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# Suidae and Tayassuidae

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## INTRODUCTION

The suids, *Suidae* and *Tayassuidae*, primarily species of the tropics and subtropics, have a worldwide distribution on all continents except Antarctica. The true wild boars were indigenous to Europe and Asia and are the ancestor to the domestic pig with which it shares the same scientific name, *Sus scrofa*. Wild boars can also survive in temperate climates and have been introduced to the Americas and many islands. Because of the close genetic relationship, in many areas they have interbred with domestic pigs and formed considerable populations of feral suids that represent wild boar and feral pig crosses.

Wild suid populations are relatively hardy and most disease research has been focused on their potential as a reservoir for diseases of concern for commercial pig production. High densities and gregarious behavior increase the chance of disease transmission. Many wild boar populations have overlapping ranges with commercial swine operations, which increases the risk for transmission among these groups. Within the *Suidae*, the Togian Island babirusa, pygmy hog, Visayan warty pig, and the Javan warty pig are listed as endangered or critically endangered. Within the *Tayassuidae*, only the Chacoan peccary is endangered. For all suids, hunting, habitat loss, and hybridization are important threats to conservation.

## UNIQUE FEATURES

Nondomestic suids share many anatomic and clinical pathologic features with domestic pigs. Placentation is diffuse, epitheliochorial and in warthogs the villi are short and blunt. ([www.placentation.ucsd.edu](http://www.placentation.ucsd.edu)). The dental formula is variable (I 1-3/3, C 1/1, P 2-4/2-4, M 3/3) (Sutherland-Smith, 2015). Desert warthogs lack upper incisors. In babirusa, the canine tusks erupt dorsally curving

caudally (Fig. 8.1). In rare cases, when they do not have the opportunity to grind them down, the tusks can pierce the skin and grow into the skull. Most suids are omnivores and diet varies with local availability, for example, including cactus in the diets of some peccaries. Most suids have a simple stomach similar to the domestic pig. Babirusa has an enlarged stomach with a diverticulum lined by mucus glands for fermentation; reduced fermentation occurs in the large colon. Peccaries have a four-chambered stomach with three nonglandular and one glandular stomach. Peccaries lack a gall bladder (Gottdenker and Bodmer, 2004). Along the dorsum cranial to the tail base, peccaries have prominent scent gland. In Chacoan peccaries, prominent salivary gland papillae have been described (Sutherland-Smith, 2015).

Histologically, wild suids share many features with domestic pigs. This includes distinct hepatic lobules bridged by fibrous connective tissue (Fig. 8.2) and an “inside-out” lymph node in which follicles are present centrally and surrounded by sinusoids.

## NON-INFECTIOUS DISEASES

### Nutritional

**Obesity** is not uncommon in captive suids. It predisposes to arthritis and may negatively impact reproduction (Sutherland-Smith, 2015).

### Metabolic

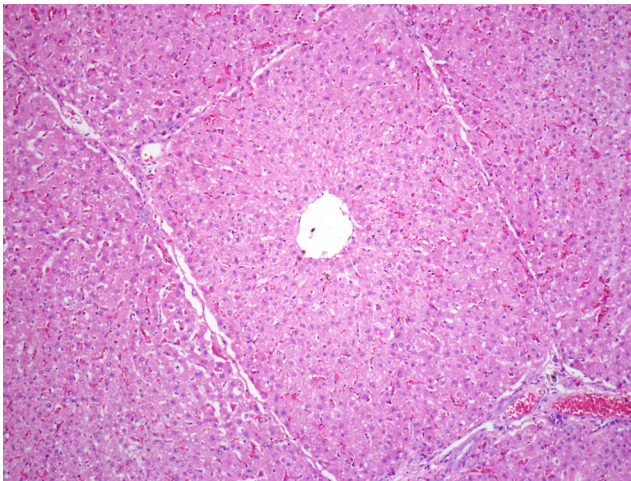
**Hyperthermia** is not uncommon in both the *Suidae* and *Tayassuidae*, and can be fatal. Beyond elevated environmental temperatures in areas with inadequate ventilation, shipping, anesthesia, and stressful situations can precipitate hyperthermia. Capture and restraint are common causes

of mortality, possibly exacerbated by hyperthermia, as is capture myopathy (Batista et al., 2008, 2014; Sutherland-Smith, 2015).

Piglets are sensitive to **hypothermia**. Unlike other mammals, suids appear to lack brown adipose hindering their ability to thermoregulate when young. Domestic pigs, European wild boars, Bornean bearded pigs, warthogs, and red river hogs all have deletions within the *uncoupling protein 1* (UCP1) gene (Berg et al., 2006). UCP1 is expressed exclusively in brown fat and located along the inner membrane of mitochondria where it catalyzes protein leakage that results in release of heat. The European wild boar shivers when cold and builds farrowing nests, which may be behavioral adaptations to more temperate climates.



**FIGURE 8.1** Normal babirusa tusks. The canine teeth are modified in this species into dorsally erupting curved tusks, which, if not worn through use, can pierce the skin and skull. (Photo Courtesy of the Wildlife Conservation Society)



**FIGURE 8.2** Normal liver from a warthog. Note the thin fibrous connective tissue septae separating the liver into lobules.

**Gastric ulceration** in the glandular stomach is not uncommon, especially during periods of stress.

### Congenital/Genetic

Rare congenital anomalies have been described. These include duplication of the pelvis and rear extremities suggestive of ischiopagus, a type of conjoined twin, in Chacoan peccaries (Benirschke et al., 1995), and cyclopia and limb deformities in collared peccaries (Hellgren et al., 1984).

### Age-Related/Degenerative

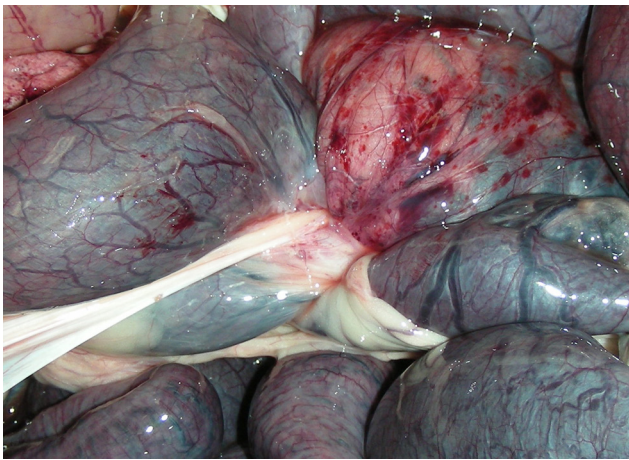
In both captivity and in the wild, nondomestic suids are generally hardy animals and develop common age-related degenerative conditions. **Arthritis** is most common in joints of the distal appendicular skeleton although intervertebral disc disease also occurs. In captivity, hoof problems are also common. **Periodontal disease** can also occur and primarily affects the cheek teeth. Studies of wild red river hog and warthog skulls noted an increase in prevalence of periodontal disease with age in red river hogs but not warthogs, presumably due to their more abrasive diet (Woodall, 1989). **Polycystic kidney disease** has also been reported in peccaries and is presumed to be acquired rather than genetic (Batista et al., 2014). Similar to domestic species, **cystic endometrial hyperplasia** (Fig. 8.3) can develop in nondomestic suids (Thompson et al., 2015). Cyst epithelium lacks vacuoles typical of progesterone stimulation, thus the pathogenesis is presumed similar to that of domestic pigs where cystic endometrial hyperplasia is associated with prolonged estrogen stimulation (endogenous or exogenous).



**FIGURE 8.3** Cystic endometrial hyperplasia in a minipig. Bilaterally the uterine horns are enlarged and dilated. The left uterine horn is opened and contains numerous, variably sized, cystic structures that expand the endometrium. In suids, this condition is associated with prolonged estrogen stimulation.

## Miscellaneous

Both **intestinal** and **gastric volvulus**, occur in a variety of suids (Fig. 8.4) (Batista et al., 2014). **Trichobezoars** are somewhat common in captive collared peccaries (Batista et al., 2014). Most cases are incidental findings at necropsy but they may be associated with hyporexia and weight loss. Bezoars occur in adults without apparent sex predilection. They tend to be single, round to stomach-shaped (Fig. 8.5), and up to 5–7 cm diameter and 14 cm long and filling most of the stomach's lumen. Bezoars are composed almost entirely of compacted hair but may contain areas of mineralization. Mild to severe erosions of the



**FIGURE 8.4** Intestinal volvulus in a red river hog. Torsion at the root of the mesentery occurs in multiple species of suids. In this case, 360-degree torsion around the cranial mesenteric artery (center of the image) is associated with distension and congestion of the small intestine and multifocal serosal hemorrhage.



**FIGURE 8.5** Trichobezoars from the stomachs of collared peccaries. Note that some bezoars take on the shape of the stomach.

gastric mucosa can occur. Low fiber diets may predispose to the formation of bezoars, and excessive grooming due to stress is contributory.

## Neoplastic

Neoplasms are infrequently described in suids and include lymphosarcoma, intestinal carcinoma, squamous cell carcinoma, uterine adenocarcinoma, uterine leiomyosarcoma, pheochromocytoma, plasmacytomas, heman-giomas, and mammary carcinomas (Batista et al., 2014; Cole et al., 2008; Olinda et al., 2016; Sutherland-Smith, 2015).

## INFECTIOUS DISEASES

Much of the research on infectious disease in wild suids is focused on diseases of concern for domestic pigs or potential zoonoses. Wild suids are important reservoirs for maintenance and transmission of several viruses and bacteria of concern to livestock and human health (Meng et al., 2009; Ruiz-Fons, 2017). Supplemental Table e1 contains a list of several infectious diseases to which wild suids may be infected and serve as either a reservoir or part of the multihost disease ecology but for which associated disease has not been described or is mild.

## DNA Viruses

**African swine fever (ASF)** is a devastating, OIE listed, notifiable systemic disease in domestic pigs that causes up to 100% morbidity and mortality in naive populations. The disease is caused by a DNA arbovirus (family *Asfviridae*) with multiple genotypes from different geographic regions. **ASF virus (ASFV)** is endemic in most of sub-Saharan Africa and Sardinia, and the virus is spreading through the Caucasus, Eastern Europe, and Baltic countries (Guinat et al., 2016; Sanchez-Vizcaino et al., 2015). ASFV is transmitted by direct contact with infected animals, contaminated fomites, feces, ingestion of contaminated feed, and ticks of the genus *Ornithodoros* (Guinat et al., 2016; Ravamoana et al., 2010).

Warthogs, bushpigs, and *Ornithodoros* species of ticks are natural hosts of ASFV. A sylvatic cycle involving wild suids and soft ticks of the *Ornithodoros moubata* complex has been established in East and Southern Africa, and possibly the Indian Ocean region (Jori et al., 2013). However, the tick/warthog sylvatic cycle is questionable in other regions of Africa where the disease is also endemic (Guinat et al., 2016; Jori and Bastos, 2009; Jori et al., 2013). Warthogs are susceptible to infection but do not develop clinical signs of disease (Kleiboeker and Scoles, 2001). The majority of warthogs are infected during the first few weeks of life and develop a generalized



**FIGURE 8.6** Experimental African swine fever virus (ASFV) infection in a European wild boar. (A) Splenomegaly; while not pathognomonic, is more common in ASFV than with classical swine fever infections. (B) Renal petechiae can occur in infections with both African swine fever and classical swine fever virus infections and cannot therefore be used as a differentiating feature (see also Fig. 8.10C). (C) Hemorrhagic lymphadenitis. (Photos Courtesy of the Friedrich Loeffler Institute)

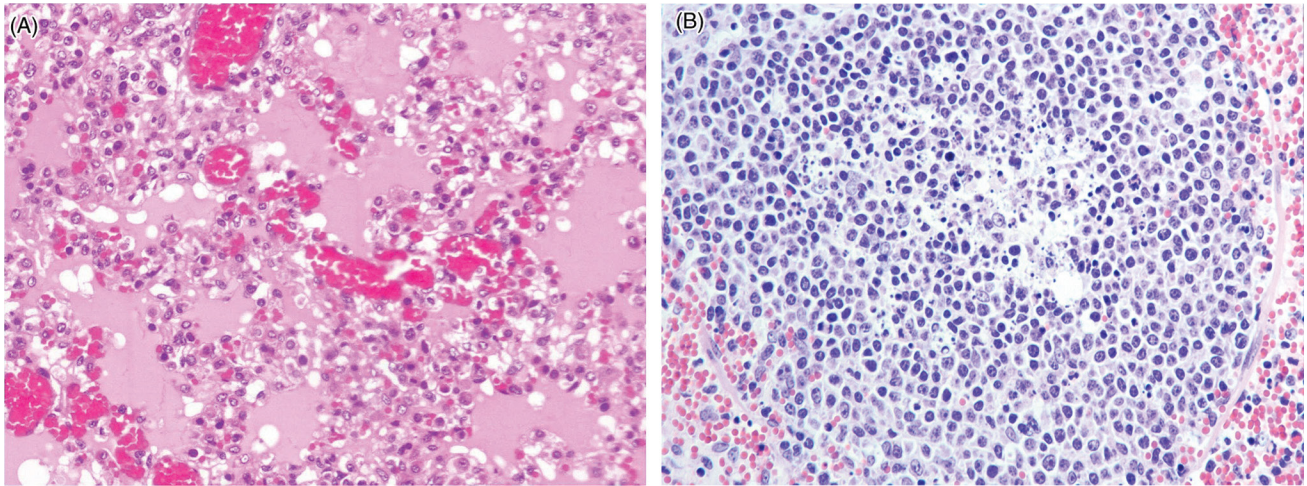
viremia, sufficient to infect ticks that feed on them. The virus then remains in high numbers for a limited period in systemic lymph nodes. Adult warthogs remain infected but remaining viral load is low. Ticks in warthog burrows feed on infected animals and become infected (Jori and Bastos, 2009; Jori et al., 2013). Transmission to domestic pigs in endemic areas is by tick bites; maintenance of ASFV in domestic pigs and pig to pig transmission also occurs (Guinat et al., 2016; Sanchez-Vizcaino et al., 2015).

Bushpigs are also suspected to be natural reservoirs of ASFV, but the role they play in the ecology of ASF is considered of lesser importance than the warthog (Ravamoana et al., 2010, 2011). Natural infection is known to occur without clinical signs, and blood levels in infected bushpigs are high enough to infect both ticks and domestic pigs. However, unlike warthogs, bushpigs do not live in burrows where contact with ticks usually takes place. In Madagascar, where ASF was introduced in the late 1990s and evolved into an enzootic disease, bushpigs were sporadically infected but, to date, do not appear to play a role in maintenance and transmission of ASF (Ravamoana et al., 2010, 2011; Roger et al., 2001). Although the frequency and prevalence of infection is lower than in warthogs, bushpigs should be considered possible reservoirs and a source of transmission of ASFV in endemic countries in east and southern Africa (Jori et al., 2013). Peccaries were not susceptible to ASFV in experimental studies (Dardiri et al., 1969).

The role of other wild pigs in the epidemiology of ASF is often obscure and variable, particularly because of differences between the four wild suid genera, *Phacochoerus*, *Potamochoerus*, *Hylochoerus* and *Sus* in taxonomy, distribution, ecology, ASF clinical signs, and potential contact with domestic pigs, among other factors (Jori and Bastos, 2009). For instance, though ASF infection in warthogs and bushpigs in endemic regions is asymptomatic, several experimental studies suggest that the Eurasian wild boar is highly susceptible to acute disease similar to domestic pigs. Additional experimental studies demonstrated transmission

occurring directly between wild boars and domestic pigs (Gabriel et al., 2011). Furthermore, infection of free-ranging wild boars and deaths caused by ASF has been observed in Russia (Gogin et al., 2013; Guinat et al., 2016). However, it is unclear whether ASFV can be sustained in these wild populations, and persistence of ASF may be self-limiting (Guinat et al., 2016; Lange et al., 2014).

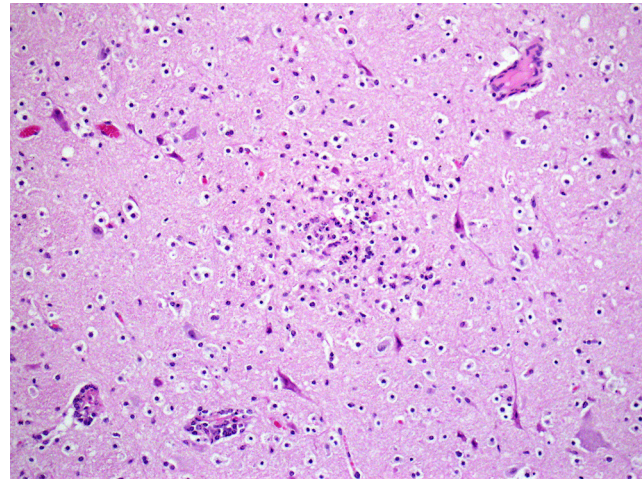
In experimental studies, the Eurasian wild boar suffers peracute disease and 100% mortality after infection with ASFV (Blome et al., 2012; Gabriel et al., 2011). Clinical signs in boar include lethargy, depression, anorexia, diarrhea, respiratory distress, and high fever; less commonly, epistaxis, and neurological signs may be observed. In experimental studies, boars died within 7–9 days postinfection regardless of age or sex (Blome et al., 2012). Lesions include acute hemorrhagic lymphadenitis, hemorrhagic gastritis, pulmonary edema, splenomegaly, petechiae, and ecchymoses (Figs. 8.6 and 8.7). Skin lesions have not, to date, been reported in natural or experimental disease in boar; however, because their skin is dark colored, they are difficult to assess. Domestic pigs with ASF have concurrent thrombocytopenia and leukopenia. The pathogenesis in boar is unknown but in domestic pigs both viral and host factors impact disease development (reviewed in Blome et al., 2013). In general, the monocyte/macrophage lineage is a key viral target, and subsequent production of cytokines, such as TNF- $\alpha$ , IL-1 $\alpha$ , IL-1 $\beta$ , and IL-6 are critical to the development of disease. It is the production of these proinflammatory cytokines, rather than direct viral damage, that is associated with clinical signs and activation of endothelial cells resulting in activation of the clotting cascade as well as lymphocyte apoptosis. In more chronic infections, immune complexes may be important in the pathogenesis of vascular damage. Infection needs to be differentiated from classical swine fever infection. PCR for the p72 gene and immunofluorescent antigen detection in tissue smears offer rapid diagnostic options. Sections of tonsil, lymph node, kidney, and/or spleen as well as blood samples are the most useful for diagnosis.



**FIGURE 8.7** African swine fever virus infection in a domestic pig. (A) Diffuse pulmonary edema with flooding of alveolar air spaces by edema and foamy macrophages. There is hemorrhage within alveolar septae. (B) Necrosis of lymphocytes within a lymph node with central cellular debris and apoptotic bodies. Surrounding the follicle are extravasated erythrocytes. (Photos Courtesy of the Department of Compared Anatomy and Anatomic Pathology, Veterinary Faculty of the University of Cordoba, Spain [Archive].)

**Suid herpesvirus 1 (SuHV1) (syn. Pseudorabies virus [PrV])** is an *Alphaherpesvirus* and the causative agent of **Aujeszky's disease** or **pseudorabies**. This disease is OIE listed and notifiable. It has been eradicated from domestic pigs in North America, New Zealand, and some countries in Europe. However, it remains an important disease in domestic pigs in other areas of the world and is a disease for which wild boars are considered reservoir hosts. Seroprevalence in wild boars varies geographically and in some regions viral infection may be endemic (Köppel et al., 2007; Montagnaro et al., 2010; Müller et al., 2011; Ruiz-Fons et al., 2006; Vengust et al., 2006; Župančić et al., 2002). Rare seropositivity has also been noted in peccaries in the southwestern United States and SuHV-1 has been identified by PCR in postmortem samples from white-lipped and collared peccaries in Brazil (Corn et al., 1987; De Castro et al., 2014). Multiple genotypes are present in wild boar populations, some of which suggest transmission from domestic or feral pigs while others are distinct from strains found in domestic pigs inhabiting the same geographic region. Oral, nasal, and venereal shedding are suggested based on virus isolation studies with higher viral DNA loads in genital swabs (Müller et al., 2011; Romero et al., 1997). Piglets may have both anti-PrV antibodies and virus suggesting vertical transmission (Verin et al., 2014). Taken together, these findings support the importance of venereal transmission in adult boars.

The pathogenesis of PrV infection in wild boars is incomplete as outcome of experimental infection has varied and disease has generally been milder than what is noted in domestic pigs. To what extent this is due to PrV strain factors, host adaptation or earlier maturation of CD8 T cells in wild boars is uncertain (Müller et al., 2011; Page et al., 1992). While young domestic piglets (<7 days) are highly susceptible, high mortality has not been described



**FIGURE 8.8** Suid herpesvirus infection encephalitis in a pig. Necrotizing nonsuppurative encephalitis is associated with neuronal necrosis and perivascular cuffing. Eosinophilic intranuclear inclusions may be present but are generally more easily seen with immunohistochemical labeling.

in similar age wild suids; disease in young wild suids may be underrepresented due to the difficulty in recovering carcasses from very young wild animals. In domestic pigs, primary replication occurs in nasal and oropharyngeal mucosa with viral latency within ganglia. Clinical disease and lesions, when described, appear to be more common and more severe in juvenile boars (4–18 months) than adults (Gortázar et al., 2002; Schulze et al., 2010). Similar to domestic pigs, neurologic signs and lesions are more common in younger boars. Gross lesions in natural outbreaks include lymphadenomegaly and meningeal congestion. Histologically, nonsuppurative encephalitis with neuronal necrosis, lymphoplasmacytic perivascular cuffs and edema have been described (Fig. 8.8). Intranuclear inclusions are

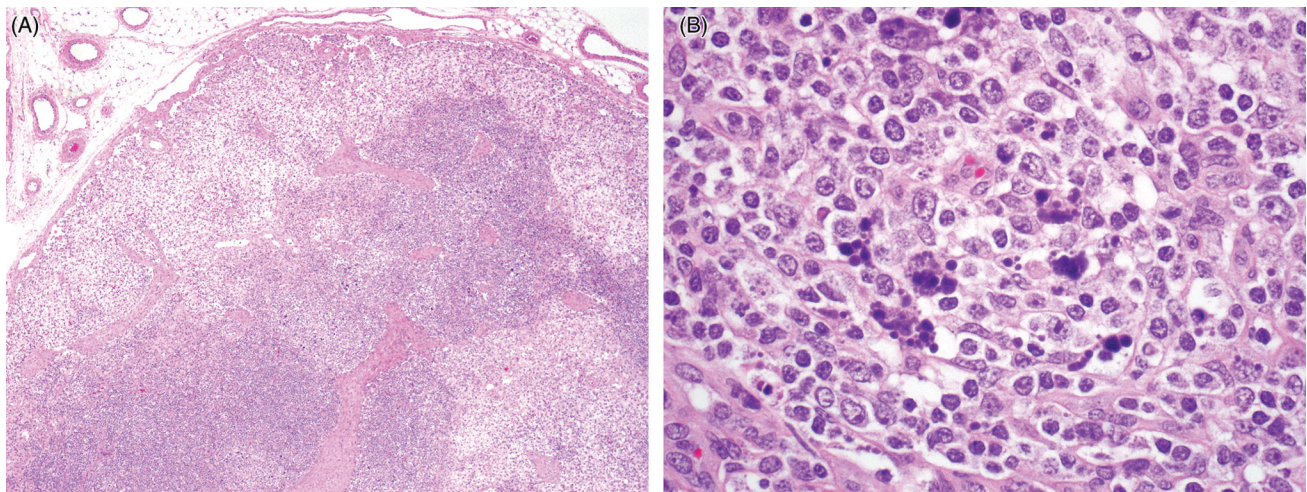
variably noted but virus can be demonstrated by immunohistochemistry (IHC) and immunofluorescence in sections of tonsil and brain. In adult domestic pigs, respiratory signs are a hallmark of PrV infection but they have not been described in natural infections in boars. Mild sneezing, nasal discharge, and conjunctivitis have been described after experimental infection of wild boar with strains of PrV isolated from wild boar (Müller et al., 2001). However, wild boar treated with immunosuppressant drugs develop severe pleuropneumonia as do wild boar inoculated experimentally with some domestic pig PrV strains. Further research is needed to better understand the pathogenesis of these varying strains in the differing hosts.

**Porcine circoviruses (PCV)** occur as two genotypes, PCV1 which is considered nonpathogenic in domestic pigs and PCV2, the cause of **post weaning multisystemic wasting syndrome (PMWS)**. PCV1 has been detected by PCR and serology in wild boar populations but not associated with disease, similar to domestic pigs (Cságola et al., 2008; Tischer et al., 1986). PCV2 seroprevalence in European wild boar populations is generally high suggesting the virus is endemic (Cano-Manuel et al., 2014; Ruiz-Fons et al., 2006). Some populations have evidence of exposure without evidence of disease (Reiner et al., 2010a), but other populations have had histologic lesions and increased piglet mortality. In some cases, disease has followed confinement of wild animals suggesting potential cofactors in pathogenesis (Ellis et al., 2003). Affected animals are generally young (4–10 m).

Lesions in wild boars are similar to those described in domestic pigs and include emaciation with adipose and skeletal muscle atrophy, lymphoid depletion with sinus histiocytosis in lymph nodes (Fig. 8.9A) and Peyer's patches, lymphoplasmacytic interstitial pneumonia, and mild erosive enterocolitis associated with mononuclear inflammatory cell infiltrates (Ellis et al., 2003; Hohloch

et al., 2015; Lipej et al., 2007; Schulze et al., 2003; Sofia et al., 2008; Vicente et al., 2004). Characteristic basophilic, botryoid intracytoplasmic and less common intranuclear inclusions are present within macrophages (Fig. 8.9B). Diagnosis can be confirmed by immunohistochemical labeling of PCV antigen in macrophages, lymphatic endothelial cells, and in gut associated lymphoid tissue. PCR is also available, although as the virus appears to be endemic in wild suids in some areas, identification of viral DNA may not imply causality. Furthermore, in some studies, PCV2 DNA could not be detected even in seropositive boars with histologic lesions of PMWS (Hohloch et al., 2015). Other lesions, such as hyperplasia of bronchial-associated lymphoid tissue may be due to secondary infections with *Mycoplasma* sp. or *Pneumocystis* sp. (Borba et al., 2011). Populations of boar with higher PCV2 prevalence also have higher prevalence of *Mycobacterium bovis* infection, and more severe and more generalized disease (Risco et al., 2013, 2014). However, findings in other studies have been less clear suggesting that the interaction between PCV2 infection and potential copathogens may be more complex than simply PCV2 induced immune suppression (Díez-Delgado et al., 2014). In addition to boars, PCV2 has been identified by PCR in tissues from white-lipped and collared peccaries in Brazil but no lesions have been described (De Castro et al., 2014). Collared peccaries lack antibodies to PCV2 (Gerber et al., 2012); however, absence of detectable titers can be noted in domestic pigs as well and in peccaries this could be due to viral or host factors or potential geographic differences.

A novel papillomavirus, **Sus scrofa papillomavirus 2 (SsPV2)** has been associated with cutaneous fibropapillomas in a wild boar (Link et al., 2017). The distribution of this virus in wild suids is unknown.



**FIGURE 8.9** Porcine circovirus type 2 infection in a lymph node in a pig. (A) Sinuses contain increased numbers of histiocytes and there is diffuse lymphoid depletion. (B) Intrahistiocytic, basophilic, intracytoplasmic inclusions have a characteristic botryoid appearance.

## RNA Viruses

**Classical Swine Fever (CSF)**, also called **hog cholera**, is caused by an enveloped RNA virus of the *Pestivirus* genus (family *Flaviviridae*) and is OIE notifiable. The natural hosts for **CSF virus (CSFV)** are members of *Suidae*. The disease causes major economic losses in domestic pig populations. The Eurasian wild boar is also susceptible and plays an important role in the transmission and epidemiology of the disease in Europe (Artois et al., 2002; Rossi et al., 2005). The wild boar is not a classic reservoir but is capable of maintaining infection over long periods of time (Artois et al., 2002). Wild African *Suidae*, such as the common warthog and the bushpig have been experimentally infected (Everett et al., 2011; Gers et al., 2011).

Transmission routes are similar in the domestic pig and wild boar. The virus is spread by direct contact between infected animals and indirectly by contact with contaminated food, feces and carcasses. Naïve populations are infected accidentally by indirect or direct contact with infected domestic pigs or wild boars (Moennig, 2015). CSF outbreaks with highly virulent strains of CSFV in small populations of wild boars can be self-limiting (Artois et al., 2002; Moennig, 2015). However, there is concern in Europe about the wild boar becoming a dangerous reservoir in large populations where CSFV tends to become endemic and persist for years (Artois et al., 2002; Moennig, 2015; Penrith et al., 2011). Animal density also plays an important role in maintenance of the virus, since a higher turnover rate increases the number of younger susceptible pigs (Moennig, 2015). A study in France supported the idea that denser populations with more young favored the persistence of CSF. Additionally, populations of wild boar occupying larger forests had a higher incidence of CSF than populations occupying smaller spaces (Rossi et al., 2005).

The pathogenesis is not fully understood but involves cytopathic effects of the virus on lymphoreticular cells and macrophages, the vascular endothelium, and epithelial cells. Virus enters through mucous membranes, replicates in the tonsillar epithelium, and spreads to cervical lymph nodes. Acute infections of CSF cause hemorrhagic fever, but

subacute, chronic and subclinical forms of the disease also occur depending on virulence of the strain and the immune response of the pig (Penrith et al., 2011). Clinically, animals with acute disease show anorexia, lethargy, fever, and leukopenia (Penrith et al., 2011; Robinson and Robinson, 2015). Hemorrhages in the periphery of lymph nodes and renal petechiae are common findings. Experimental studies in common warthogs and bushpigs demonstrated intra-species transmission (Everett et al., 2011; Gers et al., 2011). Bushpigs developed similar clinical signs and lesions as domestic pigs. Consistent lesions included necrotizing and ulcerative enteritis, suppurative rhinitis and pneumonia (Fig. 8.10). Renal petechiae can also be present, similar to ASFV infection (Fig. 8.10c). Histologically, affected animals also had lymphoid necrosis and depletion. Perivascular cuffs in multiple organs were observed in surviving animals. In contrast, infected warthogs may be clinically normal. Histologically, warthogs may have rare or subtle lesions consisting of lymphoplasmacytic infiltrates in various organs and rare perivascular cuffing (Gers et al., 2011).

Control measures for domestic pigs in the European Union (EU) are based on culling infected or suspicious animals and temporary animal movement restrictions; emergency vaccinations may be employed when uncontrollable spread of CSF is suspected/occurs (Moennig, 2015). In wildlife, detection of infection is more difficult and control measures are focused on reduction of population densities, intensive diagnostics, and good hygiene practice during hunting season. In some parts of Germany, these measures have been supplemented by oral vaccination combined with hunting efforts that target young wild boars (Penrith et al., 2011).

In addition to carnivores, **canine distemper virus (CDV)** (genus *Morbivirus*) can cause encephalitis in collared peccaries (Appel et al., 1991). Clinically affected animals can appear blind, reluctant to move and develop myoclonus. Lesions are restricted to the brain with variable neuronal necrosis, astrocytosis, and perivascular cuffs. Intracytoplasmic eosinophilic inclusions are present within neurons. CDV antigen can be demonstrated within neurons by



**FIGURE 8.10** Experimental classical swine fever virus infection in a European wild boar. (A) Necrotizing tonsillitis. (B) Necrotizing enterocolitis with button ulcers due to infarction of capillaries. (C) Renal petechiae are similar in gross appearance to those in ASFV infection (see also Fig. 8.6B). (Photos Courtesy of the Friedrich Loeffler Institute)



IHC. Serologic surveys suggest peccaries are enzootically infected (Noon et al., 2003) although much is unknown about disease epidemiology. Experimental studies show peccaries as also being susceptible to another morbillivirus, **rinderpest virus (RPV)** (Dardiri et al., 1969).

**Rinderpest virus** has caused fatal disease in wild suids. Natural infection has been reported in warthogs, bush pigs (red river hogs), and giant forest hogs in Africa (Anderson et al., 1996), and been experimentally reproduced in peccaries (Dardiri et al., 1969). Lesions include extensive ulceration with overlying diphtheritic membranes in the stomach and over Peyer's patches in the small intestine and cecum, although they can occur in other areas within the gastrointestinal tract. Congestion is common in the gastrointestinal tract, kidneys, and urinary bladder. Regions of cyanosis or mucosal ulceration may be seen in the skin, tongue, trachea, and larynx. The myocardium may be pale. Rinderpest was eradicated in the wild in 2011 but is still an OIE listed notifiable disease.

**Foot and mouth disease (FMD)**, caused by an *Aphthovirus*, **foot and mouth disease virus**, is a severe, highly contagious disease of a wide variety of cloven-hoofed species including suids. The disease has been eradicated in some countries and the geographic distribution of disease has decreased; however, it is still present in South America, Africa, Asia and parts of the Middle East and Eastern Europe. There are seven different serotypes but it is unknown whether there are differences in pathogenicity among wild suids. While the virus can survive within the oropharynx in persistently infected hoofstock, viral persistence has not been shown in swine. Some serologic surveys have identified seroconversion in wild boar closely related to outbreaks in livestock while others have failed to show spill-over to wild boar populations (Alexandrov et al., 2013; Elbers et al., 2003). Classic FMD lesions are cutaneous and mucosal vesicles with ulceration. Associated clinical signs depend on site. In suidae, the most common clinical sign is lameness due to vesicles and ulceration along the coronary band between the claws (MacLachlan and Dubovi, 2011). Secondary bacterial infection is common. Vesicles also occur on the snout and to a lesser extent in the mouth. Disease in wild boars appears to be less severe than in domestic pigs based on experimental studies (Breithaupt et al., 2012).

**Ebola viruses (EBOV)**, family *Filoviridae*, can cause fatal hemorrhagic diseases in humans and nonhuman primates (Chapters 14 and 15). In 2008, investigation into an unusually severe outbreak of respiratory disease and abortions in domestic pigs suggestive of a pathogenic strain of porcine reproductive and respiratory syndrome virus identified coinfection with **Reston EBOV (REBOV)** (Barrette et al., 2009). In this initial study, REBOV antigen localized to the capsular surface of lymph nodes while PRRSV was present within germinal centers. Both viruses co-localized

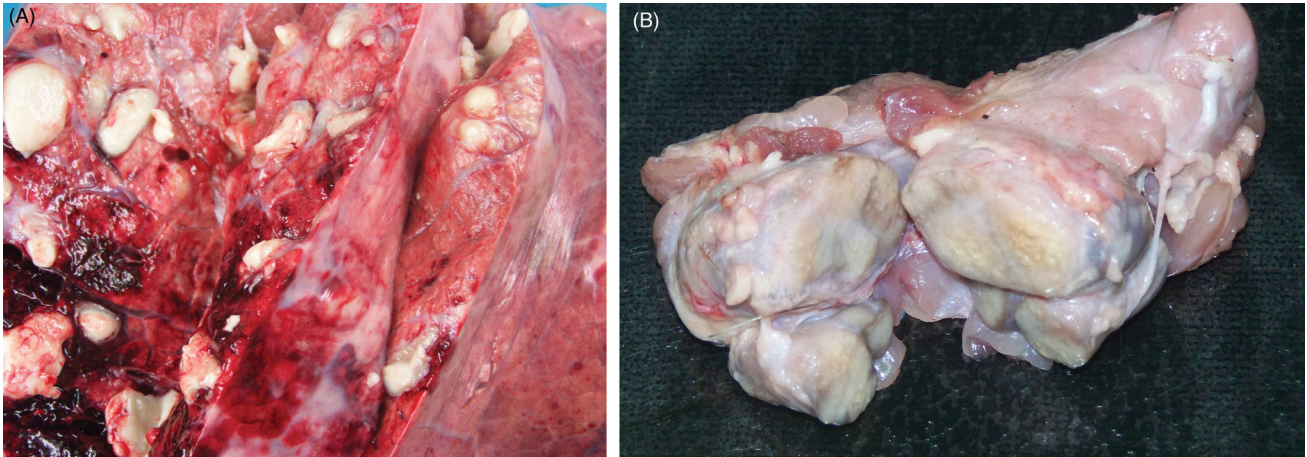
to the lung and were associated with interstitial pneumonia and necrotic debris within alveoli. Subsequent studies have found PRRSV and REBOV coinfections in other populations of domestic pigs (Pan et al., 2014). In experimental studies in pigs, Reston EBOV replicates within internal organs and can be shed from the nasopharynx without evident clinical signs (Marsh et al., 2011); experimental infection with Zaire EBOV (ZEBOV) causes severe neutrophilic and histiocytic pneumonia and respiratory distress (Kobinger et al., 2011; Nfon et al., 2013).

Fruit bats are thought to be the natural reservoir for EBOV. However, given the known susceptibility of domestic pigs to EBOV and the sympatric distribution of wild suids in regions where previous EBOV outbreaks have occurred, some have suggested suids as either an incidental host or potentially an important part of EBOV disease ecology. Serosurveys demonstrate wide variation in seroprevalence from 3% to 70% for REBOV (Sayama et al., 2012). REBOV can be transmitted from suids to humans and experimental studies suggest that suids can also transmit ZEBOV to nonhuman primates (Weingartl et al., 2012, 2013). Virus neutralization assays to test for ZEBOV have recently been optimized for use in swine (Pickering et al., 2017).

Suids are important hosts for **influenza A** viruses because they can be infected with both avian and human viruses. Coinfection increases the opportunity for viral reassortment and the development of increased pathogenicity. Confirmed infection is notifiable to OIE. Consistent with this concern is the identification in wild suids of both avian and human influenza virus infections including pandemic H1N1 2009, classical H1N1, H1N2, H3N2 (Biondo et al., 2014; Cano-Manuel et al., 2014; Cho et al., 2015; Foni et al., 2013; Kaden et al., 2008; Perera et al., 2013; Shimoda et al., 2017; Touloudi et al., 2015; Vicente et al., 2002). In one study, H1N2 virus was associated with bronchopneumonia in boars; however, it is unknown if the virus was contributory, as IHC was negative for influenza A and positive for *Mycoplasma hyopneumoniae* (Biondo et al., 2014).

## Bacteria

The *Mycobacterium tuberculosis* complex (MTC) includes mycobacteria capable of causing **tuberculosis (TB)** in countless wildlife species including suids. The wild boar is generally considered as a spillover or dead-end host for tuberculosis and similar observations have been noted in other wild suids (Corner et al., 1981; Keuling et al., 2013). Generalized lesions and mortality due to tuberculosis are low and infection usually occurs in wild boar populations in contact with livestock and wild ungulates (Keuling et al., 2013; Naranjo et al., 2008). Recent epidemiological studies show this is true for wild boar in central and northern Europe. However, the scenario differs in the Spanish Mediterranean ecosystems where evidence suggests wild boars



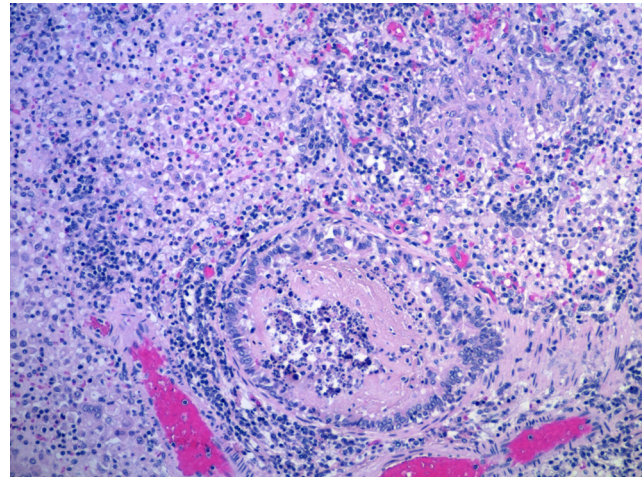
**FIGURE 8.11** Tuberculosis due to *Mycobacterium bovis* in a European wild boar. (A) Multiple pulmonary granulomas are characteristic lesions in tuberculous mycobacterial infections. (B) Granulomatous lymphadenitis. (Photos Courtesy of José Ángel Barasona García-Arévalo, SUAT-VISAVET, Department of Animal Health of the Complutense University of Madrid)

are natural reservoirs able to maintain TB infection and transmit disease to other species (Barasona et al., 2016; Martín-Hernando et al., 2007; Naranjo et al., 2008).

*Mycobacterium bovis* (bTB) and other genotypes of the MTC are prevalent in wild boar in the Spanish Mediterranean region (Barasona et al., 2016; Naranjo et al., 2008). Infection is high in dense populations and juvenile piglets play an important role in disease epidemiology. Studies show that up to one third of juvenile wild boars may become infected during the first months of life and are more likely to develop larger, generalized lesions (Barasona et al., 2016; Martín-Hernando et al., 2007). Additionally, bTB was found to be the cause of death in approximately 30% of the wild boars. The proportion of wild boars with generalized bTB lesions decreased with age as did deaths associated with bTB (Barasona et al., 2016). These results contrast with results obtained in other parts of Europe, where the death rate due to bTB is 3% (Keuling et al., 2013).

In wild boar, bTB lesions can either be localized in one anatomical region or found affecting several organ systems. In one study, head lymph nodes (LNs), particularly the mandibular, were the sites most frequently and consistently affected, often being the only site of disease. Other affected organs include lung, thoracic LNs, liver, spleen, and mammary gland (Martín-Hernando et al., 2007). Both respiratory and food-borne infections seem plausible in this species given the distribution of lesions. In most cases, mycobacteria enter the oral mucosa and disseminates to tonsils and mandibular LNs. Wild boar with extensive lesions and systemic infection are potentially capable of excreting mycobacteria through several routes (Barasona et al., 2016; Martín-Hernando et al., 2007).

Gross lesions in boar with bTB resemble those of tuberculosis in other species, and consist of focal to multifocal to miliary, variably sized granulomas (Fig. 8.11).



**FIGURE 8.12** Tuberculosis due to *Mycobacterium bovis* in a free-ranging warthog. Granulomatous pneumonia with both more diffuse granulomatous inflammation as well as organizing granulomas. (Photo Courtesy of E. Mitchell, National Zoological Gardens of South Africa)

Microscopically, granulomas are characterized by nodular aggregates of epithelioid macrophages, multinucleated giant cells, surrounded by lymphocytes, plasma cells and macrophages (Fig. 8.12). Larger or more chronic granulomas are additionally surrounded by fibrosis and contain central areas of necrosis with multifocal mineralization. Necrosis and mineralization are more extensive in generalized cases of TB, when more than one organ is affected. Acid fast bacilli are consistently found in larger numbers in pulmonary granulomas, in contrast to lymph nodes where intralésional mycobacteria are scant to absent.

Diagnosis of bTB is made by detection and identification of mycobacteria of the MTC in affected organs. Bacterial culture, molecular diagnostics, and enzyme-linked immunosorbent assays (ELISAs) are invaluable tools for



**FIGURE 8.13** *Brucella suis* epididymitis in a domestic pig. The epididymis contains numerous coalescing granulomas and granulomatous inflammation. (Photo Courtesy of T. LaBranche, University of Georgia)

diagnosis and detection of exposure (Boadella et al., 2011a; Bollo et al., 2000; Gómez-Laguna et al., 2010). Histochemical detection of acid-fast bacteria or immunohistochemical labeling of intracellular or intralesional mycobacteria within or near granulomas or necrosis, are suggestive of the disease; however, false negative results may occur when bacteria are rare. Additionally, classic and real-time polymerase chain reaction (PCR), with genome reamplification are sensitive, specific, and fast diagnostic tools for TB and are of use in the wild boar (Gómez-Laguna et al., 2010).

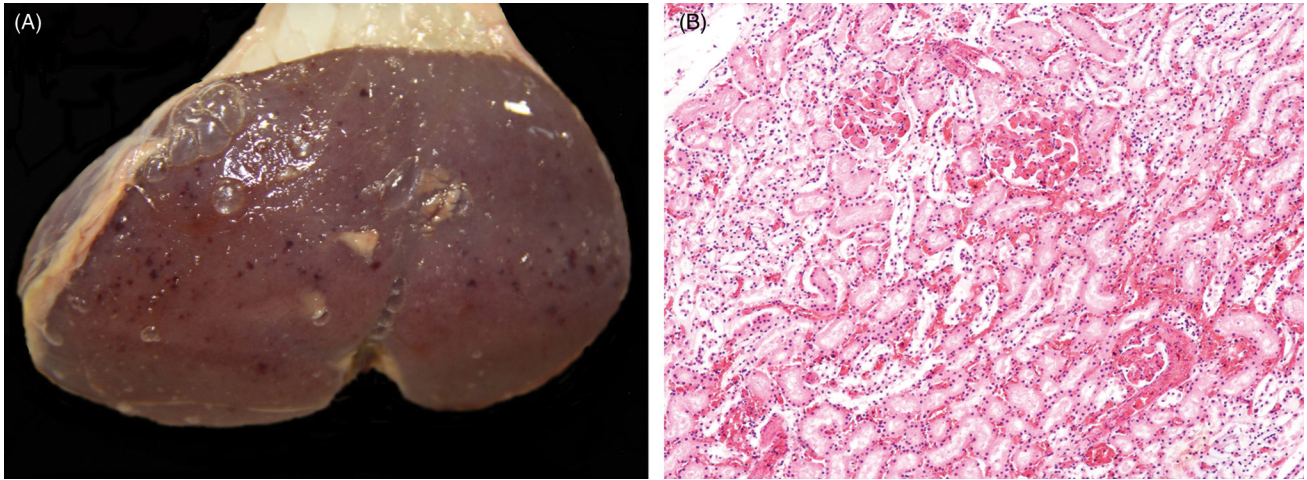
**Brucellosis** in wild swine is typically caused by *Brucella suis* although *B. abortus* and *B. melitensis* are also reported to cause mild disease. *B. suis* biovar 1 has been detected in populations of collared peccaries in Venezuela and wild boars in Croatia. In these populations, few animals develop disease but the populations are considered an important reservoir for infection. In contrast, biovar 2 is associated with disease outbreaks in European wild boars (Godfroid et al., 2013; Lord and Lord, 1991). Disease in wild swine is similar to that in domestic swine with oral or venereal transmission followed by lymphadenitis and bacteremia. Granulomatous lesions with central coagulative necrosis surrounded by suppurative exudate are typical and can be found in male and female genitalia (Fig. 8.13), bone, joints, mammary gland, lymph nodes, spleen, liver, kidney, bladder, and brain. Uterine lesions consist of focal to coalescent/miliary abscesses and granulomas in pregnant and nonpregnant endometrium and placenta. Histologically, uterine glands are dilated and filled with neutrophils and mucus, and there is a loss of endometrium. The fetal placenta is congested and there is multifocal to coalescing hemorrhage, edema, and a mucopurulent exudate with numerous bacteria. Aborted fetuses are usually autolyzed with serosanguinous fluid in body cavities, subcutaneous tissues,

and abnormal stomach content. Vertebral lumbar osteomyelitis and fibrinopurulent synovitis are common lesions in juveniles and in chronic disease. Serological cross-reaction has been reported with *Yersinia* O:9, *Escherichia coli* O:17, and some serobiovars of *Salmonella*, which complicates diagnosis based solely on serology (Godfroid et al., 2013; MacMilan, 1999; Schlafer and Foster, 2016).

*Erysipelothrix rhusopathiae* is a Gram-positive bacillus with a wide geographic distribution and host range. It is the cause of porcine erysipelas and is capable of causing outbreaks in other domestic and wild animals. Pigs are a reservoir of infection, particularly for other pigs, since *E. rhusopathiae* can persist for long periods in lesions and persists in the tonsils, bone marrow, and intestine of healthy swine (Robinson and Robinson, 2015). Aggregation and high wild boar density can influence spread (Boadella et al., 2011b; Risco et al., 2011; Ruiz-Fons, 2017). Seroprevalence has been detected in wild boars in Japan and coastal Mediterranean countries, such as Greece and Spain; it is also likely to be present in other populations of wild boars (Closa-Sebastia et al., 2011; Marinou et al., 2015; Risco et al., 2011; Shimizu et al., 2016; Vicente et al., 2002).

Porcine erysipelas occurs in swine of all ages but young animals (up to 12 months) and pregnant sows are most susceptible. Disease can be acute, causing septicemia and death. Mild and chronic forms are characterized by skin necrosis, endocarditis, and polyarthritis (Robinson and Robinson, 2015). Gross lesions in acute or chronic disease include multifocal to diffuse petechiae, erythema, and/or cyanosis. Histologically, vasculitis with microthrombi, intralesional bacteria and associated leucocytic infiltrates, mural fibrinoid necrosis, and hemorrhage occur throughout the body but are most remarkable in the kidneys, urinary bladder, and skin (Fig. 8.14). Multifocal mononuclear infiltrates may be seen in the kidneys (Risco et al., 2011), and lymphoplasmacytic infiltrates, microthrombi as well as alveolar edema and congestion may be seen in alveolar septae. Corneal edema and panuveitis with fibrinoid vasculitis has also been described. Lesions in wild boar reported by Risco et al. (2011) were similar to those of porcine erysipelas and erysipelas in other species. Septicemia in wild boars due to erysipelas has also been reported in Japan (Yamamoto et al., 1999).

*Brachyspira* are Gram negative, anaerobic spirochetes, adapted to occupy specialized niches in the large intestines of animals, including swine. Two important entities in domestic swine are caused by *Brachyspira hyodysenteriae* and *B. pilosicoli*, known as **swine dysentery (bloody scours)** and **porcine intestinal spirochetosis**, respectively (Hampson, 2012). *B. hyodysenteriae* is a highly hemolytic bacterium, capable of causing episodes of mucohemorrhagic diarrhea with a serious impact in morbidity and mortality, and thus the economy of domestic swine herds. Information about *Brachyspira* infection in wild boars



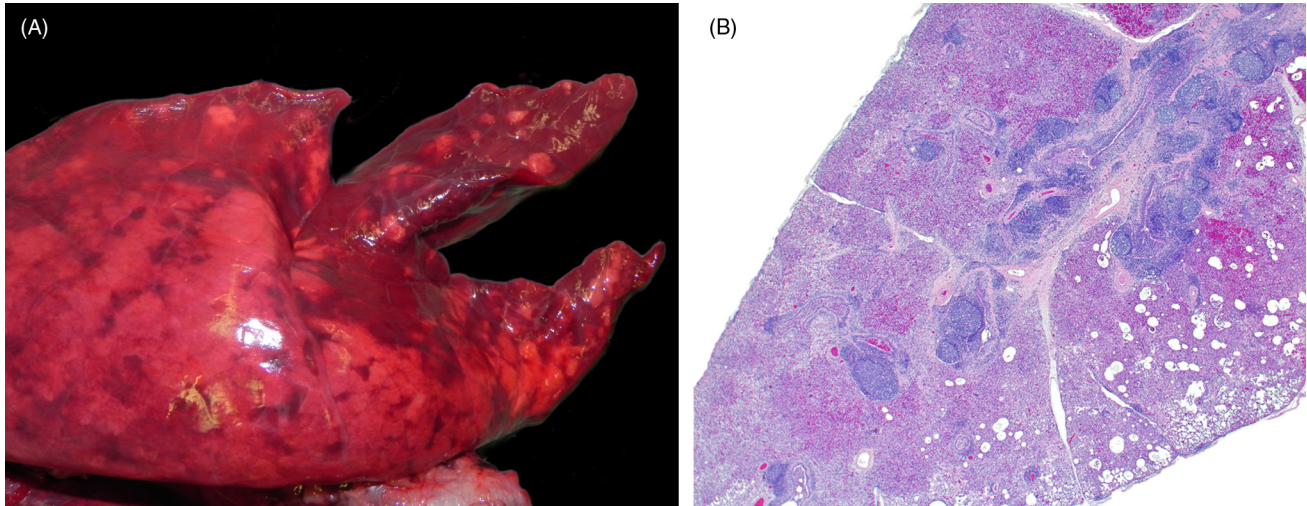
**FIGURE 8.14** *Erysipelothrix rhusopathiae* nephritis in a wild boar. (A) Renal petechiae cannot be differentiated from petechiae due to other causes in suids. (B) Hemorrhage is present in the renal interstitium and in glomeruli. Proximal tubules adjacent to hemorrhage are degenerate and necrotic due to ischemia. Glomerular capillaries contain fibrin thrombi. (Photos Courtesy of David Risco Perez, INGULADOS S.L.)

or in other wild suid genera is limited. Several studies in free-ranging wild boar in Sweden and Spain failed to demonstrate *Brachyspira* in fecal samples, which seemed to indicate that these spirochetes are not a natural part of the intestinal flora (Jacobson et al., 2005; Osorio et al., 2013; Vadillo et al., 2017). However, in one study in Spain that included 40 wild boars from several intensive growth farms, *B. intermedia*, *B. pulli*, and *B. hyodysenteriae* were isolated and associated with clinical disease in 3 animals (Vadillo et al., 2017). In addition to documenting infection, this study also supported the hypothesis that weakly hemolytic species, such as of *B. intermedia* and *B. pulli* have pathogenic potential in nondomestic suids. Details of the lesions in wild boar were not specified. Several species of *Brachyspira*, including a highly hemolytic strain of *B. hyodysenteriae*, have also been identified in feral pigs in Western Australia (information on associated disease or lesions has not been described) (Phillips et al., 2009).

*Mycoplasma hyopneumoniae* is the cause of mycoplasmal pneumonia, also known as **enzootic pneumonia**, of swine. *M. hyopneumoniae* is a small bacterium that lacks a cell wall and adheres to the ciliated epithelium of large airways and to a lesser extent, the bronchioles. Most mycoplasmas contain superantigens in their cell membranes that induce polyclonal lymphoid proliferation and characteristic lymphoid aggregates adjacent to airways (Caswell and Williams, 2016). Seroprevalence of *M. hyopneumoniae* has been reported in wild boars in several parts of Europe, such as Switzerland, Greece, and Spain (Chiari et al., 2013; Kuhnert and Overesch, 2014; Kuhnert et al., 2011; Sibila et al., 2010; Vengust et al., 2006). Most of these studies have not associated serology with disease. However, in one comprehensive study on the prevalence of *M. hyopneumoniae* in wild boars in Switzerland, mild to moderate lymphoplasmacytic and histiocytic bronchointerstitial pneumonia, primarily

affecting the cranioventral region of the lung, was identified (Fig. 8.15A) (Batista Linhares et al., 2015). Lesions were consistent with the acute and chronic stages of the disease described in domestic pigs. These include acute disease with multilobular, patchy red to dark red areas affecting less than 50% of the lungs versus more chronic lesions in which a higher percentage of the pulmonary parenchyma is affected by dark red to gray, atelectatic areas. Histologically, infected boars had hyperplastic bronchial associated lymphoid tissue (BALT) with reactive lymphoid follicles, interstitial infiltrates of plasma cells; lymphocytes and histiocytes, alveolar and bronchial neutrophilic infiltrates, and alveolar edema were also, but less commonly, seen (Fig. 8.15B). The amount of inflammation, number of neutrophils, and hyperplastic BALT increased with the chronicity. Juvenile and young adult boars are more susceptible to infection, which corresponds to serological reports of wild boars in Italy, and also to the age range of susceptibility in the domestic pig. While wild boars are frequently viewed as the reservoir for disease in domestic pigs, in this study it was instead more likely for wild boars to become infected by contact with domestic pigs. Based on this, the role of wild boars as reservoirs for *M. hyopneumoniae* transmission to domestic pigs is considered minor.

Several species of *Chlamydophila* have been reported both serologically and histologically in wild boars (Cano-Manuel et al., 2014; Hotzel et al., 2004); however, its significance as a pathogen is uncertain. In one study *C. psittaci* was the most prevalent species; *C. abortus* and *C. suis* were also identified but less commonly. Chlamydial inclusions in the lung were present in smooth muscle cells around bronchioli, the endothelium and interstitium of alveolar walls as well as the myometrium of the uterus (Hotzel et al., 2004). The significance of chlamydial infection in wild boars is currently uncertain.



**FIGURE 8.15** *Mycoplasma hyopneumoniae* infection (enzootic pneumonia) in the lungs of a wild boar. (A) Infection causes bronchopneumonia with consolidation that often has a cranioventral pattern. Affected parenchyma will be firm, often discolored dark red, and demarcation between affected and less or unaffected adjacent tissue may be apparent. (B) Infection is airway oriented and regionally, the airways and the alveolar interstitium contain numerous inflammatory cells. Bronchial associated lymphoid tissue is hyperplastic. With chronicity, the severity of infection increases. (Photos Courtesy of the Centre for Fish and Wildlife Health, University of Bern)

**Clostridia** are large, spore forming, strictly anaerobic to oxygen-tolerant anaerobic bacteria. *Clostridium perfringens* types A and C, and *C. difficile* are the principal enteric clostridial pathogens of domestic swine. All subtypes of *C. perfringens* can be commensals in the intestines of many animal species; therefore, diagnosis must be made by detecting the toxin in association with consistent lesions and in the absence of other potential pathogens.

*C. perfringens* type A is part of the normal intestinal flora in pigs but can cause enteric disease in neonatal and occasionally weaned pigs. The pathogenesis of *C. perfringens* type A in pigs is poorly understood but it is likely to be multifactorial. *C. perfringens* is a known cause of enteritis in a variety of wild animal species and has been reported in wild suidae, such as wild boar and pygmy hogs (Das et al., 2008; Glenn Songer and Uzal, 2005; Li et al., 2017; Shome et al., 2010). In an outbreak occurring in wild boar in China, *C. perfringens* type A and C were isolated by serological and molecular assays, and found to be the cause of death. Gross lesions consisted of extensive hemorrhage in lungs, intestine, and inguinal lymph nodes (Li et al., 2017). Hemorrhagic enteritis associated with *C. perfringens* type A has also been reported in a research and breeding center for pygmy hog in Assam, India. Lesions were similar to those reported in the domestic pig (Shome et al., 2010). Hemorrhagic and necrotizing enteritis with diffuse small and large intestinal edema occurred in another adult, female pygmy hog at the Assam State Zoo, in Assam, India. Molecular assays confirmed *C. perfringens* type A infection (Das et al., 2008). *C. perfringens* type C occurs worldwide and causes necrotizing and hemorrhagic enteritis that is often fatal in domestic swine. Hallmark histologic lesions are deep mucosal necrosis and hemorrhage with emphysema in

small intestine that sometimes also affects the colon. It is a primary pathogen and usually causes more severe disease in young animals.

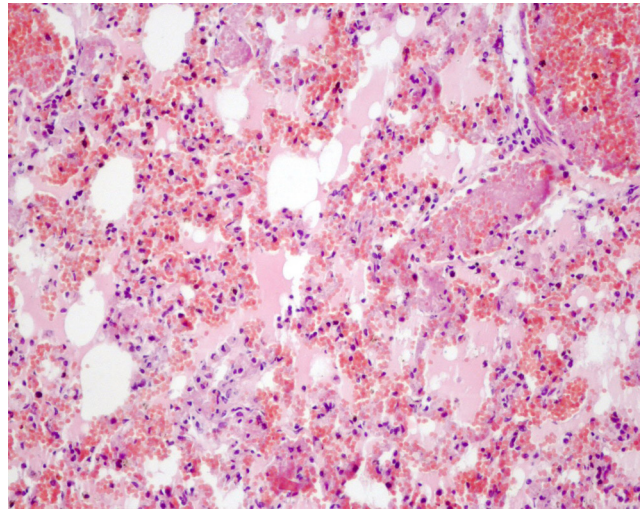
*C. difficile* is a cause of enteritis in neonatal pigs that usually develops between 1 and 7 days of age. Hallmark lesions include edema of the mesocolon, suppurative inflammation of the colonic lamina propria, and mesenteric lymph node edema (Glenn Songer and Uzal, 2005). *C. difficile* has been isolated from feral pig feces in areas with intensive commercial swine production in Canada, and feral pigs could be reservoirs (Thakur et al., 2011).

Wild suids are important reservoirs for *Salmonella* species and infection is reported worldwide, particularly in wild boars and pygmy hogs. Wild boars may be asymptomatic carriers and maintenance reservoirs in areas with sufficient boar density (Cano-Manuel et al., 2014; McGregor et al., 2015). Transmission occurs directly or indirectly by contact with contaminated water, food, feces, or other infected individuals. Several acute, fatal outbreaks have occurred in both adult and young (<1 year of age) pygmy hogs (Rahman et al., 2001). Only adults showed clinical signs, which included generalized loss of body condition, anorexia, high fever, loose feces that contained mucus and blood, weakness, and tremors. All animals had necrohemorrhagic enterocolitis with multifocal, discrete areas of necrosis in the cecum and colon. Additionally, the liver, epicardium, and pericardium contained areas of hemorrhage, and the spleen was enlarged. Cerebral congestion was also a consistent feature. **Enterotoxigenic *Salmonella* Enteritidis (*Salmonella enterica enteritidis*)** that carried fimbrial genes for colonization of enterocytes was isolated in other pygmy hog outbreaks, *Salmonella typhimurium* has been isolated (Rahman et al., 2005).

*Haemophilus parasuis*, the cause of **Glasser's disease** or porcine polyserositis and arthritis syndrome, causes fibrinosuppurative inflammation of one to several serosal surfaces and joints; it may also cause pneumonia or meningoencephalitis (Craig et al., 2015). Antibodies to *H. parasuis* have been reported in wild boars in Slovenia, Spain, and Germany (Olvera et al., 2007; Reiner et al., 2010b; Vengust et al., 2006), but information on disease development in this species or other wild suids is limited. Fatal infections have been reported in free-ranging wild boars in central Spain. Clinical signs included respiratory distress and weakness; however, there was only one documented fatality suggesting a low mortality rate (Cuesta Gerveno et al., 2013). The one young boar that died had a fibrinous bursitis, interstitial bronchopneumonia, interstitial nephritis with hemorrhage, and mild meningitis. *H. parasuis* was cultured and confirmed with molecular methods. The polyserositis and fibrinous serosal exudates usually associated with infection in domestic pigs were lacking in the boar (Craig et al., 2015; Cuesta Gerveno et al., 2013).

*Pasteurella multocida* is a Gram-negative bacillus with five known serovars, A, B, C, D, and E. Coinfection with toxin producing strains of *P. multocida* and other bacterial pathogens are the cause of **progressive atrophic rhinitis (PAR)** in domestic pigs and sporadic outbreaks of septicemia can occur. The disease usually affects 6–12 week-old pigs and causes sneezing, nasal discharge, unilateral epistaxis, and nasal deformity (Caswell and Williams, 2016). Serovars A and D are known causes of pneumonia in weaner and finisher pigs, but are rarely primary pathogens. Predisposing factors, such as environmental conditions or coinfection with *Mycoplasma hyopneumoniae*, porcine herpesvirus 1, or other respiratory pathogens are considered necessary for disease development in domestic pigs (Caswell and Williams, 2016; Register et al., 2012). Serologic evidence of *P. multocida* infection without disease has been reported in peccaries (Martins Gomes de Castro et al., 2014). However, both PAR and systemic pasteurellosis have been reported in wild boars (Kaden et al., 2001; Risco et al., 2013). In cases of systemic pasteurellosis, boars were found dead with epistaxis but had no clinical signs prior death. Gross lesions consisted of subcutaneous edema in the ventral region of the neck, generalized congestion, and fibrin within the peritoneal cavity. Histologically, multiple organs were congested and multifocal hemorrhage and thrombosis were seen (Fig. 8.16).

*Streptococcus suis* is an important bacterial pathogen in swine that has worldwide distribution. Infection causes a variety of diseases including sepsis, meningitis, polyarthritis, bronchopneumonia, and endocarditis. Over 30 serotypes are recognized, but serotypes 1 and 2 are generally of greater significance in domestic pigs. Serotype 1 infects suckling pigs and is not pathogenic for other species, while serotype 2 causes disease in weaner and feeder



**FIGURE 8.16** Systemic pasteurellosis in a wild boar. Several capillaries are occluded by fibrin thrombi. Alveolar septae are hemorrhagic and capillaries contain fibrin, neutrophils, and numerous erythrocytes. Alveolar spaces are filled with edema fluid. (Photo Courtesy of David Risco Perez, INGULADOS S.L.)



**FIGURE 8.17** *Streptococcus suis* in a wild boar. Acute meningitis and hemorrhage with meningeal vascular congestion. (Photo Courtesy of David Risco Perez, INGULADOS S.L.)

pigs and humans. Serotype 7 can also be zoonotic (Craig et al., 2015). Feral swine and wild boars are known carriers of several *S. suis* serotypes, some of which are potentially zoonotic (Baroch et al., 2015; Baums et al., 2007; Sánchez del Rey et al., 2014; Seol et al., 1998; Risco et al., 2015). Several cases of *S. suis* infection in hunters in contact with wild boars have occurred (Baums et al., 2007). However, not much is known about the clinical and pathological significance of infection in wild boars. A single case of fatal *S. suis* serotype 2 infection was reported in a wild boar in Spain. The animal was young (approximately 2 weeks old) and had suppurative bronchopneumonia and meningitis with hemorrhage (Fig. 8.17), which is similar to lesions that

are described in domestic pigs (Risco et al., 2015). Other organs, such as the kidneys had congested vessels, microthrombi, and hemorrhages, consistent with septicemia.

## Fungi

*Coccidioides immitis* is a dimorphic fungus that is endemic to the southwestern United States (US), Northern Mexico, and some countries in Central and South America. **Coccidioidomycosis** has been reported in collared peccaries but, despite the species rooting behavior and the range overlap with the agent, few exposed individuals develop clinical signs and lesions. Nodular to milliary pyogranulomatous or granulomatous pneumonia is most common, but lesions may be multisystemic and involve regional lymph nodes. Microscopically, necrotic foci contain fungal spherules in different stages of maturation and are surrounded by pyogranulomatous or granulomatous inflammation that matures into organized granulomas or pyogranulomas. Intralesional basophilic, 10–80 µm diameter, round to oval and double contour walled spherules with multiple, round to oval, 2–5 µm, basophilic, PAS and GMS positive endospores supports the diagnosis. Infection has also caused a neurological-like disorder (incoordination, muscle tremors, and convulsive seizures) in the absence of gross or microscopic brain lesions in Texan Peccaries (Fowler, 1996; Lochmiller et al., 1985). High antibody titers may be suggestive of active disease. Mature sporangia may be up to 200 µm diameter, a feature that is helpful in differentiating them from blastomycosis and histoplasmosis (both are smaller fungi) (Caswell and Williams, 2016).

## Metazoa

A wide variety of parasites have been described in free-ranging suids. Many of these are important pathogens of domestic swine but appear to be asymptomatic in wild suids. Supplemental Table e2 contains additional parasites known to infect wild suids.

Gastric metazoa include *Hyostrongylus rubidus*, a trichostrongylid nematode that infects wild boars and warthogs, and *Ascarops strongylina*, a spirurid nematode that infects wild boar and pygmy hogs (Belem, 2012; Fernandez-de-Mera et al., 2003; Kakati et al., 2015; Mansouri et al., 2016). The life cycle for both is indirect and they utilize an arthropod intermediate host (dung beetle for *Ascarops*). *Hyostrongylus* burrows into the stomach wall causing ulceration and gastric nodules with increased mucus production while *Ascarops* causes a catarrhal gastritis. Hemorrhage due to infection with either parasite may cause anemia, anorexia, and melena. Diagnosis is by identification of eggs in feces or examination of mucosal scrapings at necropsy for immature larvae.

The most important small intestinal parasite is *Ascaris suum*, which has been identified in wild boars and collared

peccaries (Fernandez-de-Mera et al., 2003; Mansouri et al., 2016). Outbreaks have been reported in captive peccaries with clinical signs of respiratory distress and coughing. This nematode parasite has a direct life cycle. Pigs are infected by ingesting eggs with second stage larvae or infected paratenic hosts (e.g., earthworms). The larvae migrate to the liver via the portal circulation, molt into third stage larvae that then travel via the venous system to the lungs where they are coughed up, swallowed, and mature into adults. Visceral migration of larvae can be associated with mucosal necrosis, hemorrhage, healing fibrosis, and lymphoplasmacytic inflammation (white areas called “milkspots”); heavy larval infection in the lungs is associated with pulmonary edema and acute necrosuppurative pneumonia. Diagnosis is by identification of eggs or worms in feces or by the presence of larvae within the lungs.

*Globocephalus* sp. are globally distributed hookworms. *G. urosubulinatus* is common in domestic pigs in the western hemisphere and Europe; it has also been reported in free-ranging feral populations of wild boars and peccaries. *G. versteri* more commonly occurs in African bushpigs, and *G. samoensis* and *G. longemucronatus* are more common in wild boars in southeast Asia and Japan (Fernandez-de-Mera et al., 2003; Seguel and Gottdenker, 2017; Van Wyk and Boomker, 2011). The life cycle is direct. Following larval ingestion or percutaneous infection, migration can be extensive and cause myocardial and or pulmonary inflammation before returning to the gastrointestinal tract. The adults are hematophagous and utilize specialized mouthparts to adhere to and cause ulceration of the small intestinal mucosa resulting in enteritis and blood loss. Diagnosis is by the identification of eggs in the feces by flotation.

*Strongyloides ransomi* infects the intestine of wild boars and is pathogenic in suckling piglets. Vertical transmission occurs during suckling after which the parasites tunnel into base of small intestinal villi resulting in blood loss, anemia, and diarrhea in piglets. Infection in adults is asymptomatic. Diagnosis is most commonly made by identification of eggs in feces collected from the rectum, mucosal scrapings at necropsy, or recovery of immature worms using the Baermann technique.

*Macrocanthorhynchus hirudinaceus*, the thorny-headed worm, infects the ileum and caudal small intestine of wild boar (Fernandez-de-Mera et al., 2003; Mansouri et al., 2016; Sutherland-Smith, 2015). The life cycle of this nematode parasite is indirect with arthropod intermediate hosts. While eggs are passed in the feces, diagnosis via flotation is difficult because the eggs do not float. Infection may cause multifocal chronic granulomatous enteritis with the formation of intramural granulomas that can be identified along the serosal surface. Nodules rarely perforate.

**Onchocerciasis** dermatitis and lymphadenitis has been described in free-ranging suidae in many countries; however, descriptions are largely limited to parasite morphology

without description of gross or microscopic lesions (Uni et al., 2001, 2015). Transmission is typically mechanical via arthropod vectors including Simulium flies. In *O. dewittei japonica* infection of Japanese wild boars, adult filarial worms are encysted in fibrous subcutaneous nodules in the distal limbs and the fat bodies of the foot pads. Microfilariae are more commonly found in the skin of the ears, back, tail, and less frequently on limbs. In contrast, *O. tacaokai* microfilariae and filarial adults infest the skin of ears, neck, and back; these adult parasites are not associated with cutaneous nodules. Coinfection with *O. dewittei japonica* and *O. tacaokai* has been reported in Japanese wild boars. In *O. ramachandrini* infection of African warthogs, microfilariae are present in skin of the neck, back, and feet, and filarial adults are found coiled in the subcutis of the distal limbs. Diagnosis is by identification of larvae or adults in skin sections. Larvae will emerge from sections of skin placed into saline.

**Trichinellosis** is OIE listed, reportable disease with worldwide distribution. While several species of *Trichinella* may infect wild suids, *T. spiralis* and *T. pseudospiralis* are most commonly reported (Nöckler et al., 2006; Zamora et al., 2015). No gross lesions and only mild histologic inflammation has been noted in infected wild boars, and infection is of importance because of the zoonotic potential. Real-time PCR of muscle tissue is a sensitive diagnostic tool (Guenther et al., 2008).

## Protozoa

**Trypanosomiasis** is recognized as a clinically significant vector-borne, protozoal disease of humans and livestock, with distinct agents and disease entities recognized in Africa and Central and South America. Subclinical infection may be regionally common in free-ranging *Suidae*, and warthogs, bushpigs, red river hogs, giant forest hogs, peccaries, and wild boars are important in the epidemiology due to the preference of insect vectors to feed on them. These suid species are reservoirs for trypanosomes pathogenic to humans and domestic animals including *T. congolense*, *T. simiae*, *T. suis*, *T. evansi*, and *T. brucei*. Warthogs are probably the main wild reservoirs of *T. simiae*, which causes acute fulminating infection in domestic pigs in Africa; peccaries are important in the maintenance of *T. cruzi* and *T. evansi* in Brazil; and red river hogs are important in the epidemiology of human sleeping sickness in Africa caused by *T. brucei* (Claxton et al., 1992; Herrera et al., 2008; Leak 1999; Pollock, 1982).

Suids generally do not develop severe disease as a result of *Trypanosome* infection but they can maintain parasitemia for a long period of time (Claxton et al., 1992). In white lipped peccaries, *T. evansi* causes severe anemia, weight loss, and immunosuppression that predisposes to opportunistic infections. In juvenile peccaries, disseminated intravascular coagulation develops secondary to the deposition

of immune complexes in the heart, liver, brain and kidneys (Herrera et al., 2008). Encephalitis due to *T. suis* infection has been reported in some species (Leak, 1999). Disease severity in suids is variable with *T. brucei* and *T. congolense* typically only causing mild disease, and *T. suis* and *T. simiae* often causing more clinically significant disease in suids (Pollock, 1982). Presence of sarcocysts in the myocardium with or without mild myositis and inflammation can occur with *T. cruzi* (Comeaux et al., 2016). Intralesional trypanosomes, hemorrhages and extravascular hemolysis may be seen in some organs. The splenic parenchyma may contain large numbers of histiocytes with marked periarteriolar sheath lymphoid hyperplasia, lymphoid depletion, and/or hemosiderosis due to extravascular hemolysis. Cerebral endothelial hypertrophy and intralesional organisms may also be present.

Free-ranging wild boars, warthogs, and bushpigs are a reservoir for *Babesia trautmanni* and *B. perroncitoi*; however, clinical diseases are best described in wild boars. *Babesia* spp. are typically transmitted between *Suidae* and *Tayssuidae* by ticks in the genera *Rhipicephalus*, *Boophilus*, and *Dermacentor*. *B. trautmanni* in domestic pigs and wild boars results in a chronic hemolytic disease with clinical signs including emaciation, pyrexia, anorexia, icterus, and hemoglobinuria. Gross lesions may be limited to poor body condition or emaciation, splenomegaly, and hepatomegaly, with or without polycavitary effusion, and multisystemic hemorrhages. Microscopic lesions reflect hemolytic disease with hemoglobin nephrosis, and degeneration and necrosis with a pattern suggestive of hypoxia in multiple tissues. Diagnosis is based on identification of the parasite in erythrocytes. *B. gibsoni*-like organisms have been detected by PCR in wild boar in Asia, but have not yet been linked to disease (Masatani et al., 2017).

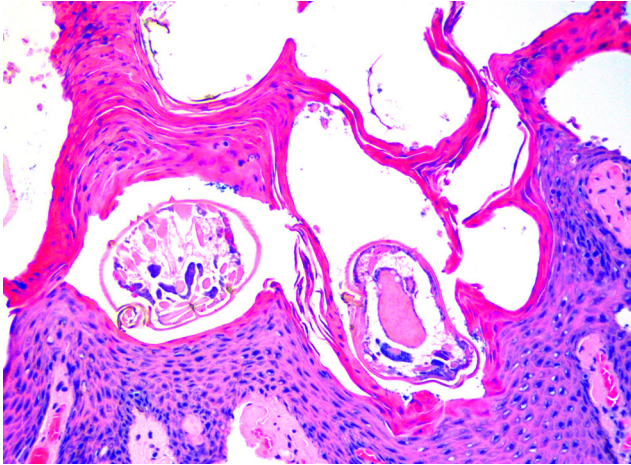
*Cryptosporidium suis* and *C. scrofarum* are ubiquitous in domestic pigs and wild boar, and all *Suidae* and *Tayssuidae* appear to be susceptible to infection (Fowler, 1996; Kaupke et al., 2017; Kvac et al., 2013; Nemejc et al., 2013; Rodriguez-Rivera et al., 2016; Ryan et al., 2004). *Cryptosporidium* spp. have a direct life cycle, and while most naturally occurring infections are subclinical, infection has infrequently been associated with mild enteritis and diarrhea in suids. The protozoa are most commonly identified in young animals; adults appear to have a moderate degree of resistance to infection. Zoonotic transmission is the most significant feature of infection in these species, and transmission to human and nonhuman primates by wild boar, warthogs, and bushpigs is a primary concern (Parsons et al., 2015).

Additional pathogenic and commensal protozoa are presented in Supplemental Tables e2 and e3.

## Ectoparasites

*Sarcoptes scabiei* var *suis*, the cause of **sarcoptic mange**, are global pests of domestic pigs that have also been





**FIGURE 8.18** Scabies due to *Sarcoptes scabiei* infection in the skin of a babirusa. Marked epidermal hyperplasia and hyperkeratosis are associated with the intracorneal mites. (Photo Courtesy of E. Mitchell, National Zoological Gardens of South Africa)

reported in free-ranging wild boars, warthogs, and peccaries (Fowler, 1996; Haas et al., 2015). Lesions are similar to those described in other species and include alopecia and crusting dermatitis with lichenification that primarily affects the ventrum and extremities. Histologically, parakeratotic hyperkeratosis with intracorneal mites and serocellular crusts are common findings (Fig. 8.18). Dermal inflammation is primarily eosinophilic. Diagnosis is by skin scrape.

Free-ranging *Suidae* and *Tayassuidae* are hosts to numerous tick species, and infestations are not uncommon in captive animals. In free-ranging animals, tick burdens may be extremely high, and most *Suidae* and *Tayassuidae* have developed behavioral adaptations, such as wallowing that rubs ticks off of the skin and commensal relationships with other animals that scavenge ticks. Tick infestation is most commonly associated with chronic multifocal dermatitis; it is not generally associated with systemic disease. Ticks are more important due to their roles as vectors for infectious disease transmission, such as in the pathogenesis of ASF.

Additional information on ticks and other ectoparasites is provided in Supplemental Table e2.

**E-SLIDES**

- 8.e1** **Cystic and adenomatous endometrial hyperplasia, babirusa, uterus.** The endometrium is moderately, multifocally expanded by numerous endometrial glands, some of which are mildly to moderately dilated. (see [Fig. 8.3](#)). eSlide: [VM05031](#)
- 8.e2** **Uterine adenocarcinoma, Ossabow Island hog, uterus.** An unencapsulated, non-demarcated proliferation of neoplastic cells and glandular or tubular structures that is supported by a dense neoplastic stroma expands the uterine wall. Neoplastic cells are polygonal to columnar, multifocally piled up, show mild to moderate anisocytosis and pleomorphism, have a prominent nucleolus to granular chromatin, and contain abundant lightly basophilic cytoplasm. Mild multifocal lymphoplasmacytic inflammation and hemorrhage are seen throughout the tumor. eSlide: [VM05074](#)

## E-ONLY CONTENTS

**TABLE e1** Additional Infectious Diseases of Domestic Suids and Evidence for Infection in Wild Suids

Pathogen	Disease in Domestic Pig	Wild Host	Disease in Wild	Evidence in Wild Suids
<i>DNA Virus</i>				
Porcine parvovirus	Stillbirth, abortion, fetal death, and loss	Bushpig, Wild boar	None described	Metagenomic (Blomström et al., 2012); Serology (Vengust et al., 2006; Montagnaro et al., 2010)
Torque teno sus virus	Uncertain; possibly increases pathogenicity of porcine circovirus	Bushpig, Wild boar	None described	Metagenomic (Blomström et al., 2012); PCR (Martínez et al., 2006)
<i>RNA Virus</i>				
Hepatitis E	Unknown	Wild boar	None described; zoonotic concerns	Serology and PCR (Sonoda et al., 2004; Michitaka et al., 2007; de Deus et al., 2008; Kaba et al., 2010)
Transmissible gastroenteritis virus (coronavirus)	Gastroenteritis, diarrhea	Unknown	None described	
Porcine respiratory coronavirus	Interstitial pneumonia, mild	Wild boar	None described	Serology (Vengust et al., 2006)
Porcine epidemic diarrhea virus (coronavirus)	Gastroenteritis, watery diarrhea, vomiting	Wild boar	None described	Serology (Lee et al., 2016)
Porcine reproductive and respiratory syndrome virus (arterivirus)	Subclinical to abortions, stillbirths, and cyanosis of ears and vulva in adults; pneumonia in neonatal pigs	Wild boar	None described	Serology (Montagnaro et al., 2010; Choi et al., 2012); Serology and RT-PCR (Stankevicius et al., 2016)
Vesicular stomatitis virus ( <i>Rhabdoviridae</i> ; vesiculovirus)	Vesicular lesions along the snout and coronary bands	Feral swine, peccaries	None to mild similar to domestic (Dardiri, 1969)	Serology (Fletcher et al., 1985; Stallknecht et al., 1986; Corn et al., 1987; Stallknecht et al., 1993)
<i>Bacteria</i>				
<i>Lawsonia intracellularis</i>	Proliferative enteritis	Wild boar	None described	Serology and/or PCR (Tomanova et al., 2002; Dezorzova-Tomanova et al., 2006; Yeh et al., 2014)
<i>Leptospira interrogans</i> (multiple serovars)	Often subclinical; can cause sepsis, nephritis, hepatitis, meningitis, and abortion	Peccaries, Wild boar	None described	Serology (Clark et al., 1983; Corn et al., 1987; Vale-Gonçalves et al., 2015; Zmudzki et al., 2016; Ruiz-Fons, 2017)
Enterotoxigenic <i>Escherichia coli</i> (Shiga-toxin and O157 strains)	Neonatal and postweaning diarrhea	Wild boar	None described	Culture (Miko et al., 2009; Wahlstrom et al., 2003; Wacheck et al., 2010; Ruiz-Fons, 2017)

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**TABLE e2** Additional Parasites of Suids

Pathogen	Disease in Domestic Pig	Wild Host	Disease in Wild	Evidence in Wild Suids
<b>Protozoan Parasites</b>				
<b>Microsporidia</b>				
<i>Enterocytozoon bieneusi</i>	Infection rates are highly variable in domestic pigs. Infection is typically subclinical. Zoonotic risk	Wild boar, warthogs, Visayan warty pigs	Subclinical, zoonotic risk.	Fecal PCR (Nemejc et al., 2014; Slodkowitz-Kowalska et al., 2007).
<i>Encephalitozoon cuniculi</i>	Infection rates are highly variable in domestic pigs. Infection is typically subclinical.	Wild boar	Subclinical.	Fecal PCR. (Nemejc et al., 2014; Slodkowitz-Kowalska et al., 2007).
<i>Encephalitozoon intestinalis</i>	Rarely identified in domestic pigs. Infection is typically subclinical.	Wild boar	Subclinical.	Fecal microscopy, fecal PCR (Nemejc et al., 2014; Slodkowitz-Kowalska et al., 2007).
<i>Neospora caninum</i>	Natural disease not reported in domestic pigs.	Wild boar, warthogs	Disease not reported	Serology. (ELISA, IFAT) (Buxton et al., 2002; Donahoe et al., 2015; Reiterova et al., 2016).
<b>Sarcosporidia</b>				
<i>Sarcocystis dubeyella</i>	Infrequently reported in domestic pigs. Typically, subclinical. White-tan parasitic cysts up to 12 mm in length and 1 mm wide within skeletal and cardiac muscle. Cyst encapsulated by host cell membrane.	Warthogs	Subclinical. Similar to domestic pigs. *Ddx <i>Cysticercus cellulosae</i> / <i>Taenia solium</i> .	Histopathology (Junker et al., 2015; Stolte et al., 1998).
<i>Sarcocystis phacochoeri</i>	Not reported in domestic pigs.	Warthogs	Subclinical. White-tan parasitic cysts up to 1–4 mm length 0.2 mm width in skeletal and cardiac muscle. Not encapsulated by host cell membrane. *Ddx <i>Cysticercus cellulosae</i> / <i>Taenia solium</i> .	Histopathology (Junker et al., 2015; Stolte et al., 1998).
<i>Sarcocystis</i> spp.	Common in domestic pigs. Generally nonpathogenic. Parasitic cysts within skeletal and cardiac muscle. DDx for cysicercosis.	Wild boar	Parasitic cysts within the right ventricle.	Histopathology (Comeaux et al., 2016).
<b>Piroplasms</b>				
<i>Anaplasma phagocytophilum</i>	Low infection rate in domestic pigs. Infection is typically subclinical, Potential zoonosis.	Wild boar	Disease not reported.	Serology, DNA PCR (Reiterova et al., 2016; Silaghi et al., 2014).
<i>Babesia bigemina</i>	Regionally common in domestic pigs. Infection results in hemolytic anemia, petechial hemorrhages, edema. Disease is generally mild.	Wild boar	Disease not reported.	DNA PCR (Zanet et al., 2014).
<i>Babesia trautmanni</i>	Uncommon in domestic pigs. Most frequently occurs in Africa. Infection results in pyrexia, inappetence, listlessness, followed by spontaneous recovery.	Warthogs, bushpigs	Similar to domestic pigs.	Walker et al. (2005)
<i>Eperythrozoon</i> sp. (likely <i>Mycoplasma</i> sp)	Regionally common in domestic pigs. <i>Mycoplasma suis</i> ( <i>E. suis</i> ) causes potentially hemolytic anemia in naïve animals. Gross lesions include splenomegaly and icterus. Parasites identified on the surface of erythrocytes. Arthropod vector.	Wild boar (rare), South American peccaries (captive)	Parasite species not identified. Subclinical disease. Possible risk to livestock.	Hemocytology (Castellano Margardio and Mangini, 2001; Hannon et al., 1985).

Theileria sp.	Clinical disease not reported in domestic pigs. Arthropod vector.	Warthog, Wild boar	Clinical disease not reported. Risk to livestock.	Hemocytology (Fowler, 1996; Junker et al., 2015; Zanet et al., 2014).
<b>Coccidia</b>				
<i>Eimeria</i> spp.	<i>E. scabra</i> , <i>E. spinose</i> , <i>E. perminuta</i> , <i>E. cerdonis</i> , <i>E. deblickei</i> , and <i>E. neodeblickei</i> cause self-limiting diarrhea in 1–3 month-old pigs. Direct life cycle. Gross and microscopic lesions similar to <i>I. suis</i> .	All suids susceptible	Infrequently reported. <i>E. pecarii</i> , <i>E. chaparralensis</i> , and <i>E. dicotylensis</i> are only reported in collared peccaries.	Fecal microscopy (oocysts), histopathology (Fowler, 1996; Wilber et al., 1996).
<i>Isospora (Cystisporas) suis</i>	Severe, occasionally fatal enteritis in neonatal domestic pigs. Direct life cycle. Gross lesions include white-yellow diarrhea. Microscopic ileal and jejunal lesions include villus atrophy, ulcerative to fibrinonecrotic enteritis with intraepithelial coccidia.	All suids susceptible, reported in wild boar, warthogs, peccaries.	Infrequently reported. Similar to domestic pigs.	Fecal microscopy (oocysts), histopathology (Fowler, 1996).
<b>Ciliates</b>				
<i>Balantidium coli</i>	Ubiquitous in domestic pigs (definitive host). Direct fecal-oral life cycle. Ingestion of cyst, excyst in small intestine, trophozoites in cecum, colon. Resident in lumen, may be invasive into mucosa. Invasive disease results in necrotizing and ulcerative typhlocolitis with intralumenal trophozoites. Zoonotic.	Wild boar, warthogs, peccaries, likely all suids.	All suids susceptible. Episodic diarrhea, disease similar to domestic pigs.	Fecal microscopy. Cysts and trophozoites (Castellano Margardio and Mangini, 2001; Mansouri et al., 2016; Samuel and Low, 1970; Solaymani-Mohammadi et al., 2004).
<b>Flagellates</b>				
<i>Chilomastix mesnili (hominis; suis)</i>	Primarily a commensal of primates, also infects domestic pigs. Infects cecum and colon, generally nonpathogenic. Pyriform flagellates in feces, increased in number with other protozoal infections. Direct fecal-oral life cycle.	Wild boar	Disease not reported.	Fecal microscopy (Solaymani-Mohammadi et al., 2004).
<i>Dientamoeba fragilis</i>	Infects domestic pigs, disease not reported. Zoonotic, causes diarrhea in humans. Direct fecal-oral life cycle.	Wild boar	Disease not reported.	Fecal analysis; PCR (Crotti et al., 2012).
<i>Giardia</i> sp.	Ubiquitous in domestic pigs, all ages may be infected. Direct fecal-oral life cycle. May be associated with mild diarrhea, no specific gross or microscopic lesions.	Wild boar, peccaries	Similar to domestic pigs.	Fecal microscopy. Cysts and trophozoites (Rodriguez-Rivera et al., 2016).
<i>Trypanosoma pecarii</i>	Not reported.	Collared peccaries	Disease not reported.	Hemocytology (Castellano Margardio and Mangini, 2001).
<i>Tritrichomonas suis (T. foetus)</i>	Commensal in nasal cavity and lower digestive tract in domestic pigs. Incidental tissue invasion may occur secondary to infection with other pathogens (e.g., <i>Brachyspira pilosicoli</i> colitis)	Wild boar	Disease not reported.	Fecal microscopy (Solaymani-Mohammadi et al., 2004).
<b>Amoebae</b>				
<i>Iodamoeba butschlii</i>	Generally nonpathogenic in domestic pigs. May be associated with diarrhea. Zoonotic.	Wild boar	Nonpathogenic	Fecal microscopy (Solaymani-Mohammadi et al., 2004).

(Continued)

**TABLE e2** Additional Parasites of Suids (*Cont.*)

Pathogen	Disease in Domestic Pig	Wild Host	Disease in Wild	Evidence in Wild Suids
<i>Entamoeba polecki</i>	Generally nonpathogenic in domestic pigs. Some isolates have been associated with diarrhea. Zoonotic.	Wild boar	Nonpathogenic	Fecal microscopy (Solaymani-Mohammadi et al., 2004).
<i>Entamoeba suis</i>	Generally nonpathogenic in domestic pigs. Rare isolates may be associated with diarrhea and hemorrhagic colitis. Zoonotic.	Wild boar	Not reported.	Fecal microscopy (Solaymani-Mohammadi et al., 2004).
<b>Metazoan Parasites</b>				
<b>Nematodes</b>				
<b>Upper GI</b>				
<i>Diplogaster parasiticus</i> (taxonomy unresolved)	Not reported.	Bushpig ( <i>Potamochoerus sp.</i> )	Poorly described rhabditid nematodes colonizing buccal pouch. Lesions not reported.	Necropsy (Marlow, 1955; Round, 1968).
<i>Eucoleus</i> ( <i>Capillaria</i> ) <i>garfiai</i>	Infest lingual epithelium. Generally nonpathogenic.	Wild boar	Similar to domestic pigs.	Necropsy (Fernandez-de-Mera et al., 2003).
<i>Gongylonema pulchrum</i> , <i>G. spp.</i>	Infest esophageal mucosa. Mild esophagitis. Zoonotic.	Wild boar, collared peccaries	Similar to domestic pigs.	Necropsy (Fernandez-de-Mera et al., 2003).
<b>Stomach</b>				
<i>Capillaria sp.</i> (not <i>C. hepatica</i> or <i>C. (Eucoleus) garfiai</i> )	Rarely reported.	Wild boar	Gastritis, not otherwise described.	Necropsy (Lowenstein and Kutzer, 1989).
<i>Gnathostoma hispidum</i> , <i>G. spp.</i>	Gastric spiruid, nodular, and ulcerative gastritis. Fresh-water crustacean (e.g., cyclops) intermediate host.	Wild boar, warthogs, bushpigs ( <i>Potamochoerus sp.</i> )	Similar to domestic pigs.	Necropsy (Belem, 2012; Round, 1968).
<i>Parabronema peccariae</i>	Not reported.	Peccaries	Parasite ID only, disease not reported.	Necropsy (Samson and Donaldson, 1968).
<i>Phyocephalus sexalatus</i>	Primarily infests lumen, generally noninvasive. Often subclinical, heavy infestation associated with catarrhal gastritis, anemia, and melena. Dung beetle intermediate host.	Wild boar, warthogs, bushpigs ( <i>Potamochoerus sp.</i> ), peccaries	Similar to domestic pigs.	Necropsy (Fernandez-de-Mera et al., 2003; Horak et al., 1988; Junker et al., 2015; Round, 1968; Samuel and Low, 1970; Van Wyk and Boomker, 2011).
<i>Parostertagia heterospiculum</i>	Rarely reported, poorly described. Disease similar to <i>Hyostromylus rubidus</i> , less severe.	Peccaries	Parasite ID only, disease not reported.	Necropsy (Samuel and Low, 1970).
<i>Simondsia paradoxa</i>	Chronic gastritis. Occurs concurrently with <i>Hyostromylus rubidus</i> (see text), disease is similar, but less severe.	Wild boar, warthogs	Similar to pigs	Fecal microscopy, necropsy (Belem, 2012; Fernandez-de-Mera et al., 2003).
<i>Trichostrongylus falcatulus</i> , <i>T. instabilis</i> , <i>T. thomasi</i> , <i>T. deflexus</i>	Nodular gastritis. Infrequently reported. Direct life cycle. Rarely found in small intestine.	Wild boar, warthogs, peccaries, bushpigs ( <i>Potamochoerus sp.</i> )	Similar to domestic pigs.	Necropsy (Boomker et al., 1991; Junker et al., 2015; Round, 1968; Samson and Donaldson, 1968).
<b>Small intestine</b>				
<i>Ascaris phacochoeri</i>	Not reported.	Warthogs, bushpigs ( <i>Potamochoerus sp.</i> )	Similar to <i>A. suum</i> .	Fecal examination, necropsy (Belem, 2012; Horak et al., 1988; Junker et al., 2015).
<i>Capillaria sp.</i> (not <i>C. hepatica</i> or <i>C. (Eucoleus) garfiai</i> )	Rarely reported. Disease not described.	Wild boar, Pygmy hogs	Enteritis (pygmy hogs)	Small intestine mucosal scraping. Microscopy (Kakati et al., 2015).
<i>Cooperia hungi</i>	Not reported.	Warthogs	Infestation rarely reported, primarily a parasite of impala.	Necropsy (Van Wyk and Boomker, 2011).

<i>Texicospirura turki</i>	Not reported.	Collared peccaries;	Nematode species originally erroneously described in stomach of collared peccaries, Disease not described.	Necropsy (Fowler and Cubas, 2001; Samson and Donaldson, 1968; Samuel and Low, 1970).
<b>Cecum, colon</b>				
<i>Morgascaridia sellsi, M. kugii</i>	Not reported.	Wild boar, bushpigs ( <i>Potamochoerus sp.</i> )	Rare. Clinical disease not reported.	Necropsy (Round, 1968; Sato et al., 2008).
<i>Osesophagostomum dentatum, O. aethiopicum; O. eurycephalum, O. goodeyi, O. mocambiquei, O. mpwapwae, O. mwanzae, O. oldi, O. roubaudi, O. santosdiasi, O. simpsoni, O. yorkei</i>	Chronic colitis, with nodular to diffuse thickening and mixed inflammation in the colon wall. Ova hatch in large intestine, migrate into submucosa, return to mucosa. Deep penetration to serosa associated adhesions.	Wild boar, warthogs, bushpigs ( <i>O. aethiopicum</i> )	Similar to domestic pigs.	Fecal examination, necropsy, histopathology (Belem, 2012; Horak et al., 1988; Junker et al., 2015; Round, 1968; Van Wyk and Boomker, 2011).
<i>Morgascaridia sellsi, M. kugii</i>	Not reported.	Wild boar, bushpigs ( <i>Potamochoerus sp.</i> )	Rare. Clinical disease not reported.	Necropsy (Round, 1968; Sato et al., 2008).
<i>Murshidia hamata, M. pugnicaudata</i>	Uncommon. Primarily infest the colon. Clinical disease not reported.	Warthogs	Similar to domestic pigs. May be present in large numbers in free-ranging warthogs, but clinical disease not reported.	Necropsy (Horak et al., 1988).
<i>Probstmayria vivipara</i>	Small equine pinworm. Infests colon, rectum. Clinical disease not reported.	Warthogs	High prevalence and heavy burdens in free-ranging warthogs. Clinical disease not reported.	Necropsy (Belem, 2012; Junker et al., 2015; Van Wyk and Boomker, 2011).
<i>Trichuris suis</i>	Whipworms primarily infest the cecum, large burdens extend to colon. Primarily disease of young animals. Parasites embedded in mucosa, cause catarrhal enteritis, may progress to necrotizing and hemorrhagic enteritis. Direct life cycle.	Wild boar, Warthogs, all suids likely susceptible	Similar to domestic pigs.	Necropsy (Fowler, 1996).
<b>Lungs</b>				
<i>Metastrongylus elongatus (apri), M. pudendotectus, M salmi</i>	Indirect, possibly direct life cycle. Earthworm intermediate host. Gross lesions include thread-like 4–7 cm long adult nematodes within airways, severe pulmonary congestion, edema, and consolidation. Larvae may migrate through liver.	Wild boar, all suids likely susceptible.	Similar to domestic pigs. Secondary bacterial pneumonia.	Necropsy, histopathology (Fowler, 1996; Mansouri et al., 2016).
<b>Liver</b>				
<i>Capillaria hepatica (Calodium heptaicum)</i>	Chronic-active eosinophilic hepatitis. Zoonotic.	Collared peccaries	Similar to domestic pigs.	Castellano Margardio and Mangini, (2001)

(Continued)



**TABLE e2** Additional Parasites of Suids (Cont.)

Pathogen	Disease in Domestic Pig	Wild Host	Disease in Wild	Evidence in Wild Suids
<b>Kidneys</b>				
<i>Stephanurus dentatus</i>	Kidney worm of domestic swine. Direct life cycle. Eggs passed in the urine and infection by ingestion of larvae or percutaneous larval invasion. Earthworm paratenic host. Larval migration to the liver via the portal system causes a chronic hepatitis, and subsequent peritoneal migration may cause peritonitis. Aberrant migration is common, and larvae may be found in the visceral adipose tissue, lungs, epaxial muscles, or spinal cord. Adults are typically found in the kidneys, ureters, and perirenal tissue, and are often enclosed in a fibrous capsule.	Wild boar.	Similar to domestic pigs.	Necropsy (Kaufmann, 2014; Sutherland-Smith, 2015).
<b>Other</b>				
<i>Dirofilaria acutuscula</i>	Not reported.	Collared peccaries	Filariae in subcutaneous dorsolumbar fascia. Disease limited to localized inflammation.	Necropsy (Samuel and Low, 1970).
<i>Impalaia tuberculata</i>	Primarily infests African antelope, accidental infection in domestic pigs. Disease not reported.	Warthogs	Disease not reported.	Necropsy (Junker et al., 2015; Van Wyk and Boomker, 2011).
<i>Parabronema pecariae</i>	Not reported.	Peccaries	Adult spiruoids within the mucosa of the stomach. Mild gastritis.	Necropsy (Samuel and Low, 1970; Vicente et al., 2000).
<i>Setariaspp.</i> ( <i>S. congolensis</i> , <i>S. bernardi</i> , <i>S. thomasi</i> , <i>S. castroi</i> )	Domestic pigs ( <i>S. congolensis</i> ). Mosquito vector. Adults in peritoneal cavity, microfilariae in microvasculature may cause ocular and neurological lesions.	Warthogs, bushpigs	Some species, microfilariae free in abdominal cavity. Incidental; not associated with disease.	Necropsy (Junker et al., 2015; Khin and Win, 1981; Round, 1968; Van Wyk and Boomker, 2011).
<b>Cestodes (larva)</b>				
<i>Echinococcus granulosus</i>	Larvae in lungs, other viscera. Canids are definitive hosts.	Wild boar, bush pigs (red river hogs)	Similar to domestic species, zoonotic risk	Histopathology, DNA PCR (Horak et al., 1988; Mansouri et al., 2016; Van Wyk and Boomker, 2011).
<i>Moniezia benedeni</i> , <i>M. mettami</i> , <i>M. spp.</i>	Uncommon ( <i>M. expansa</i> ). Incidental adults in small intestine.	Warthogs, Collared peccaries ( <i>Moniezia benedeni</i> )	Adults in anterior small intestine. Disease not reported.	Necropsy (Boomker et al., 1991; Junker et al., 2015; Round, 1968; Samuel and Low, 1970).
<i>Paramonezia phaochoeri</i>	Not reported.	Warthogs	Adults in small intestine.	Necropsy (Van Wyk and Boomker, 2011).
<i>Stilesia globipunctata</i>	Adults in small intestine. Uncommon, generally non-pathogenic.	Warthogs	Similar to domestic pigs.	Necropsy (Belem, 2012).
<i>Taenia acinonyxi</i>	Visceral cysticercosis. Cheetahs are definitive hosts.	Warthogs, wild boar	Similar to domestic pigs.	Necropsy (Sutherland-Smith, 2015).
<i>Taenia crocutae</i>	Visceral cysticercosis. Hyenas are definitive hosts.	Warthogs	Similar to domestic pigs.	Necropsy (Sutherland-Smith, 2015).
<i>Taenia hyaenae</i>	Visceral cysticercosis. Hyenas are definitive hosts.	Warthogs	Similar to domestic pigs.	Necropsy (Sutherland-Smith, 2015).
<i>Taenia hydatigena</i> ( <i>Cysticercus tenuicollis</i> )	Visceral cysticercosis. Many canids and felids are definitive hosts.	Wild boar	Cysticerci in mesentery, lungs	Necropsy (Mansouri et al., 2016).

<i>Taenia regis</i>	Visceral cysticercosis. Lions are definitive hosts.	Warthogs	Similar to domestic pigs.	Necropsy (Sutherland-Smith, 2015).
<i>Taenia solium</i> ( <i>Cysticercus cellulosae</i> )	Cysticerci in the central nervous system (brain, meninges), heart, skeletal muscle, and liver.	Wild boar	cysticerci isolated from muscle and heart. Zoonotic risk.	Histopathology, DNA PCR (Mansouri et al., 2016).
<b>Trematodes</b>				
<i>Alaria alata</i>	Rare. Mesocercaria in cheek, skeletal muscle, not associated with clinical disease. Indirect life cycle. Carnivore definitive host. Gastropod 1st intermediate host. Amphibian 2nd intermediate host.	Wild boar	Similar to domestic pigs. Zoonotic risk.	Necropsy; alaria migration technique (Paulsen et al., 2013).
<i>Dicrocoelium dendriticum</i>	Indirect life cycle. Gastropod 1st intermediate host, ant 2 <sup>nd</sup> intermediate host. Infection by ingestion of ants. Lesions similar to <i>Fasciola hepatica</i> .	Wild boar	Similar to domestic pigs.	Necropsy. (Mansouri et al., 2016).
<i>Gastrodiscoides hominis</i> ( <i>Gastrodiscus aegyptiacus</i> )	Infest cecum, colon. May result in chronic necrotizing and eosinophilic and lymphoplasmacytic typhlocolitis with goblet cell. Associated with severe cecal edema and catarrhal typhlitis/diarrhea. Pig definitive host. Metacercaria on aquatic plants and in intermediate host crayfish, tadpoles, frogs. Zoonotic.	Wild boar, warthogs, bush-pigs ( <i>Potamochoerus sp.</i> )	Similar to domestic pigs. Zoonotic risk.	Fecal analysis, necropsy (Round, 1968; Yu and Mott, 1994).
<i>Fasciola gigantica</i>	Lifecycle and lesions similar to <i>F. hepatica</i> . More common in Africa, Asia.	Wild boar	Similar to domestic pigs.	Necropsy (Mansouri et al., 2016).
<i>Fasciola hepatica</i>	Indirect life cycle. Gastropod intermediate host, infective metacercaria on aquatic plants. Larval migration to liver and bile ducts results in necrotizing hepatitis, and maturation in bile ducts results in chronic cholangiohepatitis, portal fibrosis. Larval trematodes may be present in parenchymal lesions, adults present within bile ducts. More common in Europe, North and South America.	Wild boar (esp. Nebrodi Black pigs)	Similar to domestic pigs.	Necropsy (Thompson et al., 2009).
<i>Fascioloides magna</i>	Life cycle and lesions similar to <i>Fasciola hepatica</i> , but do not invade bile ducts. Adults are encapsulated in liver parenchyma.	Wild boar, Collared peccaries	Similar to domestic pigs.	Necropsy (Samuel and Low, 1970).
<i>Ornithobilharzia turkestanicum</i>	Larval schistosomes migrate through liver, adults in mesenteric veins. Eggs deposited in walls of mesenteric vessels provoke intense inflammation and fibrosis, may have nodular lesions on gross examination. Chronic eosinophilic and granulomatous phlebitis and mesenteritis centered on ova. Egg migration into liver may result in cirrhosis. Indirect life cycle. Gastropod intermediate host.	Wild boar	Adults in mesenteric veins. Disease similar to domestic pigs.	Necropsy (Mansouri et al., 2016; Maxie and Robinson, 2007).
<i>Paragonimus westermani</i>	Uncommon. Larvae in skeletal muscle not associated with clinical disease. Canid definitive host (lung). Gastropod 1st intermediate host. Crustacean (crab, crayfish) 2nd intermediate host. Zoonotic.	Wild boar (paratenic host)	Similar to domestic pigs. Zoonotic risk.	Histology. Larvae in muscle tissue digestion, PCR (Sugiyama et al., 2015).
<b>Pentastomes</b>				
<i>Linguatula nuttalli</i>	Not described in domestic pigs. Lion, other carnivores are definitive hosts.	Warthog	Nymphs recovered at necropsy, tissue not identified (common in liver in African antelope)	Necropsy (Horak et al., 1988; Junker et al., 2015).

(Continued)

**TABLE e2** Additional Parasites of Suids (*Cont.*)

Pathogen	Disease in Domestic Pig	Wild Host	Disease in Wild	Evidence in Wild Suids
<b>Arthropod Ectoparasites</b>				
<b>Mites</b>				
See text				
<b>Fleas</b>				
<i>Ctenocephalides felis</i>	"Cat flea"	Collared peccaries	Clinical disease not reported.	Parasite identification (Gruver and Guthrie, 1996).
<i>Echidnophaga larina</i>	Not reported.	Warthog (may be host-specific)	Stick-tight fleas, favor ventrum. Clinical disease not reported.	Parasite identification (Horak et al., 1988; Matthee et al., 2013).
<i>Moeopsylla sjoestedti</i>	Not reported.	Warthog (may be host-specific)	Jumping fleas, favor head and neck. Clinical disease not reported.	Parasite identification (Horak et al., 1988; Matthee et al., 2013).
<i>Phacopsylla (Echidnophaga) inexpectata, P. spp.</i>	Not reported.	Warthog (may be host-specific)	Stick-tight fleas, favor ventrum. Clinical disease not reported.	Parasite identification (Horak et al., 1988; Junker et al., 2015; Matthee et al., 2013).
<i>Pulex porcinus</i>	"Javalina flea," or "Peccary flea."	Collared peccaries, wild boar	Clinical disease not reported.	Parasite identification (Samuel and Low, 1970).
<b>Ticks</b>				
<b>Ixodidae</b>				
<i>Amblyomma americanum</i>	"Lone star tick." 3-host life cycle, with a wide host range in all lifestages	Wild boar (USA)	Clinical disease not reported.	Parasite identification (Allan et al., 2001; Helcel et al., 2016).
<i>Amblyomma boeri</i>	Not reported in domestic pigs. 1-host life cycle.	Chacoan peccaries	All life stages of tick present on peccaries, may be host-specific	Parasite identification (Nava et al., 2009).
<i>Amblyomma cajenense</i>	"Cayenne tick." 3-host life cycle with a wide host range in all life stages.	All peccaries, wild boar (USA)	Opportunistic	Parasite identification (Fowler and Cubas, 2001; Helcel et al., 2016).
<i>Amblyomma hebraeum</i>	"South African Bont Tick," 3-host life cycle. Immature and adult ticks may infest livestock. Vector for <i>Ehrlichia ruminantium</i> infection in livestock (but not suids) and <i>Rickettsia africae</i> infection in humans Prefer ventrum, axillae, tail.	African warthogs (primary host), "bushpigs" (red river hogs)	Opportunistic is common, tick-bite wounds may be severe and are predilection sites for secondary bacterial dermatitis and old-world screwworm infection.	Horak et al. (1988); Horak et al. (1991); Levin (2016); Matthee et al. (2013)
<i>Amblyomma inornatum</i>	Not reported in domestic pigs. Life cycle incompletely characterized, likely 3-host life cycle.	Collared peccary	Clinical disease not reported.	Parasite identification (Gladney et al., 1977).
<i>Amblyomma maculatum</i>	"Gulf coast tick." 3-host life cycle, adults on medium and large animals	Wild boar (USA)	Tick-bite wounds may be severe, with secondary bacterial dermatitis and new-world screwworm infection.	Parasite identification (Allan et al., 2001; Helcel et al., 2016; Levin, 2016).
<i>Amblyomma neumanni</i>	Not described in domestic pigs. 1-host life cycle. Primarily a parasite of cattle.	All peccaries	Clinical disease not reported.	Parasite identification (Nava et al., 2009).
<i>Amblyomma parvum</i>	Not reported in domestic pigs. Life cycle incompletely characterized, likely 3-host life cycle.	All peccaries, wild boar	Clinical disease not reported.	Parasite identification (Nava et al., 2009; Ramos Vdo et al., 2014).

<i>Amblyomma paulopunctatum</i>	Not reported in domestic pigs. Life cycle incompletely characterized, likely a 1-host life cycle.	Red river hogs, wild boar, warthogs, Giant forest hog ( <i>Hylochoerus sp.</i> )	Primarily found in Africa. Clinical disease not reported. All suids likely susceptible.	Parasite identification (Keirans, 1985; Leslie and Huffman, 2015).
<i>Amblyomma sculptum</i>	Domestic pigs are primary host. Can maintain a 1-host life cycle in livestock. Disease not reported, vector for multiple rickettsial pathogens.	Wild boar (South America)	Nymphs and adults on wild boar. Disease not reported.	Parasite identification (Ramos Vdo et al., 2014).
<i>Amblyomma triguttatum</i>	“Ornate kangaroo tick.” Not reported in domestic pigs. 1-host life cycle, broad host range.	Wild boar (Australia)	Nymphs infest the ears of wild boar, other life stages not identified.	Parasite identification (Guglielmo, 1990).
<i>Dermacentor albipictus</i>	“Winter tick.” 1-host life cycle prefers large animals	Wild boar (USA),	Clinical disease not reported.	Parasite identification (Helcel et al., 2016).
<i>Dermacentor halli</i>	Not reported in domestic pigs. Life cycle incompletely characterized.	Peccaries, Wild boar (USA)	Clinical disease not reported.	Parasite identification (Helcel et al., 2016).
<i>Dermacentor variabilis</i>	“American dog tick.” 3-host life cycle, adults prefer medium-large animals	Wild boar (USA)	Common in Southern USA, disease not described.	Parasite identification (Allan et al., 2001; Helcel et al., 2016).
<i>Haemaphysalis leporis-palustris</i>	Wide distribution in North America. 3-host life cycle. Disease Primarily parasitizes lagomorphs and birds. not reported, vector for rickettsial agents.	Peccaries, likely all suidae susceptible due to broad host range.	Clinical disease not reported.	Samuel and Low (1970)
<i>Haemaphysalis longicornis</i>	Disease in domestic pigs not reported. 3-host life cycle. Vector for rickettsial agents, other piroplasms.	Wild boar (Korea, Asia)	Clinical disease not reported.	Chae et al. (2017)
<i>Haemaphysalis flava</i>	Rarely reported in domestic pigs. 3-host life cycle.	Wild boar (Korea, Asia)	Clinical disease not reported.	Chae et al. (2017)
<i>Haemaphysalis pumilio</i>		Bushpigs		Parasite identification (Horak et al., 1991).
<i>Ixodes cumulatimpunctatus</i>	Not reported in domestic pigs. Life cycle incompletely characterized.	Red river hogs	Clinical disease not reported.	Parasite identification (Leslie and Huffman, 2015).
<i>Ixodes nipponensis</i>	Not reported in domestic pigs, primarily parasitizes cattle, hares. 3-host life cycle.	Wild boar (Korea)	Clinical disease not reported.	Parasite identification (Chae et al., 2017).
<i>Ixodes scapularis</i>	“Black-legged tick,” or “Deer tick.” 3-host tick. Uncommon in domestic pigs. Vector for <i>Borrelia burgdorferi</i> and multiple <i>Ehrlichia spp.</i> , <i>Babesia spp. others.</i>	Wild boar (USA)	Common in Southern USA, disease not described.	Parasite identification (Allan et al., 2001; Helcel et al., 2016).
<i>Rhipicephalus appendiculatus</i>	Poorly described in domestic pigs, primarily parasitizes cattle. 3-host tick. Prefers to infest head and ears, may cause severe skin lesion around ears.	Warthogs, “bushpigs” (red river hogs)	Clinical disease not reported.	Parasite identification (Horak et al., 1988; Horak et al., 1991; Matthee et al., 2013; Walker et al., 2005).
<i>Rhipicephalus camicasi</i>	Not described in domestic pigs. Life cycle incompletely characterized. Disease not reported.	Warthogs	Clinical disease not reported.	Parasite identification (Walker et al., 2005).
<i>Rhipicephalus complantus</i>	Regionally common in African domestic pigs. Disease not reported. Life cycle incompletely characterized.	Wild boar, red river hogs, bushpigs ( <i>P. larvatus</i> ), warthogs	Clinical disease not reported.	Parasite identification (Leslie and Huffman, 2015; Pourrut et al., 2011; Walker et al., 2005).
<i>Rhipicephalus compositus</i>	Poorly described in domestic pigs. Disease not reported. Life cycle incompletely characterized.	“Bushpigs” (red river hogs)		Parasite identification (Walker et al., 2005).
<i>Rhipicephalus congolensis</i>	Not described in domestic pigs.	Red river hogs	Clinical disease not reported.	Parasite identification (Apanaskevich et al., 2013).
<i>Rhipicephalus cuspidatus</i>	Not described in domestic pigs. Life cycle incompletely characterized. Disease not reported.	Red river hogs, warthogs	Clinical disease not reported.	Parasite identification (Leslie and Huffman, 2015; Walker et al., 2005).

(Continued)

**TABLE e2** Additional Parasites of Suids (Cont.)

Pathogen	Disease in Domestic Pig	Wild Host	Disease in Wild	Evidence in Wild Suids
<i>Rhipicephalus (Boophilus) decoloratus</i>	“African blue tick.” Infrequently reported in domestic pigs, primarily parasitizes African cattle and antelope. 1-host life cycle. Vector for <i>Babesia bigemina</i> and multiple other piroplasms.	Warthogs	Clinical disease not reported.	Parasite identification (Horak et al., 1988; Horak et al., 2007; Matthee et al., 2013; Walker et al., 2005).
<i>Rhipicephalus evertsi</i>	Not reported in domestic pigs. 2-host life cycle.	Warthogs, “bushpigs” (red river hogs)	Clinical disease not reported.	Parasite identification (Horak et al., 1988; Horak et al., 2007; Matthee et al., 2013).
<i>Rhipicephalus follis</i>	Not described in domestic pigs. Life cycle incompletely characterized. Disease not reported.	“Bushpigs” (red river hogs)	Clinical disease not reported.	Parasite identification (Horak et al., 1991; Walker et al., 2005).
<i>Rhipicephalus gertrudae</i>	Not described in domestic pigs. Life cycle incompletely characterized. Disease not reported.	Warthogs	Clinical disease not reported.	Parasite identification (Matthee et al., 2013).
<i>Rhipicephalus longiceps</i>	Poorly described in domestic pigs. 3-host life cycle. Primarily parasitizes African ruminants, but uncommon.	Warthogs	Clinical disease not reported.	Parasite identification (Horak et al., 2007; Matthee et al., 2013; Walker et al., 2005).
<i>Rhipicephalus longus</i>	Regionally common in African domestic pigs. Disease not reported. Life cycle incompletely characterized.	Wild boar, “bushpigs” (red river hogs)	Clinical disease not reported.	Parasite identification (Leslie and Huffman, 2015; Sugiyama et al., 2015; Walker et al., 2005).
<i>Rhipicephalus lunulatus</i>	Uncommon in domestic pigs. More common in cattle, African antelope. Disease not reported. 3-host life cycle.	Wild boar, “bushpigs” (red river hogs)	Clinical disease not reported.	Parasite identification (Leslie and Huffman, 2015; Walker et al., 2005).
<i>Rhipicephalus maculatus</i>	Poorly described in domestic pigs.	“Bushpigs” (red river hogs)	Clinical disease not reported.	Parasite identification (Horak et al., 1991; Walker et al., 2005).
<i>Rhipicephalus muehlensii</i>	Not described in domestic pigs. Life cycle incompletely characterized. Disease not reported.	“Bushpigs” (red river hogs)	Clinical disease not reported.	Parasite identification (Horak et al., 1991; Walker et al., 2005).
<i>Rhipicephalus muhsamae</i>	Poorly described in domestic pigs.	Red river hogs	Clinical disease not reported.	Parasite identification (Leslie and Huffman, 2015).
<i>Rhipicephalus simpsoni</i>	Not described in domestic pigs. Life cycle incompletely characterized. Disease not reported.	Red river hogs	Clinical disease not reported.	Parasite identification (Leslie and Huffman, 2015).
<i>Rhipicephalus simus</i>	Infrequently reported in domestic pigs. 3-host life cycle. Clinical disease not reported. Vector for transmission of <i>Babesia trautmani</i> from warthogs to domestic pigs.	Warthogs, “bushpigs” (red river hogs)	Clinical disease not reported.	Parasite identification (Horak et al., 1991; Horak et al., 2007; Matthee et al., 2013; Walker et al., 2005).
<i>Rhipicephalus zambeziensis</i>	Poorly described in domestic pigs, primarily parasitizes African cattle. 3-host tick.	Warthogs, “bushpigs” (red river hogs)	Clinical disease not reported. Likely to cause skin lesion in/around ears similar to domestic cattle.	Parasite identification (Horak et al., 1988; Matthee et al., 2013; Walker et al., 2005).
<i>Rhipicephalus (aurantiacus) ziemanni</i>	Poorly described in domestic pigs, life cycle incompletely characterized. Disease not reported.	Red river hogs	Clinical disease not reported.	Parasite identification (Leslie and Huffman, 2015; Pourrut et al., 2011).
<i>Rhipicephalus zumpti (reichnowi; planus)</i>	Not described in domestic pigs. Life cycle incompletely characterized. Disease not reported.	“Bushpigs” (red river hogs), warthogs	Clinical disease not reported.	Parasite identification (Horak et al., 1991; Walker et al., 2005).
<b>Argasidae</b>				
<i>Hyalomma truncatum</i>	Rarely reported in domestic pigs. 2-host life cycle. Typically parasitizes African ruminants.	Warthogs	Clinical disease not reported.	Parasite identification (Horak et al., 1988; Horak et al., 2007; Matthee et al., 2013).

<i>Ornithodoros moubata</i>	Infrequently described in African domestic pigs, but may serve as a significant vector for transmission of African swine fever from wildlife to livestock. Multihost life cycle. Also vector for <i>Borellia duttoni</i> , the cause of relapsing fever in humans.	Warthogs	Clinical disease not reported. Zoonotic risk. Vector for <i>Borellia duttoni</i> , the cause of relapsing fever in humans.	Parasite identification (Sanchez-Vizcaino et al., 2015).
<i>Ornithodoros porcinus</i> (part of <i>O. moubata</i> spp. complex)	Infrequently described in domestic pigs. Multihost life cycle. Vector for African swine fever, predominantly found in Africa.	Warthogs, bushpigs ( <i>Potamochoerus larvatus</i> ), red river hogs	Clinical disease not reported.	Parasite identification (Horak et al., 1988; Matthee et al., 2013; Roger et al., 2001; Sanchez-Vizcaino et al., 2015).
<i>Ornithodoros erraticus</i>	Vector for African swine fever, predominantly found in Europe. but role for ASF transmission unclear in many European countries,	Wild boar	Clinical disease not reported.	Parasite identification (Pietschmann et al., 2016; Sanchez-Vizcaino et al., 2015).
<i>Ornithodoros turicata</i>	Feed on wild boar exposed to animal burrows. Short feeding time (20–30 min), do not remain on animals. Vector for transmission of <i>Borrelia turicatae</i> , a cause of relapsing fever in humans.	Wild boar (USA)	Feed on wild boar exposed to animal burrows. Short feeding time (20–30 min), do not remain on animals. Vector for transmission of relapsing fever in humans.	
<i>Orthinodoros rostratus</i>		Wild boar (South America)	Clinical disease not reported.	Parasite identification (Ramos Vdo et al., 2014).
<b>Lice</b>				
<i>Hematopinus apri</i>	Sucking louse. Uncommon in domestic pigs, common in wild boar.	Wild boar (Europe, Asia)	Clinical disease not reported.	Manning and Graham (1997)
<i>Hematopinus latus</i>	Sucking louse. Uncommonly reported in domestic pigs.	Wild boar, warthogs, bushpigs ( <i>Potamochoerus larvatus</i> ), red river hogs	Clinical disease not reported.	Parasite identification (Horak et al., 1988, 1991; Junker et al., 2015).
<i>Haematopinus oliveri</i>	Sucking louse. Not reported in domestic pigs.	Pygmy hogs (possibly host restricted)	Clinical disease not reported.	Parasite identification (Mishra and Singh, 1978).
<i>Hematopinus phacochoeri</i>	Not reported in domestic pigs.	Wild boar, warthogs	Sucking louse.	Parasite identification (Horak et al., 1988; Junker et al., 2015; Matthee et al., 2013).
<i>Hematopinus suis</i>	“Common sucking louse.”	Wild boar (worldwide)		Parasite identification (Manning and Graham, 1997).
<i>Linognathida spp.</i>	Not reported in domestic pigs.	Collared peccaries	Clinical disease not reported.	Gruver and Guthrie (1996)
<i>Linognathus vituli</i>	Not reported in domestic pigs. Primarily parasitizes cattle.	Wild boar (Europe)	Clinical disease not reported.	Manning and Graham (1997)
<i>Pecarococcus javalii</i>	“Giant sucking louse.” Not reported in domestic pigs.	Collared peccaries	Collected from periocular skin. Clinical disease not reported.	Parasite identification (Samson and Donaldson, 1968; Samuel and Low, 1970).
<b>Flies/bots</b>				
<i>Rhinoestrus spp.</i>	Infrequently reported in domestic pigs. Myiasis sinusitis caused by developing larvae.	Red river hogs, warthogs	Chronic catharral and eosinophilic and lymphoplasmacytic rhinitis and sinusitis.	Parasite identification (Leslie and Huffman, 2015).

**TABLE e3** Commensal Parasites of Suids

GI Commensals	Disease in Domestic Pig	Wild Host	Disease in Wild	Evidence
<i>Telamodinium onyx</i>	Species not reported in domestic pigs.	Warthog	None. Inhabit cecum and colon.	Booyse and Dehority (2012)
<i>Megadinium aethiopicum</i>	Species not reported in domestic pigs.	Warthog	None. Inhabit cecum and colon.	Booyse and Dehority (2012)
<i>Teratodinium sphaeredon</i>	Species not reported in domestic pigs.	Warthog	None. Inhabit cecum and colon.	Booyse and Dehority (2012)
<i>Blepharoconus krugerensis</i>	Nonpathogenic.	Warthog	None. Inhabit cecum and colon.	Booyse and Dehority (2012)
<i>Blepharosphaera intestinalis</i>	Nonpathogenic.	Warthog, bushpigs	None. Inhabit cecum and colon.	Booyse and Dehority (2012)
<i>Charonina equi</i>	Nonpathogenic.	Warthog	None. Inhabit cecum and colon.	Booyse and Dehority (2012)

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**TABLE e3 Commensal Parasites (Cont.)**

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