

Review

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

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Table 1. Prevalence of viruses in wild boars and potential risks for transmission to domestic animals and humans.

virus ^a	country	% positive for viral antibody	% positive for viral nucleic acid	zoonosis	domestic animal infection	reference
HEV				yes	yes	
	Spain	42	20			de Deus <i>et al.</i> (2008)
	Germany		5			Kaci <i>et al.</i> (2008)
	Italy		25			Martelli <i>et al.</i> (2008)
	Japan (Ehime)	26	3			Michitaka <i>et al.</i> (2007)
PRV	Australia	17				Chandler <i>et al.</i> (1999)
				no	yes	
	USA	38				Corn <i>et al.</i> (2004)
	Spain	46 ± 8	31 ± 7			Ruiz-Fons <i>et al.</i> (2007)
	Italy	51				Lari <i>et al.</i> (2006)
	Slovenia	31				Vengust <i>et al.</i> (2006b)
	Spain	61				Ruiz-Fons <i>et al.</i> (2006)
	Spain	36				Vicente <i>et al.</i> (2002)
	Croatia	55				Zupancic <i>et al.</i> (2002)
	Germany	9				Müller <i>et al.</i> (1998)
PCV2				no	yes	
	Hungary		21			Cságola <i>et al.</i> (2006)
	Spain	52				Ruiz-Fons <i>et al.</i> (2006)
JEV	Spain	48 ± 2				Vicente <i>et al.</i> (2004)
				yes	yes	
JEV	Japan (Okinawa)	65				Nidaira <i>et al.</i> (2007)
	Japan (Iriomote)	4				Nidaira <i>et al.</i> (2007)
CSFV				no	yes	
	Croatia	39				Zupancic <i>et al.</i> (2002)
	Slovenia	3				Vengust <i>et al.</i> (2006a)
	Switzerland	31	34			Schnyder <i>et al.</i> (2002)
	France	<1				Albina <i>et al.</i> (2000)
PPV				no	yes	
	Slovenia	49				Vengust <i>et al.</i> (2006b)
	Croatia	42				Roic <i>et al.</i> (2005)
	Spain	57				Ruiz-Fons <i>et al.</i> (2006)
	Spain	10				Vicente <i>et al.</i> (2002)
	OK, USA	17				Saliki <i>et al.</i> (1998)
	TN, USA	14				New <i>et al.</i> (1994)
PRRSV				no	yes	
	France	11				Albina <i>et al.</i> (2000)
	OK, USA	2				Saliki <i>et al.</i> (1998)
	Spain	0				Ruiz-Fons <i>et al.</i> (2006)
ASFV	Croatia	0				Zupancic <i>et al.</i> (2002)
				no	yes	
ASFV	Spain	10				Pérez <i>et al.</i> (1998)
TTV				no	yes	
TTV	Spain	84				Martínez <i>et al.</i> (2006)
swine influenza virus				yes	yes	
	OK, USA	11				Saliki <i>et al.</i> (1998)
	Spain	4				Vicente <i>et al.</i> (2002)

^aHEV, hepatitis E virus; PRV, pseudorabies virus; PCV2, porcine circovirus type 2; JEV, Japanese encephalitis virus; CSFV, classical swine fever virus; PPV, porcine parvovirus; PRRSV, porcine reproductive and respiratory syndrome virus; ASFV, African swine fever virus; TTV, torque teno virus.

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 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 epidemics, 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 times (swine HEV antigen, $p = 0.03$) more likely to be positive for 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

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

bacteria	country	% positive for antibody (numbers +ve and numbers sampled)	% positive for isolation	domestic animal infection	reference		
<i>Brucella suis</i>	USA (SC)	18 (46/255)	1		Wood <i>et al.</i> (1976)		
	USA (CA)	15 (21/136)			Clark <i>et al.</i> (1983)		
	Italy	20 (4/20)			Giovannini <i>et al.</i> (1988)		
	USA (CA)	3.8 (23/611)			Drew <i>et al.</i> (1992)		
	USA (FL)	23.4 (238/1,015)			van der Leek <i>et al.</i> (1993)		
	Czech Republic	6 (2/32)			Hubálek <i>et al.</i> (1993)		
	USA (TN)	0 (0/108)			New <i>et al.</i> (1994)		
	USA (SC)	44 (120/272)			Gresham <i>et al.</i> (2002)		
	Italy	0 (0/562)			Ebani <i>et al.</i> (2003)		
	Croatia	29.4 (62/211)			29.2 (7/24)	yes	Cvetnic <i>et al.</i> (2003)
	Germany	22 (168/763)			Al Dahouk <i>et al.</i> (2005)		
	Japan	7.8 (9/115)			Watarai <i>et al.</i> (2006)		
	Spain	29.7			yes	Ruiz-Fons <i>et al.</i> (2006)	
	Switzerland	9.9 (180/1826)			5 (3/60)	Leuenberger <i>et al.</i> (2007)	
<i>Coxiella burnetii</i>	Italy	0			Giovannini <i>et al.</i> (1988)		
	USA	50 (67/136)			Clark <i>et al.</i> (1983)		
	Czech Republic	6 (2/32)			Hubálek <i>et al.</i> (1993)		
<i>Francisella tularensis</i>	Czech Republic	6 (2/32)			Hubálek <i>et al.</i> (1993)		
	Germany	3.1 (24/763)			Al Dahouk <i>et al.</i> (2005)		
<i>Leptospira interrogans</i>	USA	87 (118/136)			Clark <i>et al.</i> (1983)		
	Germany	18	yes		Jansen <i>et al.</i> (2007)		
<i>Mycobacterium avium</i> Co	Italy	6 (34/562)			Ebani <i>et al.</i> (2003)		
	USA (CA)	85 (111/136)			Clark <i>et al.</i> (1983)		
<i>Mycobacterium bovis</i>	Italy	23.8 (15/63)		yes	Serraino <i>et al.</i> (1999)		
	Spain	2.3		yes	Parra <i>et al.</i> (2006)		
	Spain (C&S)	56.8 (269/474)		yes	Vicente <i>et al.</i> (2006)		
	France	37.5 (90/240)		yes	Zanella <i>et al.</i> (2008)		
<i>Yersinia pestis</i>	USA (CA)	15 (9/59)			Clark <i>et al.</i> (1983)		
	Germany	62.6 (478/763)			Al Dahouk <i>et al.</i> (2005)		

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 *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 *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 *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 *M. bovis* in the recent outbreaks of bovine tuberculosis in Ireland and UK (Delahay *et al.* 2002; Phillips *et al.* 2003) and the brushtail 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).

(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 hunt 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 tularaemia, and *Y. pestis*, the cause of bubonic and pneumonic 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 papuae* and *Trichinella zimbabwensis* are infectious for reptiles and mammals (Pozio *et al.* 2004b) but not fish (Pozio & Rosa 2005). *Trichinella pseudospiralis* is

Table 3. Characteristics of presently named species of *Trichinella*. Adapted from Pozio & Murrell 2006.

name (genotype)	muscle capsule	resistance to freezing	wild boar
<i>Tr. spiralis</i> (T1)	yes	no	yes
<i>Tr. nativa</i> (T2)	yes	strong	yes
<i>Tr. britovi</i> (T3)	yes	moderate	yes
<i>Tr. pseudospiralis</i> (T4)	no	no	yes
<i>Tr. murrelli</i> (T5)	yes	no	yes ^a
<i>Tr. nelsoni</i> (T7)	yes	no	yes
<i>Tr. papuae</i> (T10)	no	no	yes
<i>Tr. zimbabweensis</i> (T11)	no	no	yes ^b

^aExperimental infection.^bExperimental infection of domestic pigs.

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 (Hejlíček *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 (Shiibashi *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 Smokey 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 (Golab & Sadowska-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 (Schynts *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-infective after 6 min at 55°C. Larvae in samples heated to 49°C were infective 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 retardation. Gauss *et al.* (2005) found that 38.4 per cent of 507 hunters who killed wild pigs in Spain were positive 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 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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