

Chapter 2

Zoonotic Diseases of Swine: Food-borne and Occupational Aspects of Infection

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Abstract Swine and their products have become a central part of food systems around the world. Global pork production has rapidly increased over the past 30 years, leading to the intensification of the swine industry: though there are fewer farms now, those farms that do persist raise ever-larger numbers of animals. This increases the transmission of pathogens both amongst animal herds, and between animals and their human caretakers. Furthermore, increased stress to animals and the potential for amplification of pathogens in the farming environment can lead to a higher burden of disease-causing organisms in and on meat products, which then make their way to consumers world-wide. As such, swine and their meat products have the potential to introduce new zoonotic diseases into populations via multiple routes of transmission. Here we discuss several examples of zoonotic diseases of swine origin, reviewing diseases with bacterial, viral, or parasitic causes.

2.1 Background and Introduction

Pork is rapidly becoming the world's source of protein. Global pork production increased more than 80% between 1985 and 2010 (Fournie et al. 2012), and this trend has led to the intensification of swine husbandry, with fewer and fewer facilities present, but each raising larger numbers of individual animals. China has been a driver of this market, accounting for approximately 50% of total global pig production (Fournie et al. 2012). As swine production has intensified, so has concern over how these modifications in husbandry may affect the transmission of disease amongst pigs as well as to human caretakers. It has been estimated that more than 60% of emerging diseases are zoonotic (Jones et al. 2008). A recent review (Fournie et al. 2012) identified 77 pathogens that had not been described in swine prior to 1985, including 39 viruses and 32 bacterial species. Not surprisingly, the top 20% of pork-producing countries accounted for 82% of these emerging pathogens. Of these 77 novel species found to infect swine, 30 (39%) are zoonotic, and

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26% of these were identified in the context of an outbreak investigation (Fournie et al. 2012). Densely populated South East Asia is the epicenter of emergence of novel zoonotic diseases due to inter-species transmission. However, outbreaks of host specific lethal zoonoses have occurred in industrialized nations as well (Davies 2012). It is plausible that a dramatic change in swine industry demographics in recent decades without adequate biosecurity may have served as a tonic for the emergence of swine zoonosis (Davies 2012). Zoonotic diseases impose significant economic burden with increased morbidity and mortality globally. A change in ecological niche, climatic change, rapid growth in human population and socio-economic factors are among the major contributing factors for the emergence of zoonoses (Jones et al. 2008).

Outbreaks of human disease related to swine-origin pathogens, including *Streptococcus suis* in China (Lun et al. 2007), Nipah virus in Malaysia (Chua 2012) and the novel H1N1 variant influenza virus have gained significant media attention in the past decade. Here we discuss several examples of zoonotic diseases of swine origin, reviewing diseases with bacterial, viral, or parasitic causes.

2.1.1 *Yersinia enterocolitica*

Yersinia enterocolitica is a gram negative bacterium in the family *Enterobacteriaceae*. *Y. enterocolitica* is widely distributed throughout nature, having many animal and aquatic reservoirs; however, swine are considered the main reservoir for strains that are pathogenic to humans. It is the main causative agent of yersiniosis, a disease that affects animals and humans worldwide (Holt et al. 2000).

Yersinia enterocolitica can be classified into distinct subgroups based on biochemical characteristics (biotypes) and O-antigen specificity (serotypes). There are six biotypes (1A, 1B, 2, 3, 4, and 5) and 60 serotypes, 11 of which are associated with human illness (Nesbakken et al. 2006; Bottone 1997). Biotype 1B is considered the only highly pathogenic strain, while the others are considered moderately pathogenic, except for biotype 1A, which is considered nonpathogenic although this has recently become a contentious topic due to recent reports of 1A infections (Stephan et al. 2013). Biotype 1B is mainly found in North America and Japan and is different from other biotypes in that it can be found in water and other environmental sources, and can also be carried by swine and rodents. Biotypes 2 and 4 are associated with human infections in Europe; their main reservoirs are pigs and cattle. Biotypes 3 and 5 are uncommon, but are also associated with animal reservoirs (EFSA 2009; Fredriksson-Ahomaa et al. 2006a).

Yersiniosis is a gastrointestinal disease causing fever and watery, occasionally bloody, diarrhea. Rarely, *Y. enterocolitica* can cause septicemia, and in some cases long-term sequelae can occur. Symptoms generally occur 4–7 days after exposure and may last for up to a month (Bottone 1997; Huovinen et al. 2010). Approximately 16.5 cases per 1,000,000 persons occur each year in Europe (EFSA 2009), while in the United States, approximately 3.5 cases per 1,000,000 are seen each year

(Long et al. 2010). Children are infected more frequently than adults, and infections occur most commonly in temperate locations during colder months (Bottone 1997).

Pigs are commonly asymptomatic carriers of pathogenic strains of *Y. enterocolitica*. The bacteria typically reside in the gastrointestinal tract, especially the tonsils, lymph nodes, intestines and feces (Fredriksson-Ahomaa et al. 2007; Bhaduri et al. 2005). Cattle and goats have also been found to be carriers (Lanada et al. 2005a, 2005b), and milk products from these animals have been the source of numerous outbreaks in human populations (Black et al. 1978; Shayegani et al. 1983; Morse et al. 1984; Tacket et al. 1984; Ackers et al. 2000). Deer, rabbits, rodents (Quan et al. 1974), dogs (Byun et al. 2011), and cats (Fredriksson-Ahomaa et al. 2001) have also been found to carry as well as to be infected with *Y. enterocolitica*. In addition to livestock, water sources including wells, rivers and lakes can serve as reservoirs for the bacteria as a result of contamination by feces of carriers or leakage from latrines.

The major risk factors for developing yersiniosis include eating raw or undercooked pork (Boqvist et al. 2009; Fredriksson-Ahomaa et al. 2006b), drinking contaminated milk (Black et al. 1978; Tacket et al. 1984; Ackers et al. 2000), and consuming contaminated drinking water (Thompson and Gravel 1986; Christensen 1979). Porcine sources are frequently associated with the pathogenic serotypes O:3, O:9, and O:5,27 and sometimes with the highly virulent serotype O:8. Outbreaks in 2006 in Norway were identified as biotype 2 and 4 and indicated a processed pork product to be the likely source (Grahek-Ogden et al. 2007; Stenstad et al. 2007). In the United States, raw pork intestines were found to be the source of an outbreak among infants (Lee et al. 1990; Jones 2003). The occurrence of pathogenic *Y. enterocolitica* in pigs and pork has been established by PCR in several studies (Korte et al. 2003; Fredriksson-Ahomaa et al. 2003). The *ail* gene located within the genome of pathogenic *Y. enterocolitica* strains is the most frequently used target of amplification for positive identification. In Switzerland, the prevalence of *ail*-positive *Y. enterocolitica* in tonsils of slaughter pigs was shown to be 88% by PCR and 34% by culture methods (Fredriksson-Ahomaa et al. 2007). In the USA, *ail*-positive *Y. enterocolitica* were detected in 12% of pig feces sampled by PCR, and in 4% of them using culture methods. Similarly, 40% of the pig lymph nodes were positive by PCR, but none by culturing (Boyapalle et al. 2001). These results indicate that PCR based assays are the most sensitive and accurate means to detect *Y. enterocolitica* colonization.

Clinical presentations of yersiniosis are typical of enteric illness. Infants and children often present with fever, vomiting, and bloody diarrhea that can last from 3–28 days (Metchock et al. 1991; Lee et al. 1991). Adults generally have one to two weeks of fever, diarrhea, and abdominal pain that can mimic appendicitis. In more severe cases of gastroenteritis, necrotizing enterocolitis and ulceration may occur. *Y. enterocolitica* can also cause septicemia, leading to abscesses in the liver and spleen, pneumonia, septic arthritis, meningitis, cellulitis, empyema, osteomyelitis, and may evolve into endocarditis. Post-infection sequelae may also occur, particularly after infections with biotype 4, serotype O:3 (Bottone 1999). Reactive arthritis and erythema nodosum are the most common sequelae, but glomerulonephritis and myocarditis have also been reported (Bottone 1997).

Yersiniosis is diagnosed by positive identification of *Y. enterocolitica* in stool samples, although it is not routinely tested for. It can also be recovered from the throat, lymph nodes, joint fluid, urine, bile, or blood. Most cases resolve on their own, although it may take up to 3 weeks to recover. In severe cases, antibiotics such as aminoglycosides, doxycycline, trimethoprim-sulfamethoxazole, or fluoroquinolones may be prescribed. Prevention is key in avoiding infection. Raw or undercooked pork and unpasteurized milk or milk products should be avoided, as should drinking untreated water. Good hand hygiene when preparing food and after contact with animals should also be practiced to avoid infection.

2.1.2 *Staphylococcus aureus*

Staphylococcus aureus is a nonmotile, nonspore-forming, Gram positive coccus that occurs singly, in pairs, or in clusters. *S. aureus* produces protein A (*spa*), which is used in molecular testing for strain typing purposes, as well as several other toxins and superantigens (De Vos et al. 2009).

S. aureus is often isolated from the nasal membranes and skin of warm-blooded animals. Approximately 20–30 % of the human population is colonized with *S. aureus* in the nose, throat, or both (Smith et al. 2012; Gorwitz et al. 2008; Graham et al. 2006). The most important site for colonization is the anterior nares (Wertheim et al. 2005). Colonization itself is not harmful; however, it is a risk factor for developing subsequent infections (Graham et al. 2006; Fritz et al. 2009). Both asymptomatic carriers and infected individuals may transmit the bacterium to others through close contact. *S. aureus* may also be acquired via contact with fomites contaminated with the organism, as well as with animals that are colonized or infected with *S. aureus*.

Skin infections including furuncles, carbuncles, impetigo, and scalded skin syndrome, as well as more severe infections like pneumonia, osteomyelitis, endocarditis, myocarditis, pericarditis, enterocolitis, mastitis, cystitis, prostatitis, cervicitis, cerebritis, meningitis, bacteremia, toxic shock syndrome, and abscesses of muscles, skin, and organs can occur as a result of *S. aureus* infection.. Other mammals and birds are also susceptible to infections, including mastitis, synovitis, arthritis, endometritis, furuncles, suppurative dermatitis, pyemia and septicemia (De Vos et al. 2009). Pigs are common carriers of *S. aureus*; one study in the U.S. found overall MRSA prevalence was 70 % (147/209) from seven farms in the Midwest (Smith et al. 2009). In the Netherlands, surveillance for MRSA on hog farms has shown that isolates obtained from swine and their human caretakers are frequently indistinguishable, suggesting that the organism is transmitted between the two species (Smith et al. 2009; Huijsdens et al. 2006; Khanna et al. 2007).

S. aureus infections are often resistant to many antibiotics. Approximately 1.5 % of the U.S. population carries methicillin-resistant *S. aureus* (MRSA) (Gorwitz et al. 2008). Resistance to methicillin developed within 6 months of the first clinical use and has become a major cause of morbidity and mortality around the world. In the U.S. in 2011, there were 80,461 invasive MRSA infections, an incidence rate of 25.82 cases per 100,000 persons. Many animals, including cows, goats, sheep,

rabbits, and poultry, can be infected by *S. aureus*, and these infections can have large economic costs (Fitzgerald 2012).

The epidemiology of MRSA has changed rapidly in the past few decades. After developing resistance in the 1960s following methicillin introduction, MRSA became a superbug that primarily affected hospitalized patients. Due to association with the healthcare environment, these infections were called healthcare-associated MRSA (HA-MRSA). More recently, cases of MRSA infection have been detected in people without prior hospitalization and with no underlying illnesses or healthcare related risk factors; these are referred to as community-associated MRSA (CA-MRSA) infections. Cases of HA-MRSA are usually resistant to several classes of antibiotics and tend to carry the methicillin-resistance gene, *mecA*, on the Staphylococcal Chromosome Cassette (SCC) of type II (SCC*mec* type II). They are often associated with *spa* type t002 and multi-locus sequence type (MLST) ST5. Contrastingly, CA-MRSA infections tend to be resistant to fewer classes of antibiotics, carry the Panton-Valentine leukocidin (PVL) encoding gene, and carry SCC*mec* type IV, *spa* type t008, and MLST ST8. A third group of infections, livestock-associated MRSA (LA-MRSA), has recently been identified (Wulf and Voss 2008) and has typically been associated with swine or cattle. LA-MRSA include strains such as ST398 and ST9, often carry SCC*mec* type V, are typically PVL negative, and (like HA-MRSA) tend to be resistant to multiple classes of antibiotics. However, both CA-MRSA and LA-MRSA have caused nosocomial infections in hospitals (Jenkins et al. 2009; Fanoy et al. 2009; van Rijen et al. 2008, van Rijen et al. 2009; Wulf et al. 2008; Kourbatova et al. 2005; Seybold et al. 2006; Tattevin et al. 2009).

Livestock-associated MRSA first came to attention in 2005 after its identification in pigs in France (Armand-Lefevre et al. 2005) and in swine farmers in the Netherlands (Wulf and Voss 2008). Dutch researchers found that swine farmers were colonized with MRSA at a rate of 760 times higher than that of the general population (Voss et al. 2005). Since then, LA-MRSA has been found in a number of countries in Europe, Asia, and the Americas (Smith and Pearson 2011; Graveland et al. 2011; Fluit 2012).

Recent reports from Germany and the Netherlands have found a high proportion of ST398 carriage in areas that have a high density of livestock (Kock et al. 2009; Kock et al. 2011; Wulf et al. 2012). While originally thought not to cause severe infections, there have been increasing reports of invasive disease caused by ST398 (Hartmeyer et al. 2010; Mammina et al. 2010; Potel et al. 2010; Aspiroz et al. 2010). Methicillin-sensitive *S. aureus* (MSSA) ST398 isolates have also caused invasive disease in the eastern U.S. (Mediavilla et al. 2012), Europe (Witte et al. 2007; Declercq et al. 2008; van Belkum et al. 2008), South America (Jimenez et al. 2011) and Canada (Golding et al. 2010), and at least one death in France (Laurent 2009).

While the majority of individuals colonized or infected with LA-MRSA have had contact with swine, colonization with ST398 has also occurred in individuals lacking any identified contact with a livestock reservoir (Bhat et al. 2009; Aires-de-Sousa et al. 2006). It has been suggested that one mode of transmission into the community is via contaminated food. Numerous studies in the U.S. have found MRSA in 5% of 120 meat samples (Pu et al. 2008), MSSA in 16.4% and MRSA

in 1.2% of 125 meat samples (Hanson et al. 2011), MSSA in 64.8% and MRSA in 6.6% of 256 pork samples (O'Brien et al. 2012), and multi-drug resistant *S. aureus* in 52% of 136 meat and poultry samples (Waters et al. 2011). Additionally, two studies in the Netherlands found rates of 2.5% of 79 pork and beef samples (van Loo et al. 2007) and 11.9% of 2217 meat and poultry samples, respectively (de Boer et al. 2009). However, to date there have not been any confirmed infections with ST398 caused by contaminated food.

Most MRSA skin infections appear as pustules or boils which often are red, swollen, painful, and have pus or other drainage. They often are mistaken for spider or insect bites. These skin infections commonly occur at sites of visible skin trauma, such as cuts and abrasions, and areas of the body covered by hair. Health professionals may provide antibiotics and drainage if necessary to treat such infections. More severe infections may require hospitalization and intravenous antibiotic therapy. Good hygiene is the key to prevention of MRSA infections.

2.1.3 *Salmonella*

Salmonella is a genus of Gram-negative, rod shaped, non-spore forming enterobacteria with peritrichous flagella. Originally classified utilizing serotyping of the somatic lipopolysaccharide (O) and flagellar protein (H) antigens, each serological variant (serovar) was considered its own species under the *Salmonella* genus (White 1926; Kauffmann 1978) as reviewed in (Beltran et al. 1988). This methodology led to misclassifications due to horizontal transfer of cell surface antigens, leading to classification of genetically distinct strains within the same serovar (Beltran et al. 1988; Selander et al. 1990).

In 2005, the Judicial Commission of the International Committee for Systematics and Prokaryotes (JICSP) decided to change the type species of the *Salmonella* genus to *enterica* with subspecies and serovars (Prokaryotes JCotICoSo 2005). The JICSP indicated *Salmonella enterica* had seven subspecies, *enterica* (type I), *salamae* (type II), *arizonae* (type IIIa), *diarizonae* (type IVb), *bongori* (type V), *houtenae* (type IV), and *indica* (type VI). Subspecies *bongori* was shortly after promoted to species status (Grimont and Weill 2007). A third *Salmonella* species was approved by the JICSP in 2005, named *Salmonella subterranea* (Shelobolina et al. 2004), but this species may not fit within the genus *Salmonella* (Grimont and Weill 2007). *S. bongori* and all subspecies of *S. enterica* besides *S. enterica* subsp. *enterica* are associated mainly with cold-blooded animals (Aleksic et al. 1996; Woodward et al. 1997), (Aleksic et al. 1996; Woodward et al. 1997), but can rarely cause human infection (CDC 2008; CDC 2012). The primary cause of human infection is *S. enterica* subsp. *enterica* (CDC 2008), as referenced in (Desai et al. 2013).

The CDC defines salmonellosis as an infection with a *Salmonella* spp. bacterium. These infections can often manifest with diarrhea (potentially bloody), fever, and abdominal cramps between 12 and 72 h post infection (CDC 2009). The illness often lasts between 4 and 7 days and is usually self-limiting. *Salmonella* infection can necessitate hospitalization in a small number of individuals (Mead et al. 1999).

Each year, *Salmonella* spp. cause roughly 1.3 billion cases of nontyphoidal salmonellosis worldwide (Chimalizeni et al. 2010). Within the United States, there were an estimated 1.4 million cases in 1999, with 95 % of these estimated to be caused by foodborne exposure to *Salmonella* (Mead et al. 1999). The burden on the United States economy from these estimated 1.4 million cases was estimated to be between \$ 0.5 billion and \$ 2.3 billion (Frenzen et al. 2002). These estimates are likely underestimates due to the omission of secondary complications due to *Salmonella* infections. The estimates fail to include complications such as reactive arthritis or costs such as pain and suffering, or travel to obtain medical care.

The most important zoonotic reservoir for *Salmonella* are food animals, with the most important food product being eggs (Ebel and Schlosser 2000). Egg consumption has been shown to be the largest risk factor associated with *Salmonella enterica* infection (Hope et al. 2002). Pork contamination is also a possible source of human infection. In swine, *Salmonella* infection is mainly subclinical, with rare cases manifesting as enterocolitis or septicemia (Barker and Van Dreumel), as referenced in (Fosse et al. 2009). In the United States, the percentage of farms positive for *Salmonella* are estimated to range between 38.2 and 83 % with the number of positive pigs in the US from 6 to 24.6 % (Oosterom and Notermans 1983; Davies et al. 1997). Transmission from pig to pig is often due to fecal shedding of the bacteria. Within swine herds, sows were observed to have an increase in *Salmonella* shedding at weaning (Nollet et al. 2005) as well as in their weaned piglets (Kranker et al. 2003). While *Salmonella* is considered primarily fecal borne, swine feed has also been shown to be a potential source of *Salmonella* infection for swine (Harris et al. 1997) with experimental data showing animals may become infected through the consumption of contaminated feed (Smith 1960). Additional risk factors for transmission between herds of swine are: contact with humans, contaminated equipment, or contaminated slurry (Langvad et al. 2006).

Individual outbreaks of *Salmonella* spp. have also been attributed to pork products. In 1989, a small northern England town experienced an outbreak where 206 individuals were infected with serovar Typhimurium (Maguire et al. 1993). Serotyping and antibiotic resistance profiles matched the infective strain to that found in cold cuts of pork purchased from a local butcher shop. In a study by Davies et al., several of the most prevalent serotypes found in swine were also among the most common causes of human infection (Davies et al. 1997).

Attempts to control *Salmonella* spp. prevalence on farms have had mixed outcomes. The use of all-in/all-out systems with multiple sites handling different stages of the rearing process have been shown to have no benefit in reducing *Salmonella* prevalence when compared to farrow-to-finish systems (Davies et al. 1997). These all-in/all-out systems may actually have a greater prevalence of *Salmonella* in finishing pigs than farrow-to-finish systems and fecal shedding of *Salmonella* was higher than observed in farrow-to-finish (Davies et al. 1997). Number of pigs per pen was also observed to be a risk factor for fecal shedding of *Salmonella* (Linton et al. 1970). Acidification or fermentation of feed is postulated to be protective against *Salmonella* contamination as dry feed and trough feeding have been shown to have an increased contamination risk (Lo Fo Wong et al. 2004; van der Wolf

et al. 1999, van der Wolf et al. 2001), but this has not been studied extensively using experimental designs.

In North America, *Salmonella* control programs have been implemented at slaughter to decrease human exposure to *Salmonella* (Funk and Gebreyes 2004). This Pathogen Reduction: Hazard Analysis and Critical Control Point (HACCP) system established slaughter point performance standards for processing plants and has been shown to decrease contamination of pork products with *Salmonella* (Agriculture FSaISUDo 2004). In European Union countries, a farm-to-slaughter program has been implemented to reduce *Salmonella* (Lo Fo Wong et al. 2002). This plan calls for control measures at all production levels and focuses specifically on transportation and handling of the swine to limit the transmission between herds. In addition to prevention methods within the production system, consumer prevention is recommended by the CDC (CDC 2010). In addition to recommendations dealing with protecting infants from *Salmonella* exposure, the CDC suggests cooking meat and poultry thoroughly, washing hands, utensils, and kitchen surfaces following contact with raw meat or poultry.

2.1.4 *Campylobacter*

Campylobacter is a genus of gram-negative, spiral-spiral shaped bacteria that causes disease in both humans and animals (CDC 2010). *Campylobacter* is the most common cause of gastroenteritis in many developed (Nichols et al. 2012) and developing countries, causing more diarrhea than *Salmonella* globally (WHO 2011). In developing countries, infections of those under the age of two are most frequent (WHO 2011). While there are 17 species in the *Campylobacter* genus, *C. jejuni* and *C. coli* are the most frequent causes of infection (WHO 2011). Most cases are sporadic events and not part of outbreaks (CDC 2010). The main route of transmission from animals to humans is through undercooked meat and meat products, contaminated milk, or contaminated water (WHO 2011).

Disease in humans usually occurs two to five days after infection (WHO 2011) and presents with diarrhea, cramping, abdominal pain, and fever. Most infected individuals recover within five to ten days. In some severe cases, a small amount of people may develop Guillian-Barré syndrome. *Campylobacter* is thought to be responsible for between 20% (Tam et al. 2007) to 40% of cases of Guillian-Barré syndrome (CDC 2010). *Campylobacter* infections tend to be higher in males across all age groups, which suggests a higher susceptibility in males and not participation in at-risk behaviors (Nichols et al. 2012; Louis et al. 2005). In recent years, infections in those over 50 years of age have become more common, especially in men, as has infection in those between 20 and 32 years (Nichols et al. 2012). The increase in infections in those over 50 may be due to use of proton pump inhibitors (PPI's) (Nichols et al. 2012; Leonard et al. 2007). Seasonality of the infection has been noted, with the greatest impact of seasonality being in young children (Nichols et al. 2012). *Campylobacter* infections rates begin to rise in May and peak between

mid-June and mid-July (Nichols et al. 2012; Louis et al. 2005). This seasonality has been observed in many temperate countries (Nylen et al. 2002). Infection rates also tend to be higher in rural compared to urban regions (Strachan et al. 2009; Sibbald and Sharp 1985). This could be reflective of proximity to livestock or differences in access to healthcare (Nichols et al. 2012). Since 1989, there has been a steady increase in the presence of antimicrobial resistant *Campylobacter* isolates. Full and intermediate resistance to ampicillin, ciprofloxacin, nalidixic acid, tetracycline, and erythromycin has been shown (Nichols et al. 2012).

When swine are infected with *Campylobacter*, it is frequently *C. coli*, however, *C. jejuni* has been seen recently as well (Jensen et al. 2006). *Campylobacter* infections can cause diarrhea in pigs, and often colonizes the intestinal tract. Both *C. jejuni* and *C. coli* have been found in the intestinal tract of pigs and are known to be excreted in their feces (Jensen et al. 2006). *Campylobacter* has also been identified in the stomach, tonsils, liver, and carcass surfaces of swine. High colonization rates may represent an occupational health hazard, since a low dose of bacteria can cause infection (Nesbakken et al. 2003). Antimicrobial susceptibility to ciprofloxacin and nalidixic acid has been reported in swine strains. It has also been shown that *C. coli* has higher levels of quinolone resistance than *C. jejuni* in swine (von Altröck et al. 2013). However, it is unlikely that swine are a major source of foodborne *Campylobacteriosis*, as *Campylobacter* is rarely detected in retail pork, but may be a source of occupational exposure (Nesbakken et al. 2003). It has also been shown that while there is contamination of pigs in slaughter houses, *Campylobacter* spp. do not spread throughout the operation (von Altröck et al. 2013).

Campylobacter infections do not generally require treatment and are self-limiting (CDC 2010). When disease is severe, electrolyte and fluid replacement may be necessary. Antimicrobials (erythromycin, tetracycline, and quinolones) can be used to treat severe disease or to eliminate carriage (WHO 2011). Several steps can be taken to prevent *Campylobacter* infection. Proper food handling and hand hygiene can help prevent infection. All meats should be thoroughly cooked and measures should be taken to prevent cross contamination. Hands should be washed thoroughly before handling food and persons with diarrhea should wash their hands frequently to reduce the spread of infection (CDC 2010). Improved biosecurity measures and hygienic slaughtering practices will reduce the fecal contamination of carcasses (WHO 2011). Cooling meat with CO₂ has also been shown to kill the bacteria (Nesbakken et al. 2003). Adequate disposal of feces and decontamination of fecal contaminated articles will also help reduce transmission (WHO 2011).

2.1.5 *Streptococcus suis*

Streptococcus suis (*S. suis*) is a Gram-positive facultative anaerobe bacterium reported to colonize and cause infections primarily in the swine population worldwide (Fulde and Valentin-Weigand 2013; Wertheim et al. 2009). In conjunction with *Actinobacillus suis* and *Haemophilus parasuis*, *S. suis* completes the triad of the

“Suis-ide” disease agents given its association with a wide range of severe clinical conditions in the swine population (MacInnes and Desrosiers 1999). *S. suis* causes severe infections in pigs resulting in major economic losses to the porcine industry worldwide (Fittipaldi et al. 2012). Zoonotic infections due to *S. suis* have been reported in countries with a high density of pigs and intensive swine production (Lun et al. 2007; Wertheim et al. 2009). The increasing prevalence of infections due to *S. suis* both in swine and humans over the last few years have urged investigators to better understand the epidemiology and zoonotic potential of this primarily “pig pathogen”.

S. suis isolates are verified by serotyping using slide agglutination test, capsular reaction, capillary precipitation or a co-agglutination test (Staats et al. 1997). Serotyping is based on polysaccharide capsular antigen detection. Thirty-five serotypes (1–34 and 1/2) have been identified using these tests (Lun et al. 2007; Higgins and Gottschalk 1990; Gottschalk et al. 1989, 1991a, b, 1999; Higgins et al. 1995). Serotypes 32 and 34 are observed to be closely related to *S. orisratti* (Hill et al. 2005). Serotype 2 is the most frequently reported serotype worldwide and is considered the most pathogenic both in pigs and humans. Other serotypes implicated in diseases are types 1–9 and 14 (Gottschalk et al. 2007).

Pigs colonized with *S. suis* typically harbor the organism in their tonsils and may never exhibit clinical signs or symptoms (carriers). Some carrier piglets eventually develop bacteremia, septicemia or meningitis due to dissemination of *S. suis* from tonsils and other mucosal surfaces (Fittipaldi et al. 2012; Staats et al. 1997). Disease syndromes in swine also include arthritis, pneumonia, endocarditis, encephalitis, polyserositis, abscesses and abortion (Wertheim et al. 2009). Death occurs within hours of the onset of clinical signs in pigs with peracute, i.e. very violent or acute forms of infection. Acute disease typically characterized by fever (up to 42 °C), depression, anorexia and lassitude may result in deaths, chronicity, or healthy carriers. In its chronic form, lameness and/or residual central nervous system signs may be apparent (Fulde and Valentin-Weigand 2013). Clinical manifestations of *S. suis* are observed to vary by geographical location (Wangkaew et al. 2006; Yu et al. 2006; Tang et al. 2006). There have been varying reports on the incubation period of *S. suis* ranging from 3 h to 14 days (Yu et al. 2006), and 60 h to 1 week (Mai et al. 2008). Short incubation periods are found to be consistent with direct entry of *S. suis* into the blood stream through skin wounds. There have been no consistent findings in seasonal variation of *S. suis* infection (Wangkaew et al. 2006; Mai et al. 2008; Huang et al. 2005).

S. suis infection is reported in domesticated pigs (Staats et al. 1997). In addition, the organism has been isolated from the intestinal flora of wild boars, dogs, cats, horses, deer and ruminants (Staats et al. 1997; Devriese et al. 1992; Baums et al. 2007; Devriese and Haesebrouck 1992). The rate of asymptomatic carriage in pigs is estimated to be around 80%, representing a potential source of infection to other animals and humans (Lun et al. 2007; Staats et al. 1997; Arends et al. 1984; Ngo et al. 2011). Pigs acquire *S. suis* via vertical and horizontal transmission as the sow is capable of harboring *S. suis* in the genital tract (Fulde and Valentin-Weigand 2013; Fittipaldi et al. 2012; Gottschalk 2011). Carrier rates are highest in

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