviridae, is ubiquitous among swine throughout the world (Mengeling 2006). PPV is associated mainly with reproductive problems in gilts, sows and boars and causes only subclinical infections in other pigs. Large proportions of gilts are naturally infected with PPV before they conceive and develop an active immunity that probably lasts throughout their life.

PPV is highly prevalent in wild boars, with an incidence ranging from 14 to 17 per cent in Oklahoma and Tennessee, USA, to 57 per cent in Spain (table 1). The effect of PPV infection on the health status of wild boars appears to be minimal and subclinical, although it has been reported that PPV infection has a negative effect on the ovulation rate of female wild boars (Ruiz-Fons et al. 2006). It is possible that PPV is transmissible between wild boars and domestic pigs through direct contacts, although wild boar is not considered a significant reservoir for PPV transmission to domestic pigs (Ruiz-Fons et al. 2008).

(b) Wild boars as reservoirs for human infections

Antibodies to a number of zoonotic viruses have been detected in wild boar populations, including hepatitis E virus (HEV), swine influenza virus and Japanese encephalitis virus (table 1). Wild boars infected by these zoonotic viruses have the potential to transmit to humans in close contact. Direct evidence of boar to human transmission has been documented for HEV.

HEV, a single-strand positive-sense RNA virus, belongs to the family Hepeviridae. The disease
caused by HEV, hepatitis E, is an important public health disease in many developing countries, even though the disease is also endemic in many industrialized countries (Meng et al. 2000b, 2003; Meng & Halbur 2006). HEV is transmitted primarily through the faecal–oral route, and contaminated water or water supplies are the main sources of infection. The mortality caused by HEV is generally low (less than 1%), but 28 per cent was reported in infected pregnant women. There are at least four distinct genotypes of HEV worldwide. Genotypes 1 and 2 are associated with epemics, whereas genotypes 3 and 4 cause sporadic cases of hepatitis E (Meng & Halbur 2006).

The discovery of swine HEV from pigs in the USA (Meng et al. 1997) lends credence to the zoonosis concept for HEV. Thus far, the viruses identified from pigs worldwide belong to either genotype 3 or 4, which both cause sporadic cases of acute hepatitis E (Meng & Halbur 2006). Genetic and phylogenetic analyses of the complete genomic sequences of swine HEV revealed that swine HEV is very closely related, or identical in some cases, to genotypes 3 and 4 of human HEV. Seroepidemiological studies demonstrated that swine HEV is ubiquitous in pigs in the midwestern USA (Meng et al. 1997). Similar findings were also reported in many other developing and industrialized countries, indicating that swine HEV infection in pigs is common worldwide (Meng 2003). The ubiquitous nature of swine HEV infection in pigs provides a source of virus for zoonotic human infections.

Swine HEV has been shown to cross species barriers and infect both rhesus monkey and chimpanzees (Meng et al. 1998). Conversely, genotypes 3 and 4 of human HEV have been shown to infect pigs under experimental conditions (Feagins et al. 2008). However, attempts to experimentally infect pigs with genotypes 1 and 2 of human HEV were unsuccessful. Hepatitis E is now considered as a zoonotic disease and pigs (and possibly other animal species) are reservoirs. Pig handlers, such as pig farmers and swine veterinarians, in both developing and industrialized countries are at increased risk of HEV infection. For example, swine veterinarians in the USA were 1.51 per cent positive for swine HEV antibodies than age- and geography-matched normal blood donors (Meng et al. 2002).

Sporadic and cluster cases of acute hepatitis E have been linked to the consumption of raw or undercooked pig livers. Approximately 2 per cent of pig livers sold in local grocery stores in Japan (Yazaki et al. 2003) and 11 per cent in the USA were tested positive for swine HEV RNA (Feagins et al. 2007). Contaminating virus in commercial pig livers sold in the grocery stores of the USA remains fully infectious. Most significantly, the genome sequences of viruses recovered from pig livers in grocery stores were closely related, or identical in a few cases, to the viruses recovered from human hepatitis E patients in Japan (Yazaki et al. 2003).

Wild boar populations in Japan, Germany, Italy, Spain and Australia are commonly infected by HEV, as demonstrated by the detection of HEV RNA and anti-HEV antibodies (table 1). A full-length genomic sequence of HEV from a boar was found to be 99.7 per cent identical to the virus from a wild deer hunted in the same forest and to four patients who consumed deer meat and contracted hepatitis E (Takahashi et al. 2004), suggesting a potential interspecies HEV transmission between boar and deer in wildlife.

In Japan, a 53-year-old man developed severe hepatitis E after consumption of wild boar meat (Matsuda et al. 2003). Another patient, a 70-year-old man, who also ate the same wild boar meat developed hepatic coma and died of fulminant hepatic failure (Matsuda et al. 2003). In the three months prior to the onset of disease, neither patient had travelled to an HEV endemic area, but both patients ate uncooked wild boar liver on five occasions. This correlation provided strong direct evidence of HEV transmission from infected wild boar to humans.

3. BACTERIA IN WILD BOARS THAT ARE POTENTIALLY TRANSMISSIBLE TO LIVESTOCK AND HUMANS

It has been clearly shown that wild boar can act as reservoirs for a long list of zoonotic bacterial agents. Infected boars have the potential to transmit such agents to livestock and to humans, who may come in contact with their discharges or consume infected meat. Major zoonotic bacterial agents that have been detected from wild boars by isolation and/or by specific antibodies are listed in table 2. Among the major bacterial agents of agricultural importance isolated from wild boars include Mycobacterium bovis (bTB), Brucella suis (BS), Brucella melitensis (BM), Brucella abortus (BA), Coxiella burnetii (Q fever), Yersinia pestis (plague) and Leptospira interrogans (Lepto). In this article, only those major zoonotic bacterial diseases transmissible to livestock and/or humans are discussed.

(a) Tuberculosis

Mycobacterium bovis (bTB), the causative agent of bovine tuberculosis, has the broadest host range compared with any other member of the genus Mycobacterium (Mycobacterium tuberculosis complex). Mycobacterium bovis infects cattle, other ruminants, humans and wildlife. The bacterium is transmitted mainly by aerosol or by consumption of infected meat, milk and their products. It typically causes a persistent infection with production of granulomas of the lymph nodes, lung, and may become systemic affecting most internal organs. It was originally thought that wild boars (wildlife) were the dead-end hosts for M. bovis.

During the last two decades, the role of wild boars as a dead-end host, spillover host, maintenance host and true wildlife reservoir of bTB has been extensively debated. Serraino et al. (1999) argued that wild boars were dead-end hosts, as mainly localized lesions were found in the lymph nodes of bTB-affected wild boars. They did not develop generalized lesions in the respiratory tract, which gave credence to the argument, although molecular typing showed that the same spoligotype affected both livestock and wild boar in Italy. Several recent publications from Spain demonstrate that intensively raised wild boars within the
confines of fenced areas of wilderness can be a major source of bTB (Parra et al. 2006; Vicente et al. 2006). In the Mediterranean region of Spain, wild boar raised semiwild (with feeding and watering within fenced areas, so the pigs could be used as game animals by hunters) have created a unique epidemiological niche that has led to major disease outbreaks owing to \textit{M. bovis} in the wildlife with potential exposure to domestic animals. The numbers of bTB-positive wild boar in Europe were close to 50 per cent (table 2), which is alarming. There is a clear indication that wild boar could be the source of bTB infection for domestic animals and humans, especially hunters and veterinarians. Zanella et al. (2008) reported that an outbreak of tuberculosis in wildlife in France, especially in heavily infected wild boars, was due to the same strain of \textit{M. bovis} isolated from other wildlife and domestic cattle.

When bTB in domestic animals is controlled or eradicated, the incidence of infection in wildlife also decreases, suggesting that domestic animals could be the initial source of bTB infection to wild boars. If the populations of wildlife remain small and do not come in contact with domestic animals, they may act as maintenance hosts. However, when greater numbers of wild boars are raised in confined areas, they may suffer from severe bTB infections and become the source of infection to other hosts.

Examples of other wildlife serving as \textit{M. bovis} reservoirs include ruminants such as African buffalo and Canadian bison, which were implicated in the epidemiology of bTB in South Africa and Canada, respectively (Nishi et al. 2006). The European badger was shown to be the source of \textit{M. bovis} in the recent outbreaks of bovine tuberculosis in Ireland and UK (Delahay et al. 2002; Phillips et al. 2003) and the bushtail possum was found to be a major wildlife reservoir of bTB in New Zealand (Coleman & Cooke 2001). In the USA, the white-tailed deer has been shown to be one of the major sources of tuberculosis for domestic animals in disease-free states (Corner 2006).

Table 2. Prevalence of important zoonotic bacterial pathogens in wild boars.

<table>
<thead>
<tr>
<th>bacteria</th>
<th>country</th>
<th>% positive for antibody (numbers +ve and numbers sampled)</th>
<th>% positive for isolation</th>
<th>domestic animal infection</th>
<th>reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>\textit{Brucella suis}</td>
<td>USA (SC)</td>
<td>18 (46/255)</td>
<td>1</td>
<td></td>
<td>Wood et al. (1976)</td>
</tr>
<tr>
<td></td>
<td>USA (CA)</td>
<td>15 (21/136)</td>
<td></td>
<td></td>
<td>Clark et al. (1983)</td>
</tr>
<tr>
<td></td>
<td>Italy</td>
<td>20 (4/20)</td>
<td></td>
<td></td>
<td>Giovannini et al. (1988)</td>
</tr>
<tr>
<td></td>
<td>USA (CA)</td>
<td>3.8 (23/611)</td>
<td></td>
<td></td>
<td>Drew et al. (1992)</td>
</tr>
<tr>
<td></td>
<td>USA (FL)</td>
<td>23.4 (238/1015)</td>
<td></td>
<td></td>
<td>van der Leek et al. (1993)</td>
</tr>
<tr>
<td>Czech</td>
<td></td>
<td>6 (2/32)</td>
<td></td>
<td></td>
<td>Hubalek et al. (1993)</td>
</tr>
<tr>
<td>Republic</td>
<td>USA (TN)</td>
<td>0 (0/108)</td>
<td></td>
<td></td>
<td>New et al. (1994)</td>
</tr>
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<td>USA (SC)</td>
<td>44 (120/272)</td>
<td></td>
<td></td>
<td></td>
<td>Gresham et al. (2002)</td>
</tr>
<tr>
<td>Italy</td>
<td>0 (0/562)</td>
<td></td>
<td></td>
<td></td>
<td>Ebani et al. (2003)</td>
</tr>
<tr>
<td>Croatia</td>
<td>29.4 (62/211)</td>
<td>29.2 (7/24)</td>
<td>yes</td>
<td></td>
<td>Cvetnic et al. (2003)</td>
</tr>
<tr>
<td>Germany</td>
<td>22 (168/763)</td>
<td></td>
<td></td>
<td></td>
<td>Al Dahouk et al. (2005)</td>
</tr>
<tr>
<td>Japan</td>
<td>7.8 (9/115)</td>
<td></td>
<td></td>
<td></td>
<td>Watarai et al. (2006)</td>
</tr>
<tr>
<td>Spain</td>
<td>29.7</td>
<td></td>
<td></td>
<td></td>
<td>Ruiz-Fons et al. (2006)</td>
</tr>
<tr>
<td>Switzerland</td>
<td>9.9 (180/1826)</td>
<td>5 (3/60)</td>
<td></td>
<td></td>
<td>Leuenberger et al. (2007)</td>
</tr>
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<td>\textit{Coxiella burnetii}</td>
<td>Italy</td>
<td>0</td>
<td></td>
<td></td>
<td>Giovannini et al. (1988)</td>
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<td></td>
<td>USA</td>
<td>50 (67/136)</td>
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<td></td>
<td>Clark et al. (1983)</td>
</tr>
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<td></td>
<td></td>
<td></td>
<td>Hubalek et al. (1993)</td>
</tr>
<tr>
<td>Republic</td>
<td>Czech</td>
<td>6 (2/32)</td>
<td></td>
<td></td>
<td>Hubalek et al. (1993)</td>
</tr>
<tr>
<td>\textit{Francisella tularensis}</td>
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<tr>
<td>Germany</td>
<td>18</td>
<td></td>
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<td></td>
<td>Jansen et al. (2007)</td>
</tr>
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<td></td>
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<td>Ebani et al. (2003)</td>
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<tr>
<td>USA (CA)</td>
<td>85 (111/136)</td>
<td></td>
<td></td>
<td></td>
<td>Clark et al. (1983)</td>
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<td>\textit{Mycobacterium avium}</td>
<td>Italy</td>
<td>23.8 (15/63)</td>
<td>yes</td>
<td></td>
<td>Serraino et al. (1999)</td>
</tr>
<tr>
<td>Spain</td>
<td>2.3</td>
<td></td>
<td></td>
<td></td>
<td>Parra et al. (2006)</td>
</tr>
<tr>
<td>Spain (C&amp;S)</td>
<td>56.8 (269/474)</td>
<td>yes</td>
<td></td>
<td></td>
<td>Vicente et al. (2006)</td>
</tr>
<tr>
<td>France</td>
<td>37.5 (90/240)</td>
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<td></td>
<td></td>
<td>Zanella et al. (2008)</td>
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<tr>
<td>\textit{Mycobacterium bovis}</td>
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<td>15 (9/59)</td>
<td>yes</td>
<td></td>
<td>Clark et al. (1983)</td>
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<tr>
<td>Germany</td>
<td>62.6 (478/763)</td>
<td></td>
<td></td>
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<td>Al Dahouk et al. (2005)</td>
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</table>

\textit{Yersinia pestis}

Table 2. Prevalence of important zoonotic bacterial pathogens in wild boars.
(b) Brucellosis  
Brucella suis is a Gram-negative coccobacilli and facultative intracellular pathogen that can cause zoonotic infections in pigs and other animals, including humans and wild boars, leading to abortions and infertility. Currently, eight species of Brucella are recognized that primarily affect a specific group of animals, including smooth B. melitensis (sheep and goats), B. suis (pigs), B. abortus (cattle), Brucella ceti (dolphins), Brucella pinnipedialis (seals), Brucella neotome (wood rat), rough Brucella canis (dogs) and Brucella ovis (sheep). Of the three major host-specific species of Brucella, B. suis is considered the second most pathogenic (only slightly less virulent than B. melitensis) in humans. Brucellosis is a chronic zoonotic disease resulting in "undulant fever" in humans and abortion and/or infertility in affected animals.

Wood et al. (1976) showed that 18 percent of feral pigs in South Carolina were serologically positive for B. suis, and the increasing incidence with age suggested that the pathogen was being transmitted among the population. The serological results were further confirmed by isolation of B. suis biotype 1 from the lymph node of an affected feral pig. Approximately 20–30% of feral swine and wild boars have been shown to be Brucella-positive by serology in Italy, USA and Croatia (table 2). The high level of exposure suggests that there is an active transmission and thus it may pose a serious threat to nearby domestic swine and hunters. Cvetnic et al. (2003) isolated B. suis biovar 2 from 58% per cent of swine and 62.5 per cent of aborted piglets. These authors also demonstrated that approximately 23% per cent of wild boars examined in the study were seropositive for Brucella and that wild boars should therefore be considered as wildlife reservoirs of B. suis biovar 2 in Croatia.

Al Dahouk et al. (2005) found that 22 per cent of wild boar sera in Germany were seropositive. Ruiz-Fons et al. (2006) showed that approximately 10 per cent of young adults and 28 per cent of adult wild boars in open estates in southern Spain were seropositive for brucellosis. By comparison, in wild boars of fenced estates, the incidence of seropositivity was approximately 32 and 38 per cent in young adults and adults, respectively. In Japan, Watarai et al. (2006) analysed 115 wild boars from four prefectures and found that approximately 7.5 per cent were seropositive (table 2).

The minimal infective dose of B. suis for humans is in the range of 10–100 colony forming units, and there are many cases of human infection owing to handling of infected feral and wild boars. Incidence of B. suis in wild boar populations has attained a very high level in southern Spain and pose a serious threat both to local domestic pigs and hunters and animal-care professionals. Starnes et al. (2004) reported two cases of brucellosis in members of a hunting club who had killed and prepared meat from wild boars.

(c) Leptospirosis  
Domestic pigs infected by L. interrogans are known to develop a 'carrier state' with intermittent shedding of the bacterium after an outbreak. Carrier pigs can host the leptospiral organism in the anterior convoluted tubules of kidneys even in the presence of specific circulating antibodies. Ebani et al. (2003) examined 562 blood samples of wild boars from Italy and showed that approximately 6 per cent were positive. Deutz et al. (2003) found that approximately 10 per cent of hunters from southeastern Austrian federal states of Styria and Burgenland had leptospiral antibodies. Jansen et al. (2007) detected leptospiral antibodies in 18 per cent of wild boars from Berlin, suggesting that infected wild boars can be the source of infections to domestic animals and humans.

Other zoonotic bacterial agents that wild boars can serve as reservoirs for include C. burnetii, the causative agent of Q fever, Francisella tularensis, which causes tularemia, and Y. pestis, the cause of bubonic and pneumatic plagues.

4. PARASITES IN WILD BOARS THAT ARE POTENTIALLY TRANSMISSIBLE TO LIVESTOCK AND HUMANS  
Wild boars are known to harbour two important parasites of humans in their edible tissues, and ingestion of raw or undercooked meat is the mode of infection. Freezing of wild boar meat is not effective protection against all species of Trichinella.

(a) Wild boars as reservoirs for important parasitic diseases of domesticated animals  
(i) Trichinella spp.  
Trichinella spiralis is perhaps the best-known nematode parasite of swine. There are presently eight named species of Trichinella based on the presence or absence of a capsule; mouse and pig infectivity; infectivity for birds and reptiles; resistance to freezing; and molecular characteristics (table 3). Three genotypes, viz. T6, T8 and T9, have been identified (Pozio & Murrell 2006). There are no published reports on clinical signs or disease in wild boar owing to Trichinella spp. infection. In one experimental study in wild boar, Tr. spiralis was found to be highly infective, Trichinella britovi, Trichinella nelsoni, Trichinella pseudospiralis (USA) and Tr. pseudospiralis (USSR) were moderately infective and Trichinella nativa, Trichinella murrelli, Trichinella pseudospiralis (Australia) and Trichinella T6 were poorly infective (Kapel 2001). Trichinella spp. circulate in sylvatic and domestic cycles in many areas of the world. Infection of domestic livestock and companion animals is usually asymptomatic. Transmission in animals is by predation, cannibalism, scavenging or by intentional feeding of raw or undercooked meat. A major concern is the introduction of Trichinella spp. in domestic pigs, which then serve as a source of human and other animal infections. Infections in horses, which have been widely reported, usually occur when the animals have been fed uncooked or undercooked meat from infected animals (Murrell et al. 2004). Trichinella papaue and Trichinella zimbabwensis are infectious for reptiles and mammals (Pozio et al. 2004b) but not fish (Pozio & Rosa 2005). Trichinella pseudospiralis is
infectious for birds and mammals but not reptiles (Pozio et al. 2004b). Trichinella papuae is common in wild pigs in Papua New Guinea, with a prevalence of approximately 11.5 per cent (Owen et al. 2005). The prevalence in farm-raised saltwater crocodiles owing to the feeding of wild boar meat in Papua New Guinea is approximately 21 per cent (Pozio et al. 2005). There is a single report of Tr. nelsoni in wild boar from Italy (Pozio et al. 1987), and both Tr. spiralis and Tr. britovi have been isolated from wild boar in Poland (Cabaj 2006) and Spain (Rodríguez et al. 2008). Antibodies to Trichinella spp. were found in 39 per cent of feral swine from coastal South Carolina, USA (Gresham et al. 2002). Trichinella murrelli is the species usually involved in the sylvatic cycle in temperate areas of North America (Pozio & La Rosa 2000).

(ii) Toxoplasma gondii
This parasite is found in virtually all warm-blooded animals. Domestic and all other breeds of cat are the definitive host and excrete oocysts in their faeces. These oocysts sporulate and become infective in 1–2 days in the environment. Wild boar and other animals become infected by ingesting sporulated oocysts in the environment or by ingesting tissue cysts within the tissues of intermediate hosts. There are no reports of clinical toxoplasmosis in wild boar. Toxoplasma gondii can be transplacentally transmitted in domestic pigs and infections are usually asymptomatic. Young pigs are more susceptible to clinical disease than are older pigs (Dubey 1986).

Toxoplasma gondii was isolated from 2 per cent of wild boars in the Czech Republic, and a serological prevalence of 15 per cent was found in 124 wild boars (Hejliček et al. 1997). The serological prevalence of T. gondii in wild boars was 5.6 per cent from Iriomote Island (Nogami et al. 1999) and 1.1 per cent from Amakusa Island, Japan (Shibashi et al. 2004). Samples from 13 per cent of 135 wild hogs from California, USA, tested positive for antibodies to T. gondii (Clark et al. 1983). In South Carolina (USA), antibodies to T. gondii were found in 49 (Gresham et al. 2002) and 37 per cent of feral swine (Diderrich et al. 1996). Serum samples from 31 per cent of feral swine from the Great Smoky Mountains National Park, USA, were positive for antibodies to T. gondii (Diderrich et al. 1996). A study on Ossabaw Island, GA, USA, found a serological prevalence of only 0.9 per cent of 1264 wild boars and a serological prevalence of 18.2 per cent of 170 wild boars from mainland Georgia, USA (Dubey et al. 1997). The low serological prevalence on Ossabaw Island was attributed to a scarcity of domestic cats: only one domestic cat was known to be on the island and its whereabouts were not known (Dubey et al. 1997). It is well known that domestic pigs raised outdoors have a higher serological prevalence for T. gondii than do pigs raised indoors (van der Giessen et al. 2007). Exposure of wild boar to feline faeces and cannibalism are probably the reasons for an unusually high prevalence of T. gondii in wild boars.

(b) Wild boars as reservoirs for zoonotic parasitic infections in humans

(i) Trichinella spp.
Trichinosis is an emerging zoonotic disease in several European countries, and outbreaks after the consumption of wild boar meat are common (Serrano et al. 1989; Rodríguez-Osorio et al. 1999). In a survey from Spain, 75 per cent of 49 Trichinella outbreaks arose from the ingestion of wild boar meat (Rodríguez de las Parras et al. 2004). In Poland, it was estimated that 88 per cent of trichinosis cases during 2000–2005 were due to ingestion of wild boar (Gołab & Sadkowska-Todys 2006). Of special importance is the infection of wild boar with the highly freezing-resistant Tr. nativa in Spain (Pozio & Kapel 1999), and infection of wild boar with the moderately freezing-resistant Tr. britovi is common. Trichinella britovi has been reported from wild boar from Belgium (Schyns et al. 2006), Spain (Rodríguez de las Parras et al. 2004) and France (De Bruyne et al. 2006). The non-encapsulated Tr. pseudospiralis has been found in wild boar from Sweden (Pozio et al. 2004a), Texas, USA (Gamble et al. 2005) and Thailand (Jongwutiwes et al. 1998). An outbreak of Tr. pseudospiralis in Thailand involved 59 individuals and one died (Jongwutiwes et al. 1998). In Papua New Guinea where Tr. papuae is common in wild boar, 10 per cent of the population has antibodies to the parasite (Owen et al. 2005). Trichinella nelsoni is known to establish muscle infections in experimentally infected wild boar (Kapel et al. 2005). A single case of trichinellosis was reported from consumption of wild boar in the USA from 1991 to 2001 (Roy et al. 2003). Trichinella genotypes T6, T8 and T9 mainly exist in sylvatic cycles. Human infections with Trichinella T6 have been reported in individuals who consumed cougar or black bear meat, and symptoms were mild. No reports of human infection with genotypes T8 and T9 have been reported (Pozio & Murrell 2006). Because freezing does not kill all species of Trichinella, proper cooking is the best means to prevent human infection. The killing of Trichinella larvae in meat is time and temperature dependent (Kotula et al. 1983). In a well controlled study, Kotula et al. (1983) found that larvae in 2 mm thick portions of homogenized pork retained their infectivity in samples.
heated to 55°C for 4 min, but were rendered non-
infected after 6 min at 55°C. Larvae in samples
heated to 49°C were infected after 5 h, but not after
6 h. One case of trichinosis has been reported in a
person who ate bear hamburgers that had been
cooked in a microwave oven, suggesting that some
larvae may withstand microwave cooking (Nelson et al. 2003).

(ii) Toxoplasma gondii
Most cases of toxoplasmosis in immunocompetent
humans are subclinical. Congenital toxoplasmosis
has long been recognized for the devastating effects it
can have on the infected foetus (Jones et al. 2001),
including hydrocephalus, blindness and mental retar-
dation. Gauss et al. (2005) found that 38.4 per cent
of 507 hunters who killed wild pigs in Spain were posi-
tive for antibodies to T. gondii, and Ruiz-Fons et al.
(2006) found a prevalence of 36.3 per cent in hunters
who killed wild boar sows. The relative importance of
meat or oocysts as a source of human infection is
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unknown. An outbreak of unilateral chorioretinitis
owing to T. gondii was reported in three patients
from Korea who ingested raw liver and spleen from a
wild pig (Choi et al. 1997). Although no other
outbreaks have been reported from wild boar, the
generally high serological prevalence of T. gondii in these
animals indicates that consumption of raw or rare
wild boar meats is likely to lead to an infection with
T. gondii.

No freezing-resistant strains of T. gondii have been
identified: freezing meat to –12°C will kill tissue
cysts (Dubey 1996). Cooking is an effective means of
killing tissue cysts. One controlled study demonstrated that
T. gondii tissue cysts in 2 mm thick samples
remained viable at 52°C for 9.5 min, but not at 58°C
(Dubey et al. 1990). Tissue cysts in pork samples
were generally rendered non-viable by heating to
61°C or higher temperature for 3.6 min (Dubey et al.
1990) and have been reported to survive 64°C for
3 min (Dubey et al. 1990). Cooking meat to an internal
temperature of 67°C will kill tissue cysts
(Dubey 1996).

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