Potential Methods of Eliminating Swine Pathogens in Livestock Transport Trailers

Literature Review

For
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Executive Summary

Increased animal transportation has introduced additional challenges to biosecurity. A great deal of attention has been given to transportation biosecurity associated with the outbreaks of Porcine Respiratory and Reproductive Syndrome (PRRS), Transmissible Gasteroenteritis (TGE), and, more recently, the widespread outbreak of the Porcine Epidemic Diarrhea virus (PEDv) that has occurred primarily in the United States. Even though these viruses can be transmitted via several different vectors, the major concern is transportation of the virus from one farm to another or even across international boundaries via livestock transport trailers. Thus, there are two primary goals of this literature review. The first goal is to identify bacterial, viral, and parasitic swine diseases that can easily be transported through animal transport trailers. Along with this, all possible modes of transmission are noted. The second goal is to investigate the methods that have been documented in the scientific literature as being effective in deactivating each of the viruses, bacteria, and parasites of interest. This report identifies ten viral diseases, nine bacterial diseases, and two parasitic diseases that represent economically significant biosecurity challenges for the Canadian swine industry.

The viral diseases of interest include African Swine Fever (ASF), Classical Swine Fever (CSV), Porcine Epidemic Diarrhea (PED), Transmissible Gastroenteritis (TGE), Porcine Reproductive and Respiratory Syndrome (PRRS), Swine Influenza, Rotavirus, Porcine Circoviruses, Aujesky's Disease (Pseudorabies), and Foot and Mouth Disease (FMD). The bacterial diseases of interest include Salmonellosis; Colibacillosis; Brachyspiral colitis, B. hyodysenteriae, and B. pilosicoli; Swine

Erysepelas; Leptospira; Actinobacillosis; Streptococcus; and Haemophilus parasuis (Glasser's Disease). The two parasitic diseases of interest include Sarcoptic Mange (Scabies) and Ascariasis.

It was found that the majority of viruses, bacteria, and parasites are much more resistant to disinfection when there is organic matter such as feces and bedding present. This indicates that an effective biosecurity protocol requires cleaning of the trailer prior to disinfection. In addition, drying of the trailer following disinfection was found to be an important process step. Taken together, the results of this extensive study have shown that low temperature, short contact time, and high organic matter decreased the efficacy of most of the disinfectants tested. It is important to note that no disinfectant is universally effective against all pathogens. However, it appears, based on the literature, that application of a 10% sodium hypochlorite (household bleach) solution in combination with heating to a temperature of 70 degrees for ten minutes has the potential to be effective in deactivating the majority of viruses, bacteria, and parasites.

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1. Introduction

The Food and Agricultural Organization (FAO) has defined biosecurity as "a strategic and integrated approach to analyze and manage relevant biological risks to human, animal and plant life or/and health and the associated risks to environment" (FAO, 2007). The concept of biosecurity started to make an appearance in the late 1990's with an extensive literature review and series of studies conducted by Amass and colleagues from Purdue University on the importance of biosecurity as a tool to control the transmission of diseases, and the effectiveness of commonly used disinfectants to inactivate or eliminate pathogens (Amass, 2004; Amass and Clark, 1999; Amass et al., 2000a; Amass et al., 2000b). Since then, farm biosecurity has become an essential part of farm management in the pork industry, with an overall objective of preventing the introduction of new diseases to the farm and controlling the spread of endemic diseases within and between farms. As extensively discussed by (Levis and Baker, 2011), biosecurity measures may vary from farm to farm. Despite this, every effective biosecurity plan should include the following measures: bio-exclusion, bio-management, and bio-containment (Levis and Baker, 2011). Levis defined bio-exclusion as measures taken by a farm to prevent the introduction of a new disease to the swine herd in the farm, while bio-management refers to the efforts made by the farm management to minimize the economic impact of endemic diseases in a given farm using different management practices. The focus of most hog producers is on the first two biosecurity measures. However, bio-containment is equally important, not only to protect a given farm from new diseases but also to prevent the spread of diseases into

neighboring farms. In addition to protecting a given farm or neighboring farms, bio-containment is the most effective method of preventing the introduction of trans-boundary animal diseases (TAD) into Canada. It is also important to notice the importance of bio-containment in preventing consumers from contracting zoonotic animal diseases. All of the three biosecurity measures are very important, and it is difficult to effectively apply one without considering the other. The major focus of this review, however, will be on bio-containment, with the aim of preventing the introduction of diseases into a country, a region, or a farm, primarily through the trailers used for live animal transportation.

Animal transportation is becoming a major concern of biosecurity, and a great deal of attention has been given to transportation biosecurity in association with the outbreaks of porcine respiratory and reproductive syndrome (PRRS), transmissible gastroenteritis (TGE) and, more recently, the widespread outbreak of the porcine epidemic diarrhea virus (PEDv) that has occurred primarily in the United States. Even though these viruses can be transmitted between animals or farms through several different vectors, the major concern is transportation of the virus from one farm to another or even across boundaries through livestock transporting trailers. Thus, the main goal of this literature review is to identify bacterial, viral and parasitic swine diseases that can easily be transported through animal transporting trailers for the purpose of determining physical or chemical conditions that can be applied on transportation vehicles to inactivate or eliminate the pathogens. The ultimate goal of this project is to develop an automated system for effectively cleaning and disinfecting livestock transport vehicles to allow for improved biosecurity in livestock transportation.

2. Viral diseases

2.1. African swine fever

African Swine Fever (ASF) is a highly contagious hemorrhagic viral infection of pigs that is endemic to the continent of Africa. The disease is still causing serious problems in some Sub-Saharan African countries, but outbreaks have also been reported in Europe, Central Asia, South America and the Caribbean that consumed a huge amount of financial resources for eradication by stamping out all pigs in the outbreak area (Penrith and Vosloo, 2009). The disease has not yet been reported in North America and most Western countries, but, with the existing globalization and increasing trade relations among countries, there is serious concern that ASF could soon be introduced into the North American hog industry.

2.1.1. Etiology and mode of transmission

The etiology of African swine fever is a double stranded DNA virus, which belongs to the genus *Asfivirus* in the *Asfarviridae* family. The virus affects mainly the domesticated pig, but can also affect the European wild boar and the American wild pig. In Africa, warthogs and bush pigs are reservoir hosts with a persistent subclinical infection, and they are important sources of infection to domestic pigs (Spickler, 2010). Clinical signs range from hyper acute/acute infection with close to 100% mortality in domesticated pigs to subclinical infection that cannot be diagnosed based on clinical signs in the reservoir hosts. Soft ticks of the genus *Ornithodoros* are believed to be the biological vector of the virus. However, the virus can easily spread among pigs through direct pig-to-pig contact and may be spread indirectly through contaminated fomites and livestock transport vehicles (Oura, 2013).

2.1.2. Environmental survival and susceptibility to disinfectants

The virus is relatively resistant to environmental conditions and can remain viable for several days in pig products and the environment, particularly if the virus is covered with organic matter. It is highly resistant to cold temperatures and can survive for more than a year in blood and other animal products stored at 4^{o}C. It can also survive for several years in a frozen carcass. The virus can also survive for more than a month in contaminated pig pens and more than 11 days in feces at room temperature (EAZWV, 2011). The virus is also resistant to a wide range of pH (3.9 -11.5), particularly if it is covered with organic matter like blood and feces (Spickler, 2010). On the other hand, the virus is highly sensitive to high temperature and can be inactivated by heating at 56^{o}C for 70 minutes and within 20 minutes at 70^{o}C (OIE, 2012). Like any other enveloped virus, ASFv is reported to be susceptible to a wide variety of lipid solvent detergents and disinfectants including ether, chloroform, iodine, formalin, phenols, and quaternary ammonium compounds (EAZWV, 2011). In agreement with this, several in vitro studies have shown that the virus can be inactivated easily in less than 30 minutes with common disinfectants such as sodium hypochlorite and citric acid (Krug et al., 2011), as well as sodium hydroxide and calcium hydroxide (Turner and Williams, 1999). However, all these results are from in vitro studies and need to be interpreted with caution. In line with this, an extensive study from the old literature that used more than 10 disinfectants has concluded that, despite the ability of several disinfectants to inactivate ASF virus in vitro, only One-Stroke Environ, a phenolic compound, applied as a spray at 1% concentration was able to completely inactivate ASFv when applied in a contaminated pig room (Stone and Hess, 1973) This suggests that only disinfectants specifically approved for African Swine Fever should be used for effective decontamination during disease outbreaks.

2.2. Classical Swine Fever

Classical Swine Fever (CSF), also known as hog cholera, is a highly contagious disease of swine that causes systemic infection characterized by high body temperature, viraemia, vomiting, diarrhea, and purple skin discoloration around the ear and the lower abdomen. The virus affects both wild and domesticated pigs and is notifiable to the World Organisation for Animal Health (OIE) due to its high economic importance. Despite its worldwide distribution, most developed countries, including Canada, have eradicated the disease from swine herds and are declared free of CSF. However, due to the endemic nature of the disease in most of the countries of South and Central America, the risk for introduction of CSF to North America is still high.

2.2.1. Etiology and Transmission

Classical Swine Fever is caused by an enveloped RNA virus that belongs to the genus *Pestivirus* of the *Flaviviridae* family. Infected animals shed the virus in all body secretions and excretions like blood, semen, urine, feces, nasal and ocular discharges, as well as saliva. The virus is highly contagious and reproduction ratios (R_0) of 100 and 15.5 were reported within pens for weaned pigs and slaughter pigs, respectively (Klinkenberg et al., 2002). The most efficient method of transmission is by the oronasal route, either through direct contact between healthy and infected pigs or by indirect transmission through contaminated feed, such as swill (Moennig et al., 2003). Vertical transmission via contaminated semen from infected boars is also possible (Floegel et al., 2000). Furthermore, indirect transmission due exposure to contaminated fomites like pens, buckets, farm equipment, boots, clothing, veterinary and insemination equipment, as well as transport vehicles, is well documented (Edwards, 2000). For instance, contaminated vehicles are believed to be responsible for spreading the CSFv to uninfected herds during the 1997 CSF

outbreak in the Netherlands (Stegeman et al., 2000). Mechanical transmission via vectors such as flies, rodents, and birds is also a concern (Edwards, 2000). The virus has been isolated from air samples originating from infected pigs (Weesendorp et al., 2008) and aerosol transmission in the close confinement of crowded farms is a major means of direct transmission. There is no good evidence describing how far the virus can travel through the air; however, there are several reports that show airborne transmission might have reached farms within a 0.25 to 6 Km radius of an infected herd (Roberts, 1995). The general consensus is that farms within a radius of 1 km from the infected herd are considered to be at a very high risk (Ribbens et al., 2004b).

2.2.2. Environmental survival and susceptibility to disinfectants

Like other enveloped viruses, CSF virus is moderately fragile and does not persist in the environment for a prolonged period of time. It is very difficult to determine the exact length of time the virus can survive in the environment as this is influenced by the level of moisture, pH and temperature; however, studies show that it can survive in contaminated pens for about 4 weeks under winter conditions with no exposure to direct sunlight. It can also survive for months and even years in refrigerated or frozen meat, respectively (Ribbens et al., 2004a). An extensive review of the old literature by Steven Edwards on the survival and inactivation of CSF virus has shown that the virus is highly sensitive to drying and ultra-violet light. It is also susceptible to heat and can be easily inactivated within 30 minutes at a temperature greater than $65^{O}C$. The virus is also susceptible to extreme pH conditions and can be rapidly inactivated at pH $\leq$3 and pH $\geq$11, but it is generally stable at neutral to slightly alkaline conditions in the pH 5 to 10 range. Furthermore, like all enveloped viruses, CSF virus is easily inactivated by detergents and organic solvents like ether and chloroform. A wide range of chemicals, including sodium hypochlorite,

phenolic compounds, quaternary ammonium compounds, and aldehydes are used to disinfect CSF virus in the field (Edwards, 2000). For example, 1000 ppm sodium hypochlorite was found to completely inactivate CSF virus both from metallic and plastic surfaces in field conditions. In contrast, 2% citric acid, which was effective against ASF virus, was not effective against CSF virus (Krug et al., 2011).

2.3. Porcine Epidemic Diarrhea

Porcine epidemic diarrhea (PED) is a highly contagious viral disease of swine characterized by rapid onset of diarrhea, vomiting and dehydration. PED is associated with high morbidity and mortality. Mortality can reach 100% in young piglets of less than one week age. The disease is clinically indistinguishable from Transmissible Gastroenteritis (TGE), but there is no antigenic relation or cross protection between the two viruses (Pospischil et al., 2002). The disease was first reported in England in 1971 and had resulted in several devastating disease outbreaks throughout Europe in the 70s and 80s (Chasey and Cartwright, 1978). Porcine epidemic diarrhea was first detected in the United States in May of 2013 on Iowa pig farms (Stevenson et al., 2013). Since then, the disease has been spreading rapidly throughout the USA and Canada. The first confirmed case of PED in Canada was reported in January, 2014 on an Ontario farm (Ojkic et al., 2015).

2.3.1. Etiology and Transmission

The etiology is an enveloped single stranded RNA virus that belongs to the family *Coronaviridea*, genus *Alphacoronavirus*. The virus was first identified in 1978 from the 1970's outbreaks of PEDv in Europe (Pensaert and de Bouck, 1978). However, the recent outbreak in the United States and Canada is believed to have its sources in China (Huang et al., 2013). The strain circulating in the

US since April 2013 is similar to the strain that resulted in a PED outbreak in China between 2010 and 2012 (Chen et al., 2014; Stevenson et al., 2013).The virus is excreted in the feces and the fecal-oral route of transmission is the major route of direct transmission between pigs.

Like any other enveloped viruses, PEDv is moderately fragile, but can survive outside the host for some time under suitable environmental conditions like in organic matter and in cool temperatures. Therefore, contaminated fomites, including transport vehicles, can serve as a mode of indirect transmission of the disease between farms (Lowe et al., 2014). Ingestion of contaminated feed material is suspected as the source of the first PEDv outbreak in Canada (Dee et al., 2014; Pasick et al., 2014). Furthermore, virus genetic material was detected as far as 10 miles from an infected herd, suggesting that airborne transmission is a potential means for disseminating porcine epidemic diarrhea virus among farms (Alonso et al., 2014).

2.3.2. Environmental Stability and Susceptibility to Disinfectants

The survival of the virus in the environment is dependent on several factors including humidity, temperature, pH, and the presence of organic matter. The virus was reported to be stable between a pH of 5 and 9, in a temperature range of 4^{o}C to 37^{o}C in an environment with a suitable organic matter like feces, but was inactivated by heating at 60^{o}C for 30 minutes (Pospischil et al., 2002). A group of researchers, from Iowa State University have shown that, in the absence of proper washing and disinfection, the virus can be completely inactivated in livestock trailers (cleaned by scraping and sweeping only) by heating at 71^{o}C for 10 minutes. However, it took 7 days to completely inactivate the virus when the trailers were left at room temperature (Thomas et al., 2014).

Table showing summary of pig bioassay and PED virus result by treatment

Group	Percentage of PEDV positives (out of four):
Negative control	0% (0/4)
Positive control	100% (4/4)
160F10M	0% (0/4)
145F10M	25% (1/4)
130F10M	25% (1/4)
100F12H	50% (2/4)
68F24H	25% (1/4)
68F7D	0% (0/4)

F, Fahrenheit; M, minute; H, hour; D, days

The above table, taken from (Thomas et al., 2014), shows that treatment of PEDv containing feces at 160^{o}F (71^{o}C) for 10 minutes or at room temperature (60^{o}F) for 7 days completely inactivated the virus and none of the 4 pigs exposed to the heat treated feces were infected. On the other hand, treatment of the virus at a temperature less than 160^{o}F (71^{o}C) for 10 minutes or at room temperature (68^{o}F) for 24 hours did not completely inactivate the virus and 25-50% of the pigs exposed to the heat treated feces were infected.

Considering the conditions discussed above concerning time and temperature combinations required to inactivate PED virus, it is possible that the virus can easily be transmitted by the consumption of contaminated pelleted pig feed (Dee et al., 2014). The typical retention time in the conditioner machine during the pelleting process is 30 to 60 seconds at a temperature range of 71 to 99^{o}C (personal communication). This may suggest a risk that the virus may not be

completely inactivated during the short retention time when the lower temperature range (71^{o}C) is used to pellet pig diets containing porcine products. Therefore, it is recommended either to use the higher temperature range (100 to 121^{o}C) or increase the conditioning time. Further processing of the conditioned feed material with an expander may also help to completely inactivate the virus. However, the Canadian Food Inspection Agency (CFIA) could not confirm a link between feed containing porcine plasma and PED outbreaks in Canada, despite the presence of infectious virus particles in porcine plasma originating from the United States ,which was used for the preparation of pig feed in Canada (CFIA, 2015).

The virus is highly susceptible to direct sun light when it is not protected with organic material like feces. Therefore, UVC irradiation and treatment with ionizing radiations like gamma rays can be used to inactivate the virus in contaminated feed materials or surfaces. There is no published evidence on PEDv to support this hypothesis; however, studies performed on a group of viruses similar to the TGEv suggest that corona viruses, including the PEDv, can be inactivated using a UVC dose of 500 J per m^2 (Terpstra et al., 2008) or by gamma irradiation at a dose of 25 to 35 kGy (Nims et al., 2011). Similarly, like other enveloped viruses, PEDv can be easily inactivated using ionic and non-ionic detergents, as well as organic solvents such as ether and chloroform. The virus is also susceptible to most virucidal disinfectants including cresol, sodium hydroxide (2%), formalin (1%), sodium carbonate (4% anhydrous or 10% crystalline, with 0.1% detergent), strong iodophors (1%) in phosphoric acid, oxidizing agents like potassium peroxymono-sulfate and sodium hypochlorite, and phenolic compounds (Pospischil et al., 2002). These results are from an in vitro study and results may be different under natural conditions in the environment where the virus is usually covered with organic matter like feces. For example, a report from Iowa State

University researchers has shown that the use of a common virucidal disinfectant, Stalosan F powder, was not an effective means of inactivating PEDv in scraped, but unwashed livestock trailers (PorkCheckoff, 2013). These results reiterate the importance of the gold standard inactivation method that includes washing, disinfection, and drying of trailers. However, a recent study conducted at Iowa State University and published in 2015 by the PorkCheckoff showed that under circumstances when washing, disinfecting, and drying the trailer was not possible, full inactivation of the virus in trailers was accomplished within 40 minutes of contact time by using accelerated hydrogen peroxide® (AHP®) disinfectant, which is sold under the brand name Accel®, at a minimum concentration of 1:32 in a 10 percent propylene glycol solution (PorkCheckoff, 2015).

2.4. Transmissible Gastroenteritis

Transmissible gastroenteritis (TGE) is a highly contagious, acute, rapidly spreading viral infection of the intestinal tract of pigs. It multiplies in and damages the enterocytes covering the small intestine, producing villus atrophy and enteritis. The disease is characterized by diarrhea, vomiting, dehydration and high mortality in piglets less than 2 weeks of age. TGE is clinically indistinguishable from Porcine Epidemic Diarrhea virus (PEDv), but the two corona viruses are not related to each other (Neumann, 2014).

2.4.1. Etiology and Transmission

TGE is caused by a virus that belongs to the genus C*oronavirus* of the *Coronaviridae* family. The natural host is the domestic pig, but wild and domesticated carnivores can also be infected and act as reservoir hosts. The virus is shed in feces and the main route of transmission is direct pig-to-pig contact by the fecal-oral route. Indirect transmission through contaminated fomites,

including transportation vehicles, is also possible, particularly during winter because of the improved survival of the virus in the environment in cold temperatures. The virus was also isolated from flies (Gough and Jorgenson, 1983) and dogs (McClurkin et al., 1970), and, therefore, mechanical transmission by flies and other animals, including birds and rodents, is a major concern.

2.4.2. Environmental survival and susceptibility to disinfectants

The virus is highly susceptible to direct sun light; and thus cannot survive in the environment for a prolonged period of time. The virus is generally susceptible to high temperatures and can be easily inactivated by temperatures above 30^{o}C (Laude, 1981). There are also unpublished reports which show that the virus can be fully inactivated by heating at 56^{o}C for 30 minutes or at 65^{o}C for 10 minutes. However, the virus is resistant to freezing cold weather, and this results in seasonal outbreaks during the winter (Harris, 2013). The virus is highly susceptible to almost all virucidal disinfectants, including iodides, peroxygen, quaternary ammonium compounds, phenol and sodium hypochlorite (Brown, 1981). Thus, a good biosecurity policy of washing, disinfection and drying of equipment, including animal transport vehicles, can effectively control the spread of the disease between herds.

There are two other swine diseases worth mentioning here, caused by the same group of corona viruses but with a different degree of severity and economic impact. The first one is the Porcine Respiratory Corona Virus (PRCV) infection caused by a virus identical to the TGE virus that lost its tropism and virulence to the intestines; it replicates only in the lung interstitial cells. The virus causes only mild respiratory infection in young piglets of 2-3 weeks age, but its economic importance lays on its ability to quickly spread by air between farms and its cross reaction with

TGE virus, which makes it difficult to differentially diagnose TGE outbreaks (Neumann, 2014). A second disease caused by the same corona virus group that affects both the intestinal and respiratory tract of young pigs was reported in Ohio and Indiana in February of 2014. The disease rapidly spread to other states in the United States and Canada and caused a significant economic loss to the swine industry in 2014. The etiology is a novel corona virus known as porcine deltacorona virus (PdCV), which is closely related to the TGE and PED viruses and is believed to have originated in Hong Kong and South Korea (Ma et al., 2015).

2.5. Porcine Reproductive and Respiratory Syndrome (PRRS)

PRRS is a highly infectious and the most economically significant disease of pigs in North America, which is costing hundreds of millions of dollars every year to the swine industry. The total cost of productivity losses due to PRRS to the US pork industry was estimated to be around $560 million in 2005 (Neumann et al., 2005), and this increased to $664 million in 2011 (Holtkamp et al., 2013). The disease was first reported in North America in the 1980s, and spread rapidly throughout the world in a very short period of time. The disease has now been reported in almost all countries with the exception of Australia, New Zealand and the Scandinavian countries (OIE, 2008). Clinically, it is characterized by overlapping symptoms of the reproductive system (failure or impaired breeding in sows, gilts, and even boars) and respiratory illness in pigs of all age (Neumann, 2014).

2.5.1. Etiology and Transmission

The etiology of PRRS is an enveloped, single stranded and positive sense RNA virus classified under the family Arteriviridae and the genus *Arterivirus* viruses. The virus affects only pigs, and no other mammalian or arthropod host acts as a biological vector for the transmission of the

disease. The virus is shed in all body secretions and excretions of the infected pig including milk, blood, semen, saliva, nasal discharge, urine and feces. The main route of transmission is through direct contact between pigs through the oronasal route or through artificial insemination (OIE, 2008). Indirect transmission through contaminated fomites is also a major problem in the control of PRRS. A group from the University of Minnesota conducted extensive research to determine the mechanical vectors for the transmission of PRRSv and compiled evidence that the virus can be carried from pen to pen within a farm or between farms with contaminated fomites such as farm equipment, clothing, boots, veterinary equipment (Otake et al., 2002b), house flies and mosquitoes (Otake et al., 2004; Otake et al., 2002c; Pitkin et al., 2009b), as well as transport vehicles (Dee et al., 2004b; Dee et al., 2007). The same group also identified the airborne transmission potential of the PRRS virus to be as long as 3.3 km (Otake et al., 2002a; Pitkin et al., 2009a) and developed an air filtration system to control aerosol transmission of the virus (Dee et al., 2005a; Dee et al., 2009b).

2.5.2. Survival in the environment and susceptibility to disinfectants

Comparable to other enveloped viruses, PRRSv is moderately fragile in the environment and can only survive for a few hours if not covered by organic materials like feces or bedding. The stability of the virus in the environment depends on several factors including moisture, temperature and pH. (Bloemraad et al., 1994). According to this report, the virus was stable for an extended period of time at pH 6.5-7.5, but infectivity was rapidly lost at pH values below 6 and above 7.5. Similarly, the virus was stable at lower temperatures and remained viable for up to 4 months in temperatures between -70 to -20^{o}C. Viability decreased with increasing temperature and almost 90% of the viruses were inactivated within a week when stored at 4^{o}C; however, few infectious

virus were able to survive for about 30 days at 4^{o}C (Bloemraad et al., 1994). In another report, PRRS virus was reported to survive in a solution for weeks at 4^{o}C, for about 1-6 days at room temperature, and for 24 hours at 37^{o}C. Its viability decreased with increasing temperature and was completely inactivated within 20 minutes at 56^{o}C (Zimmerman et al., 2012). The virus is also highly susceptible to direct sunlight (Benfield et al., 1992).The virus is also sensitive to almost all lipid solvent detergents and virucidal disinfectants; however, a combination of Quaternary ammonium compound and glutaraldehyde (synergize 0.8%) or a modified potassium monopersulfate (Virkon 1%) are the recommended disinfectants for use under natural farm conditions (Pitkin et al., 2009c).

A good biosecurity program at the national and farm level can effectively control the spread of PRRS and over the years the focus has been on the role of livestock transporting trailers as a major means of spreading the virus between farms and biosecurity protocols that can be applied effectively on trailers and drivers. A group led by Scott Dee from the Swine Disease Eradication Center at the University of Minnesota College of Veterinary Medicine has developed a model to study the role of trailers in the spread of the virus and demonstrated healthy pigs could become infected with PRRSV through contact with the contaminated interior of the transport vehicle (Dee et al., 2004b). Using this model, they developed a number of sanitation protocols to disinfect PRRSV contaminated full size trailers applicable at room temperature (Dee et al., 2004a) and cold temperatures (4^{o}C and -20^{o}C) representing winter conditions (Dee et al., 2005b). The conclusion from all these studies is that livestock transporting vehicles could be effectively cleared from PPRS virus by washing with cold water and drying for 8 hours at room temperature instead of the usual overnight drying time. Furthermore, washing plus fumigation with a combination of

glutaraldehyde and quaternary ammonium chloride (Synergize) was able to completely inactivate PRRS virus within 90 minutes on the interiors of livestock transport trailers. The effect of Synergize was compromised under freezing weather (-20^{o}C) conditions unless it was used with antifreeze reagents such as a 10% propylene glycol or a 40% methanol solution (windshield washer fluid).

2.6. Swine Influenza

Influenza is a viral infection of birds and mammals, including humans. Swine influenza is a highly contagious and economically important disease that can infect almost all pigs in a herd within 1-3 days. In the absence of secondary complicating bacterial infections, the mortality rate is generally very low, ranging between 1% and 4%, (Dee, 2014), but the high morbidity of infected pigs results in high production losses to the hog industry. As shown on The Pig Site, Holtkamp and colleagues estimated the economic losses due to swine influenza in 2007 to be only second to PRRS (Anonymous, 2013). Moreover, swine influenza is important because of its zoonotic significance (Khan et al., 2013); the virus has been isolated from lung tissues of man (Smith et al., 1976). In addition to this, pigs possess pulmonary epithelial cells with receptors that can bind both to avian and human influenza viruses (Ito et al., 1998). Therefore, it is possible that different types of influenza viruses infecting the same host cell in the pig mix and produce novel strain of virus by genetic re-assortment (Castrucci et al., 1993). Due to this, pigs are considered as “mixing vessels” for human and avian influenza viruses (Thacker and Janke, 2008). The new viruses that arise from genetic re-assortment as a result of antigenic shift or antigenic drift are highly virulent type of influenza viruses that can cause severe and fatal disease outbreaks even in the natural hosts. A typical example is the H3N2 influenza virus reported in the United States swine

population in the late 90's and was composed of genes from swine, avian, and human influenza viruses (Webby et al., 2000) .

2.6.1. Etiology and Transmission

Influenza in birds and mammals is caused by enveloped and single stranded negative-sense RNA viruses that belong to the *Orthomyxoviridae* family. There are three types of influenza virus affecting mammals and birds, designated as influenza type A, B, and C. While type B and C are limited to humans and result in mild respiratory infection, type A is wide spread in birds and mammals, including man. The natural hosts for influenza A virus are wild aquatic birds, but it can also affect domestic birds, swine and humans (Webby and Webster, 2001). There are many subtypes of influenza A viruses, which differ based on the combination of two glycoproteins on the surface of the influenza virus designated as protein 'H' (Hemagglutinin) and protein 'N' (Neuraminidase). There are at least 16 H (H1-H16) and 9 N (N1-N9) proteins, and many combinations of H and N proteins are possible (Fouchier et al., 2005). The three subtypes of influenza A virus that cause infection in swine are H1N1, H1N2 and H3N2 (Ma and Richt, 2010). The first influenza virus isolated from pigs was the H1N1 subtype known as the classical swine influenza virus (Myers et al., 2007). Even though all three subtypes of the swine influenza can affect humans, most of the recent outbreaks particularly the 2009 pandemic in humans, are widely believed to be related to the lineage of the swine H1N1 influenza virus (Dawood et al., 2009). The virus is spread among pigs by aerosols, through direct contact between pigs, and through indirect contact with contaminated fomites. Transmission may also occur between humans and pigs as well as other animals and pigs (Tellier, 2006).

2.6.2. Environmental resistance and susceptibility to disinfectants

Mammalian influenza viruses in general, including swine influenza viruses, are labile in the environment and can only survive for 8 to 24 hours on the surface of materials depending on the nature of the surface (Greatorex et al., 2011); however, an earlier report has shown that human influenza A virus could survive for more than 3 days on the surface of bank notes (Thomas et al., 2008). Survival of influenza viruses in the environment is influenced by several factors such as presence of organic matter, temperature, humidity and pH. For example, swine influenza viruses survived for 9 weeks in a fecal slurry stored at 4^{o}C, 2 weeks at 20^{o}C, but only for 1 to 2.5 hours at 50 to 55^{o}C (Haas et al., 1995). In contrast, influenza A viruses were inactivated by exposure to direct sunlight for 30 minutes and by heating at 56^{o}C for 30 minutes, 60^{o}C for 10 minutes and 70^{o}C or 1 minute (Zou et al., 2013). Influenza viruses are also susceptible to a wide variety of disinfectants including sodium hypochlorite (1:10 dilution of household bleach), aldehydes (formalin, glutaraldehyde, formaldehyde), quaternary ammonium compounds (Lysol, No-Rinse sanitizer), phenolic compounds (Tek-Trol and One-Stroke Environ), peroxygen compounds (Virkon-S), 70% ethanol, oxidizing agents and lipid solvents (Suarez et al., 2003). Even though trans-species transmission of the virus may complicate control of swine influenza, good biosecurity and sanitation measures at a farm level can be effective in eradicating influenza from a given farm.

2.7. Rotaviral enteritis

Rotaviral enteritis is a disease that affects the small intestine of animals and humans. It was first identified in calves in 1969 and subsequently reported from humans and pigs (Neumann, 2014). Porcine rotavirus enteritis is reported in all age groups of pigs; however, it is a major cause of

diarrhea in nursing or post-weaning piglets. Passive immunity through colostrum is believed to protect piglets in the first 7 to 10 days of life, and hence rotavirus diarrhea outbreaks usually occur in post weaning or later stage nursery piglets (Wieler et al., 2001).

2.7.1. Etiology and Transmission

The etiological agent, rotavirus, is a non-enveloped double stranded RNA virus that belongs to the *Reoviridae* family of viruses. There are 7 serotypes of rotavirus designated A to G based on the main viral protein (VP6) of the intermediary capsid layer (Yuan et al., 2006). Even though serotypes A, B and C have been reported from pigs (Medici et al., 2011), the most important cause of diarrhea in young piglets is rotavirus group A (Will et al., 1994). The viruses are excreted in the feces of infected piglets and transmission is mainly through direct contact by the fecal-oral route. The viruses are resistant to the external environment and were isolated from dust and fomites of a nursery room that had been free of pigs for three months (Fu et al., 1989), suggesting that the spread of the virus to long distance farms either by aerosol or via contaminated fomites, including vehicles, is a big concern.

2.7.2. Environmental resistance and susceptibility to disinfectants

The virus is shed in feces and can stay viable for an extended period in manure or manure contaminated environment. For example, the virus was reported to stay viable for 7 to 9 months at room temperature and even for years when the feces were stored at a lower temperature in a pH range of 3 to 9 (Ramos et al., 2000). Based on data from different published articles, it was concluded that the viral particles are able to remain infectious for an extended period of time (enough time to infect other hosts) in the air, soil, water, and fomites (CAST, 2008). However, the virus can be easily inactivated by heating at 50^{o}C for 5 minutes (Ramos et al., 2000). Rotaviruses

are also highly susceptible to UV and gamma irradiation (Ojeh et al., 1995). Rotaviruses are non-enveloped viruses and hence relatively resistant to inactivation by lipid solvent such as ether and chloroform, as well as, nonionic disinfectants such as sodium hypochlorite and formaldehydes. However, 95% ethanol, phenols, formalin and chlorine compounds have been reported to be very effective in killing the virus (Ojeh et al., 1995; Yuan et al., 2006). Chlorine at a concentration of 0.2 to 0.3 mg/L was found to be the best disinfectant to inactivate rotaviruses in water (Vaughn et al., 1986).

2.8. Porcine Circovirus Associated Disease

In the mid 90's, pig farmers in North America and Europe reported a disease similar to the previously described postweaning multisystemic wasting syndrome (PMWS), but causing more severe illness characterized by rapid weight loss and higher rates of mortality in finisher pigs (Harding, 1996; Segales et al., 1997). The disease was manifested as a syndrome affecting the respiratory, intestinal and reproductive tracts and the full expression of the disease sometimes required a concurrent viral infection with PRRSv, Porcine Parvovirus, and TGEv (Allan and Ellis, 2000). An ad hock committee established by the American Association of Swine Veterinarians (AASV) identified the new Porcine Cirocvirus in 2006 and subsequently designated the virus and the disease as PCV2 and Porcine Circovirus Associated Disease (PCVAD), respectively (Halbur, 2006).

2.8.1. Etiology and Transmission

Porcine Circoviruses (PCV) are extremely small, non-enveloped, and single-stranded circular DNA viruses found in pigs throughout the world. This original PCV, now termed PCV1, is nonpathogenic to pigs and was usually reported as a common laboratory contaminant

(Neumann, 2014). A new genotype of circovirus associated with a severe and fatal PMWS was reported in the late 1990's and subsequently designated as PCV2 by the AASV in 2006 (Halbur, 2006). The virus is excreted in almost all body secretions of the pig including nasal, ocular, saliva, urine, feces (Patterson et al., 2011b; Yang et al., 2003) and even semen from infected boars (Larochelle et al., 2000). Therefore, transmission is mainly by direct nose-to-nose contact between pigs through the nasal-oral route or indirectly through contaminated fomites including artificial insemination with semen containing a high viral load (Rose et al., 2012). There is no clear evidence yet that show porcine circoviruses can spread to distant farms or between pens in a given farm by aerosol transmission; however, detection of the virus from air samples taken from swine confinement buildings in Canadian pig farms (Verreault et al., 2010) strongly suggests that airborne transmission of PCV2 is a possibility.

2.8.2. Environmental stability and susceptibility to disinfectants

Porcine circovirus 2 (PCV2) is a hardy virus that can survive in the environment under a wide range of pH and temperature (Welch et al., 2006). It can resist heating at 56^{o}C for 1 hour and it required heating at 70^{o}C for 6 hours to completely inactivate the virus (Kim et al., 2009). In addition to this, Kim and colleagues showed that PCV2 is also resistant to many lipid solvent disinfectants and irradiation. As shown in the figure 1 below, out of the 8 disinfectants tested, only Virkon S (1:100), Clorox Bleach (1:23), and 3% Sodium Hydroxide were the most effective disinfectants that completely inactivated the virus within 10 minutes of contact time. Roccal D Plus from Pfizer animal health (1:256) and Synergize (1:256) were also able to kill the virus in 30 minutes and 12 hours respectively (Kim et al., 2009). A similar experiment conducted in vitro reported only partial reduction in virus titers following a 10 minutes exposure of PCV 2 virus to

the same disinfectants (Royer et al., 2001). However, the highest effect was also observed with Virkon S, Sodium hydroxide, Roccal D Plus, and Clorox Bleach, but only resulted in 74%, 61%, 50% and 46% virus titer reduction, respectively (Royer et al., 2001).

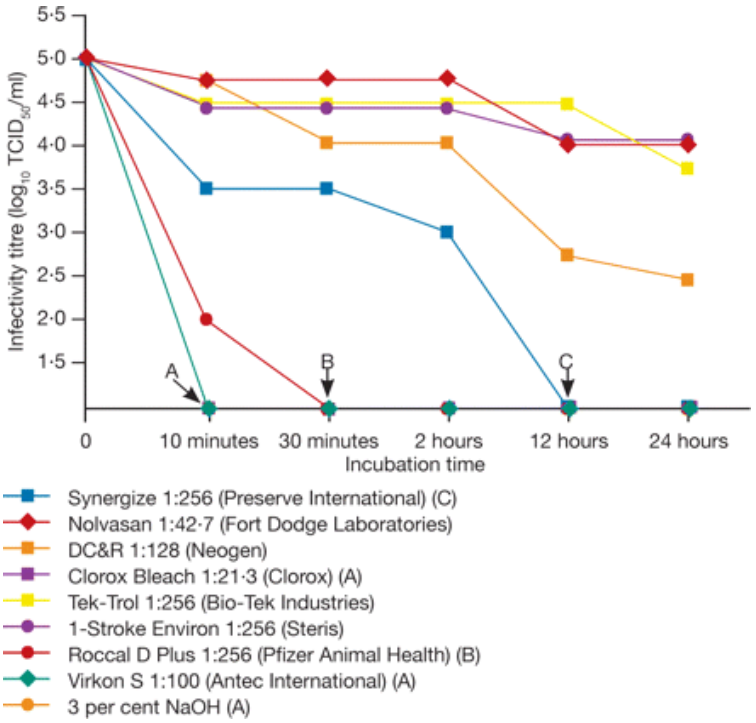


FIG 1: Reduction of porcine circovirus type 2 (PCV-2) infectivity following treatment with eight different disinfectants, at a final dilution in line with manufacturers' recommendations, or 3 per cent sodium hydroxide. Arrows indicate the time of complete inactivation of PCV-2 by certain disinfectants (source Kim, *et al*, 2009)

These results coming from in vitro studies need to be interpreted with caution, as the effect of the disinfectant in the field could be affected by several factors. For example, Patterson and colleagues (Patterson et al., 2011a) tried a combination of four different livestock transporting trailer washing and disinfection protocols using disinfectants reported to be effective in the in vitro studies. Their results showed that none of the disinfectants used alone was better than washing only to inactivate the PCV 2 viruses in the trailer, but a combination of oxidizing agents

followed by sodium hypochlorite was enough to reduce the number of viruses to a level where they were not able to induce infection in naïve pigs kept for some time in the trailer. The ability of PCV2 to resist common disinfectants and its ubiquitous distribution makes it difficult to effectively control PCVAD through strict biosecurity measures; however, studies in France by Madec and colleagues have shown that a list of management measures, now known as Madec's 20-point plan, were effective in managing PCVAD and minimizing its economic impact in farms (Madec et al., 2000). Furthermore, there are now commercially available effective vaccines that can help in the fight against PCVAD in pig farms (Chae, 2012).

2.9. Pseudorabies (Aujesky's Diseases)

Aujesky's disease is a highly contagious and economically important disease of pigs that cause a fatal central nervous system (CNS) infection and high mortality in young pigs. It also causes respiratory and reproductive diseases in adult pigs. The natural host is the pig, but nearly all mammals except horses can be infected, resulting in fatal CNS infection (Neumann, 2014). The disease does not pose a significant risk to human health (Spickler, 2006).

2.9.1. Etiology and Transmission

The etiology of Pseudorabies, the Aujesky's diseases virus (ADV), is an enveloped, double stranded DNA virus, which belongs to the genus *Varicellovirus* of the Herpesviridae family. The virus is distributed globally, but has been eradicated from most of the developed countries including Canada and USA; however, the presence of the virus in feral pigs in North America is a major concern for hog farmers in the US and Canada (Spickler, 2006). The virus is shed in most body secretions including saliva, nasal discharges, urine, and semen. Hence, transmission is mainly by direct nose-to-nose contact between pigs or through indirect contact with

contaminated fomites, including feed (Spickler, 2006). Long distance airborne transmission (Christensen et al., 1990; Scheidt et al., 1991) as well as venereal transmission in feral swine (Romero et al., 2001) were also reported.

2.9.2. Environmental resistance and susceptibility to disinfectants

The virus is fairly resistant outside the host and can survive for several days in a moist and cool environment. An extensive review by Whittmann showed that the virus is stable over a wide pH range (5.0 to 12) and can survive for about 6 to 9 weeks at room temperature, about 9 weeks at 15°C, 20 weeks at 4°C and for years at -40°C. However, the virus is inactivated within 30 to 60 minutes at 60^{o}C, within 10 minutes at 70^{o}C, within 3 minutes at 80°C and within a minute at 100°C (Wittmann, 1985). The virus is rapidly inactivated by direct sunlight and drying. Furthermore, the virus is highly sensitive to most commonly used disinfectants like phenols, bleach, iodine based disinfectants, formaldehyde and quaternary ammonium compounds (Spickler, 2006). However, chlorine based disinfectants were reported to be the best disinfectants; a 3% chloramine solution inactivated the virus within 10 min and a 1% solution within 30 min (Brown, 1981; Lee and Wilson, 1979). In contrast, the use of caustic soda solution is not advised, since the virus was not completely inactivated by 1% NaOH even after 6 hours of contact time (Wittmann, 1985). Therefore, good biosecurity measures supplemented by control of wild animals and birds can be effective in preventing a herd from contracting the disease.

2.10. Foot and Mouth Disease

Foot and mouth disease (FMD) is a highly contagious vesicular disease of cloven-hooved mammals including domestic and wild pigs. In pigs, the disease is characterized by vesicular lesions on the feet, snout and around the mouth. All age groups can be affected, but mortality is

high only in young animals. Although infected animals usually recover, the morbidity is very high and results in huge economic losses to the livestock industry (Neumann, 2014). Economic losses associated with FMD can be direct, as a result of decrease in meat and milk production, or indirect economic losses associated with diseases control measures. In addition, there are losses due to the trade embargoes imposed on FMD endemic countries that make these countries un-eligible to export animals and animal products to disease free countries. A recent review (Knight-Jones and Rushton, 2013) on the economic impacts of FMD has estimated losses in FMD endemic regions to be between USD 6.5 and 21 billion per annum, while sporadic outbreaks in disease-free countries could cost them around USD 1.6 billion a year. Because of the potential that FMD viruses have for rapid spread within and between countries, and the associated economic impacts that include trade barriers between endemic and disease-free countries, FMD is one of the transboundary animal diseases (TAD) that should be reported to the world organization on animal health, OIE (Leforban and Gerbier, 2002).

FMD has been eradicated from North America in the early 1950s by a combination of vaccination and stamping out measures (Sutmoller et al., 2003); however, the disease is still devastating the livestock industry in developing countries, particularly in Sub-Saharan Africa (Sinkala et al., 2014), South East Asia (Perry et al., 1999) and South America(Clavijo et al., 2015). Despite the FMD free status of Europe, an outbreak in 2001 in the UK has resulted in the death of close to 10 million livestock that cost the UK economy around 8 billion pounds (Kao, 2003). More recently, there was a devastating outbreak in East Asian countries including Japan, South Korea and Taiwan as a result of the spread of the virus from endemic countries in mainland South East Asia (Knowles et al., 2012; Valdazo-Gonzalez et al., 2013). Given the endemic nature of the disease in South

America, there is always a risk that FMD can spread into North America including Canada. Therefore, it is important to know about the disease (epidemiology, routes of transmission and control measures) so that appropriate biosecurity measures will be set at all levels to prevent introduction of the disease into the Canadian livestock industry.

2.10.1. Etiology and transmission

The etiology of FMD is a non-enveloped, positive sense single stranded RNA virus that belongs to genus *Alphthavirus* of the picornaviridae family. There are seven different serotypes of FMD virus designated as O, A, C, South African Type 1 (SAT 1), SAT 2, SAT 3 and Asian 1. Serotypes A and O are the most common serotype responsible for most of the outbreaks in Africa, South East Asia and South America. In contrast, serotype C is very rare and has not been reported from any country since 2004 (Spickler, 2014). Cross protection between serotypes is very rare and specific vaccines should be prepared during outbreaks. There are more than 60 strains within these serotypes and cross protection between strains is variable. The FMD virus common in pigs is the Cathay strain within the serotype O group (Spickler, 2014).

The virus is shed in all secretions and excretions from an acutely infected animal, including saliva, milk, urine, feces, expired air and semen, as well fluids from the vesicular lesions in the mouth and feet area. The FMD virus is the most contagious virus known (Grubman and Baxt, 2004). Transmission of FMD virus can occur as a result of direct contact between healthy and infected pigs through skin abrasions or aerosol. Indirect transmission through contaminated fomites, including ingestion of FMD virus contaminated swills is also possible. However, the primary route of transmission is through the respiratory tract, as a result of inhalation of virus-containing aerosols arising from secretions and excretions of infected animals. Pigs respire more infectious

FMD virus than other animals, and they are an important source of airborne transmission for cattle, which are highly susceptible to aerosol infection than pigs are (Alexandersen et al., 2012). The virus can easily spread via air under suitable environmental conditions, such as high relative humidity, steady wind and level topography. Airborne transmission up to a distance of 50km overland (Gloster et al., 2005) and 200km over water (Gloster et al., 1982) has been previously reported. Furthermore, movement of people and vehicles are claimed to be the reason for the spread of the virus between farms in the southern part of Japan during the 2010 FMD outbreak in East Asia (Muroga et al., 2013).

2.10.2. Environmental survival and susceptibility to disinfectants

The virus is hardy and can remain infective for a prolonged period of time in the environment. Data compiled by (Alexandersen et al., 2012) has shown that the virus can remain viable for up to 6 months in beddings and fecal slurry, but the typical survival time is for a couple of weeks in dry feces, up to 4 weeks on hair, up to 39 days in urine, and between 3 to 28 days in soil depending on the temperature. Survival in the environment is temperature dependent, and the virus remained infective for about 50 to 70 days on a wool stored at 4^{o}C, for about 12 days at 18^{o}C to 20^{o}C and 2 to 3 days at 37^{o}C (McColl et al., 1995). On the other hand, FMD virus is rapidly inactivated by high or low pH outside the pH range of 7 to 8 and temperature above 40^{o}C, resulting in complete inactivation of the virus within one hour at 49^{o}C and in a matter of seconds at 55^{o}C (Bachrach et al., 1957). The virus is not as such sensitive to sunlight or UV irradiation, but exposure to direct sunlight kills it within minutes due to the combined effect of drying and temperature (Alexandersen et al., 2012).

Like any other nonenveloped viruses, FMD virus is not sensitive to lipid solvents such as ether and chloroform, but can be easily inactivated by acidic or alkaline disinfectants, such as acetic acid, sodium hydroxide and sodium carbonate. Furthermore, oxidizing disinfectants, including sodium hypochlorite, Virkon S, are very effective against FMD viruses (Neumann, 2014). A recent study by Krug and colleagues has shown that drying alone can decrease FMD virus by about 3 logs, and disinfectants such as sodium hypochlorite (1000ppm), 1 to 2% citric acid and 4% sodium carbonate completely inactivated FMD virus on metallic, plastic, and wooden surfaces (Krug et al., 2012; Krug et al., 2011).

3. Bacterial diseases

3.1. Salmonellosis

Salmonellosis is a disease that affects a wide variety of host species, including man, and is caused by any one of the more than 2000 serotypes of salmonella (Carlson et al., 2012). In swine, the disease is characterized by septicemia and/or enterocolitis. Diarrhea may appear in both types of salmonella infection, but it is more common in the enterocolitis form of salmonellosis, which is characterized by severe inflammation and necrosis of the small and large intestine (ISU, 2015b; Reed et al., 1986). Infection does not always result in disease; most infected pigs remain asymptomatic carriers, shedding the bacteria for some time and thus infecting other pigs (Carlson et al., 2012). Pigs are also important sources of infection to humans via contaminated pork products or as a result of direct contact with infected pigs (Alsop, 2005). Infection can occur in all age groups of pigs, but the disease is primarily observed in weaned piglets or grower/finisher pigs between weaning and 180 days of age (Carlson et al., 2012). The disease is precipitated by stressful conditions like weaning, change of diet and transportation (Isaacson et al., 1999). Clinical cases of salmonellosis is higher in younger pigs, but the rate of bacterial shedding is very high in breeding sows (Wilkins et al., 2010). Special attention should be given to farrowing sows, as they are the primary source of infection to newborn piglets.

3.1.1. Etiology and Transmission

Salmonellosis in swine is caused by small, rod shaped, gram negative, facultative intracellular and facultative anaerobic bacteria that belong to family enterobacteriaceae, genus *salmonella*. Salmonella species are classified into several serotypes based on the lipopolysaccharide somatic (O) and flagella protein (H) antigens. There are more than 2000 serotypes and more than a dozen

of them have been isolated from pigs, but the two major causes of salmonellosis in swine are *Salmonella cholerasuis* and *Salmonella typhimurium*, which are responsible for the septicemic and enterocolic forms of salmonellosis, respectively (Reed et al., 1986). Salmonella species are shed in the feces of infected animals; sub clinically infected animals (carriers) are a special concern as they can shed the bacteria for a long period of time without showing any clinical signs. Carrier pigs can be important sources of infection to healthy pigs and even humans. Feeding probiotics or prebiotics to pigs was reported to decrease shedding of salmonella in the feces of subclinically infected carrier pigs (Letellier et al., 2000).

The major route of salmonella transmission is direct contact by the fecal-oral route through ingestion of contaminated feed and water or indirectly through contact with contaminated fomites. Salmonella species were isolated from pig feed and vehicles used to transport feed to pig farms (Fedorka-Cray et al., 1997), suggesting the possibility that contaminated fomites including vehicles can act as a reservoir host for salmonella infection. Some reports also showed that aerosol transmission, either through direct nose-to-nose contact or through long distant airborne dissemination, was a possible means of transmission (Proux et al., 2001). A role for other animals like cats, rodents, flies and birds in the transmission of salmonella to pigs has also been documented (Barber et al., 2002).

3.1.2. Environmental survival and susceptibility to disinfectants

Salmonella are hardy bacteria that can resist environmental conditions and stay viable for months and even years in suitable organic substances such as manure and wet bedding. Salmonella species were recovered after 180 days from hog manure treated soil and up to 21 weeks in contaminated water (Cote and Quessy, 2005; Holley et al., 2006). The host adapted

salmonella (*S. cholerasuis*) was believed to be sensitive to the environment and unable to survive outside the host; however, a group from the United States Department of Agriculture-Agricultural Research Service were able to recover the host adapted *S. cholerasuis* after 3 months from fecal samples stored under wet condition and after 13 months from a desiccated fecal sample (Gray and Fedorka-Cray, 2001).

On the other hand, salmonella species can be easily killed in a short period of time by moist heat. Moist heat at 71^{o}C or higher can kill salmonella species in less than a minute if not covered with any organic matter (Spickler, 2005a). However, inactivation of salmonella species covered with feces or chicken litter required more than 80 to 100 minutes, depending on the moisture level, for complete inactivation by heating at 70^{o}C (Kim et al., 2012a). Salmonella are also susceptible to many disinfectants and can be easily inactivated with chlorine compounds (1% Sodium Hypochlorite), formaldehyde, iodine-based disinfectants, 2% glutaraldehyde, 70% Ethanol, and phenols (Spickler, 2005a). In addition to this, ozone has been used in the food industry to inactivate a broad range of microbes including *S.typhimurium* (Restaino et al., 1995). The difficulty with effective disinfection of salmonella species occurs when they form biofilms on the surface of biotic and abiotic materials. In this regard, the emerging *S. typhimurium* strain DT104 is of particular concern due to its resistance to major antibiotics and its ability to form biofilms on abiotic surfaces (Ngwai et al., 2006). Biofilm forming salmonella are tough to kill with common disinfectants; however, a combination of ozone and organic acids was reported to have a synergistic effect to inactivate biofilm forming *S. typhimurium* on the surface of abiotic materials (Singla et al., 2014).

3.2. Colibacillosis

Colibacillosis is a disease caused by *E.coli* and characterized mainly by diarrhea and occasionally septicemia and bowel/gut edema. Diarrhea in the newborn (neonatal diarrhea) occurs between 0 to 4 days after birth, while post-weaning diarrhea (PWD) and bowel edema, also known as ED, occurs later in the nursery or 1 to 2 weeks post weaning. PWD and ED are economically important diseases for hog producers due to the high morbidity, mortality and weight loss in piglets, as well as the high cost of drugs and vaccines incurred to control the disease (Fairbrother and Gyles, 2012). Some serotypes of *E.coli,* such as the O157:H7 are also zoonotic and can infect humans through contaminated pork products; however, pig does not seem to be the natural host for this strain of zoonotic *E.coli* (Chapman et al., 1997).

3.2.1. Etiology and Transmission

Escherichia coli are gram negative, facultative anaerobic, flagellated and rod shaped bacteria classified under the family *Enterobacteriaceae*. Species of *E.coli* are classified into different serotypes or strains based on the Somatic (O), Capsular (K), Flagellar (H) and Fimbrial (F) antigens. Currently, at least 175 O, 80 K, 56 H, and 20 F antigens have been identified (Fairbrother and Gyles, 2012). In swine, five antigenically distinct types are reported. F4 (K88) and F18 are common in postweaning pigs, while F4 (K88), F5 (K99), F41 and F6 (987P) are commonly isolated from neonatal piglets (Francis, 1999). These strains of *E.coli* are further classified into pathotypes based on their virulence mechanisms as Enterotoxigenic E.coli (ETEC) or attaching and effacing E.coli (AEEC) also known as EPEC, Enteropathogenic E.coli (Fairbrother and Gyles, 2012). The primary sources of infection are infected animals shedding the bacteria in their feces, urine, nasal and oral secretions. Sows are assumed to be the silent carriers that infect newborn piglets

immediately after birth (Fairbrother and Gyles, 2012) and transmission is either directly through the fecal-oral route by ingestion of contaminated feed and/or nose-to-nose contact or indirectly through by the aerosol route (Cornick and Helgerson, 2004; Cornick and Vukhac, 2008).

3.2.2. Environmental survival and susceptibility to disinfectants

Generally *E.coli* are resistant to adverse environmental conditions and can remain viable and infectious outside the host for prolonged period of time. Reviews that compiled data from several published papers on the environmental survival of *E.coli* showed the ability of the bacteria to resist high temperature fluctuations, acidic pH, and drying/desiccation conditions and survive for more than a year in soils, manure and water (Cote and Quessy, 2005; van Elsas et al., 2011). *E. coli* was also reported to survive for more than 14 months on the surface of inanimate objects or fomites that can then act as a source of infection to pigs through indirect contact (Kramer et al., 2006).

On the other hand, *E.coli* bacteria are susceptible to heating and can be killed within seconds by boiling at 70^{o}C (Oie et al., 1999). The bacteria are also highly susceptible to most of the commonly used disinfectants; however, some strains of *E.coli,* such as, the O157:H7 are able to make biofilms when they are outside the host and become resistant to most oxidizing disinfectants (Vogeleer et al., 2014). Treatment of biofilms with aqueous chlorine dioxide followed by drying at 22^{o}C and 43% relative humidity completely inactivated biofilm forming *E.coli* within 6hrs (Bang et al., 2014), suggesting that washing, disinfection, and drying of livestock transporting vehicles can completely inactivate all kinds of *E.coli* species.

3.3. Swine Brachyspiral Colitis

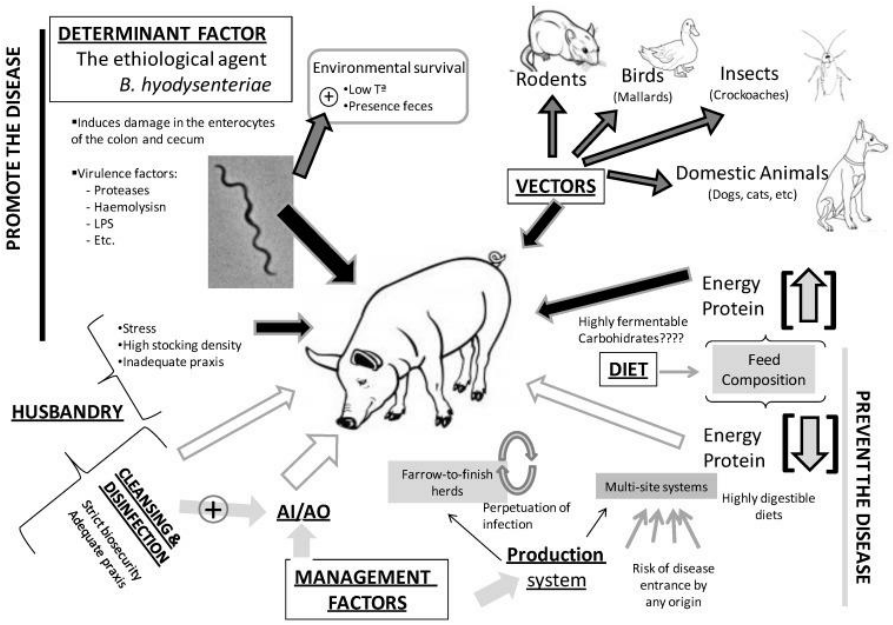
Swine Brachyspiral colitis is a severe bacterial infection of pigs characterized by mucohemorrhagic diarrhea known as swine dysentery (SD) and mild spirochaetal colitis (SC) associated with inflammation of the large intestine (cecum and/or colon) of pigs (Neumann, 2014). Swine dysentery is seldom reported in young pigs, but it is wide spread in the growing/finishing period of the pig cycle and can result in a huge economic losses to the hog industry due to mortality and suboptimal performance of pigs with reduced feed conversion efficiency and retarded growth during diseases outbreaks (Wills, 2000). The second form of Brachyspiral infection, spirochaetal colitis (SC) usually occurs as mild non-hemorrhagic colitis in young pigs between the age of 2 to 3 weeks (Neumann, 2014).

3.3.1. Etiology and Transmission

Swine dysentery is caused by gram negative, motile, spiral shaped, anaerobic bacteria that belong to the genus *Brachyspira* in the *Spirochaetaceae* family (Paster and Dewhirst, 2000). *Brachyspira* inhabit the large intestine of birds and mammals and six species of *Brachyspira* have been isolated from the large intestine of swine (Hampson, 2012). Most *Brachyspir*a species in swine are nonpathogenic commensals; however, *B. hyodysenteriae* and *B. pilosicoli* are pathogenic and induce disease in pigs characterized by severe hemorrhagic dysentery (SD) and mild spirochaetal colitis (SC), respectively (Hampson, 2012). Previously, *B. hyodysenteriae* and *B. pilosicoli* were classified under genus *Treponema* and then genus *Serpulina* until they were finally classified under genus *Brachyspira* (Ochiai et al., 1997).

The natural host for *B. hyodysenteriae* and *B. pilosicoli* is the pig, including feral pigs; however, the bacteria have also been isolated from birds and other mammals like mice, rats and dogs that

act as an important source of infection to pigs (Alvarez-Ordonez et al., 2013). The bacteria are shed in feces of both sick and asymptomatic carrier animals and transmission is through ingestion of feces contaminated feed and water. Transmission can also occur through indirect contact with contaminated fomites like boots, clothing and livestock transport vehicles, as well as by mechanical vectors like rodents, flies and birds (Hampson, 2012; Neumann, 2014). In addition to this, a comprehensive review on Brachyspira, recently published in the *International Journal of Environmental Research and Public Health* (Alvarez-Ordonez et al., 2013) has extensively described the determinants that influence survival of the bacteria in the environment, all the sources and routes of transmission, and factors contributing to the establishment of swine dysentery in pigs. The figure shown below taken from the above review is a summary of the factors involved in the transmission of Brachyspira species.



Factors influencing the establishment and transmission of Swine Dysentery (SD), taken from Alvarez-Ordonez et al., 2013.

3.3.2. Survival in the environment and susceptibility to disinfectants

Brachyspira species are anaerobic, but can stay viable for a few weeks to several months depending on the presence of organic matter, moisture level, and temperature of the environment. Compared to other Brachyspira species, *B. hyodysentery* is sensitive to environmental factors and was reported to survive only for 10 days in pure soil held at 10^{o}C. The survival time in soil was increased to 78 days in the presence of 10% pig feces and was even longer (112 days) in pure wet pig feces. On the other hand, the less pathogenic *B. pilosicoli* was reported to survive for 119 days in pure soil and for more than 210 days in soil in the presence of 10% pig feces (Boye et al., 2001). Brachyspira species, however, are highly susceptible to high environmental temperatures and drying, which can be killed by desiccation and temperatures above room temperature (Chia and Taylor, 1978). The optimal environmental temperature for long term survival of Brachyspira is 0-10^{o}C and survival decreases with decreasing moisture and increasing temperature and pH. Temperature above 37^{o}C and alkaline pH $\geq$8 is lethal to Brachyspira and they can be effectively inactivated by heating at 56^{o}C for few minutes (Alvarez-Ordonez et al., 2013; Chia and Taylor, 1978), suggesting that control of the diseases will be more effective during the hot summer season using alkaline solutions.

Brachyspira species are also susceptible to common disinfectants like phenols and sodium hypochlorite (Hampson, 2012). A study conducted to examine the effect of seven different disinfectants (quateranery ammonium compounds and organic acids) showed that *B. pilosicoli* can be easily inactivated by most disinfectants in the presence or absence of organic matter, even though the inactivation process was reduced or/and slowed down in the presence of organic matter like feces (Corona-Barrera et al., 2004). For example, the efficacy of Virkon S was highly

reduced in the presence of feces and required a more concentrated solution (1:10) than the recommended dose (1:200) to attain 100% inactivation. Comparable results were also reported from another experiment, which used Virkon S and Chirox to inactivate *B. hyodysenteriae* in the presence of 10% fecal slurry from SPF pigs as source of organic matter, (Lobova and Cizek, 2004). The implications of all these results is that good biosecurity measures applied in the farm and on fomites as well as livestock transport vehicles can effectively control *Brachyspira* infection and spread. These results also suggest the importance of cleaning and removal of organic matter by washing before disinfection.

3.4. Swine Erysipelas

Erysipelas is an infectious disease of pigs that can occur in all stages of pork production cycle. It manifests itself clinically either as an acute septicemia associated with endocarditis and sudden death in grow-finisher pigs or as chronic arthritis and lameness in adult pigs (Opriessnig and Wood, 2012). The septicemic form of the disease is also characterized by a typical diamond-shaped skin discoloration around the ear, snout and the abdomen (Neumann, 2014). If uncontrolled, both the septicemic and the chronic form can result in significant economic losses to the pork industry due to mortality, reduction in pig growth, and a decrease in meat quality (Opriessnig and Wood, 2012). Swine Erysipelas is also a zoonotic disease and infection in man is mostly occupationally related, affecting primarily farm and abattoir workers as a result of contact with animals, their products, or their wastes. In humans, Erysipelas can cause mild cutaneous infection known as erysipeloid, or a serious systemic complication with septicemia and endocarditis (Brooke and Riley, 1999).

3.4.1. Etiology and Transmission

The causative agent of Erysipelas in pigs is *Erysipelothrix rhusiopathiae*, which is a gram positive, slightly curved, rod shaped bacteria that belongs to the genus *Erysipelothrix*. The bacteria can grow under a wide temperature range (5^{o}C to 44^{o}C), but the optimal temperature is 30^{o}C to 37^{o}C (Opriessnig and Wood, 2012; Reboli and Farrar, 1989). The domestic pig is the natural host, but the bacteria can also infect a wide range of domestic and wild mammals including chicken, turkeys, ducks and lambs that act as reservoir hosts and hence as sources of infection to the domestic pig (Wang et al., 2010). Not all infected pigs show symptoms of infection, and 30 to 50% of infected pigs remain as carriers harboring the bacteria in the oropharynx and tonsils (Okolo, 1986). However, both sick and carrier animals shed the bacteria in their feces, as well as oral and nasal secretions, representing a significant source of infection to healthy animals and an important source of environmental contamination (Okolo, 1986; Takahashi et al., 1987). Transmission is mainly by the fecal-oral route through ingestion of contaminated feed and water, but direct inoculation through skin wounds can also occur. Indirect transmission through contaminated fomites and carrier vectors including wild animals, birds, rodents, reptiles, and flies is also an important source of infection to domestic pigs (Neumann, 2014).

3.4.2. Environmental survival and susceptibility to disinfectants

Erysepalothrix are stable in the environment and can survive for a prolonged period of time. Therefore, the environment is an important source of Erysepalothrix infection to susceptible animals. A comprehensive review compiled by (Brooke and Riley, 1999) showed that the organism prefers cooler temperatures, alkaline pH (6.7 to 9.2) and the presence of organic matter like feces for long time survival. Erysepalothrix can survive for up to 5 days in drinking water,

about 14 days in sewage, at least 35 days in the soil, and 1 to 5 months in feces. However, the bacteria are sensitive to heat and can be killed in moist heating at 55^{o}C in 15 minutes (Reboli and Farrar, 1989). They are also highly susceptible to most household disinfectants (Fidalgo et al., 2002). As an important consideration for food safety, Erysepalothrix are resistant to salting, curing and many other food preserving techniques, which make it difficult to control the dissemination of the bacteria to humans (Opriessnig and Wood, 2012). Despite the susceptibility of the bacteria to many antimicrobial agents, including common disinfectants, and the availability of an effective vaccine, controlling the diseases and eradication of Erysepalothrix from swine herds is still difficult and complicated primarily. This could be, as discussed above, due to the involvement of a wide variety of animals able to act as reservoir hosts and the ability of the bacteria to survive in the environment for a prolonged period of time.

3.5. Mycoplasmal Pneumonia

Mycoplasmal pneumonia, also known as Enzootic pneumonia, is an infection of the respiratory tract of pigs resulting in chronic bronchopneumonia associated with suppression of immunity in the lung, thereby creating a suitable condition for opportunistic bacterial and viral pathogens to flourish and infect the lung (ISU, 2015a). Even though Enzootic pneumonia can occur as a single entity by itself, it is usually reported as part of the porcine respiratory diseases complex (PRDC) together with swine influenza, PRRS, porcine circovirus infections, and other bacterial infections (ISU, 2015a; Thacker et al., 1999). It is worldwide in distribution, reported from almost all pork producing countries, and is commonly seen in its chronic form characterized by dry cough, high morbidity and low mortality rates. Acute fatal cases, however, can also occur in naïve and immunocompromised animals (Dee, 2014). It usually affects grower-finishers and incurs

significant economic losses to swine farmers due to reduced feed conversion and retarded growth rate of infected animals, and also due to the central role it plays in the pathogenesis of PRDC (Pointon et al., 1985; Thacker et al., 1999). A review compiled by Thacker and colleagues on the economic importance of enzootic pneumonia to pig farmers found a decrease of 17% and 14% for daily weight gain and feed efficiency, respectively. The cost of the disease was estimated to be around $4.08 per pig, and when the cost of control measures is included, the disease may cost the US hog industry close to $500 million per year (Thacker and Minion, 2012).

3.5.1. Etiology and Transmission

The causative agents of enzootic pneumonia, *Mycoplasma pneumonia*, also known as *Mycoplasma hypopneumenia*, are the smallest bacteria classified under the Mollicutes class of bacteria that lack a cell wall. They are very sensitive to environmental factors outside the host. However, the absence of the cell wall structure allows these organisms to be resistant to antibiotics such as penicillin that target the cell wall. The absence of a cell wall also prevents the bacteria from staining using gram's stain (Razin et al., 1998). Despite difficulties in staining, they are believed to be descendants, by mutation, of gram positive bacteria, and thus they are grouped under gram positive bacteria (Pieper et al., 1995). Mycoplasma are the smallest known bacteria that can grow in a cell-free media, but the bacteria need special requirements for successful in vitro cultivation. Hence, Mycoplasma are termed fastidious bacteria (Thacker, 2004).

M. hypopneumonia (MHP) is excreted in nasal secretions of infected or carrier pigs and the main route of transmission is by direct nose-to-nose contact between infected and healthy pigs (Meyns et al., 2004). Even though the role of indirect transmission through contaminated fomites is not

well investigated, there is still a possibility of transmission through this medium as the organism can live for a few days in a cool and wet environment (Goodwin, 1972). In one study, however, where personnel were using routine standard hygienic measures before entering a susceptible herd, enzootic pneumonia was not transmitted from infected to healthy pigs during the 20 weeks observation period when the workers had a frequent contact with both the infected and healthy pigs (Batista et al., 2004). Therefore, even though a possibility for indirect transmission of mycoplasma by contaminated fomites (Fano et al., 2005), including animal transport vehicles (Hege et al., 2002) was previously reported, it is limited to poorly managed farms. Thus, introduction of enzootic pneumonia into a farm or population of pigs is most probably caused by infected or/and carrier pigs (ISU, 2015a). Despite its sensitivity to the external environment, there is convincing evidence in the literature that show airborne dissemination of the bacteria is possible. Airborne transmission up to 150 m was reported by (Cardona et al., 2005), while others reported a spread of the bacteria to distant farms up to 3.2 to 9.2 Km is possible (Dee et al., 2009a; Goodwin, 1985; Otake et al., 2010).

3.5.2. Environmental survival and Susceptibility to disinfectants

Due to lack of the cell wall, Mycoplasma bacteria are highly sensitive to the external environment and cannot survive outside the host body for an extended period of time. However, they can stay alive for some time in a cool and moist condition. For example, (Cardona et al., 2005) reported survival of the bacteria for more than 30 days in water at 2 to 7^{O}C. Similarly, the bacteria was reported to survive up to 7 days if covered in organic matter such as nasal discharges or mucous (Hurnik, 2005). Nonetheless, the bacteria are killed within a few hours under dry and hot weather conditions in the environment as well as by heating to temperatures above 45^{O}C. Typical

mycoplasma inactivation protocols to avoid laboratory contamination are conducted by heating for 30 minutes at 45^{O}C or for 10 minutes at 60^{O}C, or by irradiation at 25 to 35 kGy (personal communication), suggesting that heating and irradiation could be used to inactivate *M. hypopneumonia* from fomites, including transport vehicles. Mycoplasma are also highly sensitive to almost all commonly used disinfectants (ISU, 2015a) making it easy to control the spread of mycoplasma in pigs farms using good biosecurity procedures.

3.6. Leptospirosis

Leptospirosis is a highly contagious disease of both domestic and wild animals. The disease is distributed globally, with a major economic impact on the livestock industries (Ellis, 2015). Leptospirosis is also an important zoonotic disease in humans (Bharti et al., 2003). Swine leptospirosis is wide spread in all pig farming countries, but its economic impact is largely confined to intensive pig farming systems in western countries, including Australia, New Zealand and Brazil (Ellis, 2012). The causative agent of leptospirosis persists in the kidney and genital tract of pigs, resulting in urinary and reproductive tract infection (Ellis et al., 1986b). Reproductive tract infection is associated with huge economic losses in breeding sows due to abortion, still birth, reduced litter size, and reduced number of weaned piglets (Kemenes and Suveges, 1976; Ramos et al., 2006).

3.6.1. Etiology and Transmission

The etiological agents of leptospirosis are small, motile, aerobic, gram negative spirochetes that belong to the genus *Leptospira*. The genus includes several species of pathogenic and saprophytic bacteria, which are further classified into more than 250 serovars. Pigs can be infected by several serovars of Leptospira, but the most prominent one is *L. interogans* serovar *Pomona* (Spickler,

2013). The bacteria persists in the urinary and genital tract of pigs and is excreted in urine and genital discharge of infected or carrier animals (Ellis et al., 1986b). Transmission is either by direct contact with infected pigs, particularly aborted sows, or by indirect contact via contaminated feed and water. Live animal vectors, including rats, mice, skunks, raccoons, foxes, and possums are also an important source of infection (Neumann, 2014). Water contaminated with urine from carrier animals including boars (Ellis et al., 1986a), foxes (Kingscote, 1986a), and skunks (Kingscote, 1986b) is reported to be important source of infection for healthy pigs, and infection is primarily through skin wounds or contact with the mucus membrane.

3.6.2. Environmental survival and susceptibility to disinfectants

Leptospira can survive outside the host for a considerable period of time, making the environment an important reservoir of infection. The length of time Leptospira survives outside the host depends on moisture, temperature, and pH of the environment, and the bacteria prefers warm, moist, and close to neutral pH conditions (Levett, 2001). It is difficult to determine the exact length of time Leptospira survives under different conditions, as the requirement for various serovars could be different, but a review of several published data compiled by (Levett, 2001) has shown that Leptospira species can remain viable for several months in water at room temperature in a pH range of 6.7 to 7.3. Survival was prolonged in fecal slurry kept at cooler temperature under shade. On the other hand, Leptospira were reported to be sensitive to salty water, exposure to direct sun light, and to desiccation (Khairani-Bejo et al., 2004). The bacteria are also sensitive to heating, and were killed in a short period of time by heating at temperatures $\geq$ 45^{o}C (Parker and Walker, 2011). Leptospira are highly sensitive to most commonly used detergents and disinfectants including 1% sodium hydroxide, 70% ethanol, iodine compounds,

quaternary ammonium compounds, hydrogen peroxide, glutaraldehyde, and formaldehyde (Spickler, 2013).

3.7. Actinobacillosis

Actinobacillosis is a highly contagious respiratory infection of pigs caused by several species of bacteria that belongs to the family Pasteurellaceae and genus *Actinobacillus.* There are more than 22 species of bacteria under this genus, but only four species are associated with diseases in animals and two of these, *Actinobacillus pleuropneumoniae* and *Actinobacillus suis*, are important pathogens in pigs (Smith, 2013).

3.7.1. Actinobacillus pleuropneumoniae (APP)

APP is gram negative coccobacillus that affects only pigs and causes a highly contagious and severe pleuropneumonia, also known as contagious porcine pleuropneumonia (CPP). The disease is one of the most economically important respiratory diseases of pigs and is characterized by sudden onset and death in susceptible populations (Neumann, 2014). It is worldwide in distribution and economically important to the hog industry because of high morbidity (retarded growth) and also mortality. Mortality is high when the disease occurs in conjunction with other respiratory infections as a component of the porcine respiratory diseases complex, PRDC (Gottschalk, 2012). It is still a problem in many European countries and is also widely distributed in North America. In one study, 78% and 70% of the herds in Ontario were positive for APP based on PCR or ELISA tests, respectively (MacInnes et al., 2008). It can affect all age groups of pigs, but it is primarily reported from pigs between 6 to 20 weeks of age (Neumann, 2014).

3.7.1.1. Etiology and Transmission

The etiological agent of APP is *Actinobacillus pleuropneumoniae*. There are two biotypes depending on their requirement for nicotinamide adenine dinucleotide (NAD) for in vitro growth and cultivation (Pohl et al., 1983). There are more than 15 serotypes within these two biotypes, which differ in their virulence and epidemiological distribution. The most virulent strains include serotype 2 in Europe, serotype 15 in Australia, and serotypes 1 and 5 in North America (Gottschalk, 2012). However, in Canada, there seems to be a shift in prevalence from the most virulent serotypes 1 and 5 into the less virulent serotypes 3 and 7, which explains the high prevalence of the diseases in swine herds but very low clinical disease reported from those farms (MacInnes et al., 2008).

APP is primarily a disease of swine and resides in the tonsils of asymptomatic carriers and recovered pigs. Healthy carriers and survivor pigs can shed the virus for months in nasal discharges, and transmission is mainly by direct contact through nose-to-nose. Therefore, dissemination between farms or pens within a farm is believed to be mainly by the introduction of infected live animals (Gottschalk, 2012). However, there are also studies that show indirect transmission through contaminated fomites or aerosol transmission through droplets are also possible ways to disseminate APP within or between swine herds (Desrosiers and Moore, 1998; Tobias et al., 2014). Regardless, the direct transmission is 10 times more efficient than the indirect transmission in spreading the bacteria between pigs on a farm (Tobias et al., 2014). Airborne transmission between pens over a short distance, up to 2.5 meters, has also been reported (Jobert et al., 2000). Even though the role of vectors like rodents and birds is limited, long-distance dissemination of the bacteria to other farms via air and livestock transport vehicles

should be considered in any control plan to prevent the spread of APP between farms. Epidemiological investigation, using molecular typing methods, on the route of transmission of the disease during an outbreak in specific pathogen-free herds in Denmark implicated airborne transmission in 5 out of 12 cases. Transmission by trailers was implicated in 6 out of 9 cases investigated (Fussing et al., 1998).

3.7.1.2. Environmental survival and susceptibility to disinfectants

APP are delicate bacteria that are highly fragile in the environment, particularly under hot and dry conditions; therefore, it is unlikely that APP would survive for a prolonged period of time outside the host on inanimate objects. Nevertheless, there are several reports which show APP can survive outside the host for enough time to be a concern for indirect transmission through contaminated fomites and even livestock transport vehicles. In one study, APP was reported to survive for 3 to 4 days in a dried nasal secretion and even for weeks in physiologically buffered saline (PBS) stored at 4^{o}C. Furthermore, APP was reported to survive storage at -20^{o}C or -70^{o}C for about 4 months, suggesting the possibility of long term survival of the bacteria in frozen pig carcasses (Assavacheep and Rycroft, 2013). The survival time of APP decreases with increasing environmental temperatures. APP was reported to survive for less than 8 hours at 37^{o}C and less than 4 hours at 42^{o}C (Morozumi and Hiramune, 1982).

Actinobacillus are also highly sensitive to most disinfectants; however, the efficacy of various disinfectants against APP may vary depending on the presence or absence of organic matter such as nasal discharges. In one study which tested the efficacy of 23 different disinfectants against APP, Chloramine-T, hydrogen peroxide, glutaraldehyde, quaternary ammonium compound, 2.5% glutaraldehyde, 6.8% glyoxal, and 6% formaldehyde were found to be effective in killing APP even

in the presence of organic matter (Gutierrez et al., 1995). In the same study, the efficacy of most disinfectants was reported to decrease in the presence of serum as an organic matter, suggesting the importance of cleaning and washing organic matter from the surface before applying disinfectants for effective inactivation of AAP.

3.7.2. Actinobacillus suis

Actinobacillus suis is an infectious disease of pigs characterized by hemorrhagic septicemia that mainly affects the lungs but is also seen in other organs. The bacteria reside in the upper respiratory tract of pigs, and infection is asymptomatic in most cases. For example, in one study, more than 94% of the herds tested in Ontario, Canada were estimated to be infected with *A. suis* without showing clinical signs (MacInnes et al., 2008). In a naïve herd, the bacteria may affect primarily suckling piglets, but weaned, fattening and adult pigs can also be affected (Yaeger, 1996). In a naïve and high health status herds, the disease may manifest itself in various clinical forms including sudden death and acute septicemia in suckling and recently weaned piglets, respiratory disease characterized by hemorrhagic and necrotizing pleuropneumonia in grow-finish pigs, and septicemia in adult pigs (Yaeger, 1995). Actinobacillosis due to *A. suis* infection may not be economically as important as infection with *A. pleuropneumonia*; however, all the transmission and epidemiological features are more or less the same as what has been discussed for APP.

3.8. Streptococcosis

Streptococcosis refers to a variety of infectious disease that affect both man and animals. Streptococcosis is caused by a group of gram positive cocci that belong to the phylum Firmicutes, order Lactobacillales, family Streptococcaceae, and genus *Streptococcus.* The bacteria reside in

the body of animals and humans as part of the normal microflora, and disease occurs either as a result of decreased immunity in the host, appearance of more virulent strains by mutation, or transfer of the bacteria to an unusual site within the host body (Spickler, 2005b). Some species of streptococcus are zoonotic, in that they use animals as reservoir hosts and cause severe illness in humans. The most important zoonotic streptococcus is *S. suis,* reported to affect around 1600 people in 2012 alone, primarily in south east Asia but also in Europe and North America (Huong et al., 2014). Streptococci are classified into three groups, based on their ability to lyse red blood cells on blood agar, as Alpha hemolytic, Beta hemolytic and non-hemolytic groups. They are further classified into different groups (Lancefield grouping) and serotypes based on serological reaction to cell wall and capsular antigens of the bacteria (Spickler, 2005b). Even though several species of streptococcus are able to cause sporadic disease in pigs, emphasis will be given here to *S. suis* due to its economic significance in the swine industry worldwide (Staats et al., 1997) and its zoonotic significance in humans (Huong et al., 2014). The bacteria primarily affect young pigs immediately after weaning or in the nursery; however, the disease can also affect adult pigs. The disease in young pigs is characterized by acute onset of illness, septicemia, meningitis, polyarthritis, polyserositis, and bronchopneumonia usually followed by sudden death, and sometimes endocarditis in adult pigs. The infection may also occur in other domestic animals, including cattle, sheep, goats, horses and dogs, as well as in humans (Neumann, 2014) .

3.8.1. Etiology and Transmission

Streptococcus suis is a gram positive, non-hemolytic streptococcus with cell wall antigenic determinant related to the Lancefield group D (previously, R, S, RS, and T groups). There are 35 serotypes identified based on serological reaction to the capsular antigens. Several serotypes of

S. suis may infect pigs, but serotype 2 is the most commonly isolated serotype from sick pigs and humans worldwide (Gottschalk et al., 2007). The bacteria normally reside in the upper respiratory tract (tonsils and nasal cavity), gastrointestinal tract, and genital tract of healthy carriers. It is estimated that 60 to 100% of healthy pigs in a herd carry *S. suis* without showing clinical signs of the disease, but only 2 to 15% develop the clinical disease (Spickler, 2005b).

Streptococcus suis are excreted in the nasal discharge of healthy carriers or sick pigs, and transmission is primarily by direct nose-to-nose contact between pigs or aerosol over a short distance. Thus, introduction into a farm is mainly through live animals (Staats et al., 1997). However, the organism has been isolated from manure-coated farm and veterinary equipment, suggesting the possibility of indirect transmission through contaminated fomites (Dee and Corey, 1993). The bacteria was also isolated from house flies (Enright et al., 1987) implicating the role of mechanical vectors like flies, birds, and rodents in the transmission of *S. suis.* Furthermore, the organism is commonly isolated from the reproductive tract of aborting sows, and piglets can be infected in the birth canal during delivery (Robertson et al., 1991). The role of transport vehicles in the dissemination of *S. suis* has not been fully investigated, but, as the organism can survive on fomites for a considerable period of time, it is possible that trailers can spread the bacteria in the absence of good biosecurity measures. In agreement with this, manure containing *S. suis* spread on truck tires was isolated from the tire treads after the truck was driven for 4.82 km at an average speed of 64.3 km/h, but not after an additional 12.87 km at a speed of 96.5 to 120.6 km/h (Dee and Corey, 1993).

3.8.2. Environmental resistance and susceptibility to disinfectants

Streptococcus suis is relatively resistant to the external environment, and it can survive for a considerable period of time outside the host, particularly in moist and cool conditions. In an in vitro study conducted by (Clifton-Hadley and Enright, 1984), the organism was reported to survive for 1 to 2 weeks in water at 4^{o}C; it also survived for 104 days in feces stored at 0^{o}C, but only for 25 days when stored at 9^{o}C. In dust stored under similar conditions, *S. suis* remained viable for about 54 days at 0^{o}C and 10 days at 9^{o}C. However, increasing the temperature to about 25^{o}C (room temperature) decreased the survival time of the bacteria to about 8 days in feces and to less than 24 hours in dust. Furthermore, heating to 60^{o}C inactivated *S. suis* in 10 minutes. *S. suis* are also sensitive to detergents and disinfectants, and can be inactivated in less than a minute with most of the commonly used disinfectants, as well as ordinary liquid hand soaps (Clifton-Hadley and Enright, 1984). In one study, which tested the efficacy of seven commonly used disinfectants including 70% ethanol, Chlorhexadine, formaldehyde, 3% Iodine, One-Stroke Environ (Pheno), Roccal-D (quaternary ammonium compound), and 5% sodium hypochlorite, no growth of bacteria was reported in any of the disinfectants except for 70% ethanol (Dee and Corey, 1993). The efficacy of disinfectants can be reduced by the presence of organic matter such as feces (Dvorak, 2008b); therefore, careful cleaning and washing of equipment before disinfection is very important for complete inactivation of the bacteria and to limit its spread among pigs.

3.9. Glässer's Disease

Glässer's disease is an infectious disease of pigs characterized by fibrinous polyserositis, polyarthritis, and meningitis with bacterial septicemia (Amano et al., 1994). It may also have a

pulmonary form when it occurs as a co-infection with other pulmonary diseases like PRRS and swine influenza (Aragon et al., 2012). The disease usually affects young pigs between the ages of 4 to 8 weeks, although sporadic cases may occur in adult pigs. The disease is manifested clinically with sudden onset and death during stressful conditions like weaning, mixing of piglets, transportation, or experiencing a change in diet (Neumann, 2014). Glässer's disease is global in distribution, reported from all swine-raising countries. Recently Glässer's disease is becoming significant health problems in the nursery piglets of modern and high health standard pig production systems (Aragon et al., 2012).

3.9.1. Etiology and Transmission

The etiological agent of Glässer's disease is *Haemophilus parasuis*, which belongs to the family Pasteurellaceae and genus Haemophilus bacteria. The bacteria are small gram negative highly fastidious rods requiring the NAD (V factor), but not X (hemin factor) for their in vitro cultivation (Biberstein and White, 1969). More than 15 serovars have been identified so far (Kielstein and Rapp-Gabrielson, 1992; Rapp-Gabrielson and Gabrielson, 1992), and pigs may harbor several of them depending on the geographical location. For instance, Serovars 4, 5, 13 and 14 are more prevalent in North America (Neumann, 2014). *Haemophilus parasuis* is a commensal organism of the upper respiratory tract of healthy pigs causing diseases in young pigs only under certain circumstances. It is among the first colonizers of the respiratory tract in piglets and was isolated from piglets immediately after birth (Oliveira and Pijoan, 2004). The organism has never been isolated from the reproductive tract of sows, thus infection of piglets is most probably by direct contact with the sows immediately after birth *(*Aragon et al., 2012). In agreement with this, pigs born by "snatch farrowing" where piglets were taken away directly from the birth canal to avoid

contact with the mother, were free of *H. parasuis* and were used as animal models to study pathogenesis of *H. parasuis* infection in pigs (Oliveira et al., 2003). Under normal conditions, piglets are protected by maternal immunity and are usually asymptomatic carriers (Solano-Aguilar et al., 1999). However, a disease flare-up may occur as a result of decreased immunity or increased stress due to several factors such as weaning, mixing, overcrowding, poor ventilation, or concurrent infection by other pathogens. So far, the primary route of transmission is believed to be through direct contact between pigs, usually from dams to newly born piglets and therefore, introduction into a farm is primarily through live animals (Neumann, 2014).

3.9.2. Environmental resistance and susceptibility to disinfectants

There is lack of information in the literature regarding the environmental survival of *Haemophilus parasuis*; however, it is generally considered to be a liable organism that cannot last long outside the host body. In one study that compared the resistance of different bacteria to heat treatment, *H. parasuis* was reported to be highly sensitive and was completely inactivated within 1 hour at 42^{o}C, within 2 hours at 37^{o}C, and within 8 hours at 25^{o}C, but survived for more than 8 hours at 5^{o}C (Morozumi and Hiramune, 1982). *Haemophilus parasuis* are also highly sensitive to commonly used detergents and disinfectants. A recent study tested the efficacy of several disinfectants and formulations against *H. parasuis* when the bacteria was either prepared as a suspension or spread on a carrier surface. The results showed that the bacteria could be inactivated by all of the disinfectants when tested in suspension, and most of the disinfectants were able to inactivate the organism from the carrier surfaces. However, the efficacy of most of the disinfectants was reduced when they were tested in the presence of serum as a source of organic matter, suggesting the importance of washing before disinfection for effective

inactivation of *H.parasuis* (Rodriguez Ferri et al., 2010). The most important conclusion made from this study include that Chloramine-T was a highly effective disinfectant against *H.parasuis* both in the suspension and carrier tests, regardless of the presence of serum/organic matter, while the efficacy of most of the quaternary ammonium compounds (QACs) was low when tested on a carrier surface, particularly in the presence of organic matter. QACs are the most commonly used antiseptics to disinfect contaminated surfaces of materials, but components of the surface material, like metal or wood, may react with the chemical ingredients of QACs and decrease their efficacy against the germs on the surface (Meggison and Mueller, 1956).

4. Parasitic diseases of swine

Pigs can be affected by both external and internal parasites. However, with the introduction of modern and high standard swine rearing techniques in Canada, the economic impact of parasitic diseases to the pork industry is decreasing dramatically. Here we will only discuss a few examples from both the external and internal parasite groups that may have relatively higher economic significance, on farms under poor hygienic conditions.

4.1. Mange

Mange, also known as scabies, is the most important external parasitic disease of pigs. It affects the skin in both breeding and farrow to finish pigs. The high hygienic standards of the modern pig production system in North America have greatly reduced the economic impact of scabies; however, in infected herds the prevalence may reach as high as 95%, resulting in significant economic losses due to decreased feed efficiency, retarded growth rate, and decreased fertility in breeding sows (Greve and Davies, 2012). In one study, a 9.2 to 12.5% decrease in mean growth rates and a similar decrease in feed conversion efficiencies were reported as a result of experimental infection of pigs with scabies (Cargill and Dobson, 1979). The disease is characterized by intense pruritus that results in damage to the skin due to severe scratching of the affected areas. Intense scratching during severe pruritus may also result in damage of facilities such as pens and farrowing crates. Scabies results in huge economic losses due to decreased fertility in breeding sows, decreased feed conversion efficiency, and retarded growth in young pigs (Davies, 1995). In addition to its economic importance, scabies is also an important animal welfare issue due to the intense pruritus in infected pigs.

4.1.1. Etiology and Transmission

Mange in pigs is caused by one of the two species of mites known as *Sarcoptes scabiei* and *Demodex phylloides*; however, sarcoptic mange (scabies) caused by *Sarcoptes scabiei var suis* is by far the most common and economically important infection (Neumann, 2014). The mites are an obligate parasite of the skin, which burrow through the skin layers to finish their life cycle (egg-larva-nymph and adult) within the epidermis. The parasites are highly susceptible to the external environment and do not survive for long outside the host. Introduction of the parasite to a farm is almost always through asymptomatic carrier pigs. Once the disease is introduced into a farm by carrier animals, transmission occurs between pigs by direct body contact, primarily between sows and newborn piglets (Greve and Davies, 2012). On the other hand, the parasite and eggs may also survive in the outside environment for a short period of time, and thus the disease may spread by contact with recently contaminated materials. In particular, materials contaminated by scratching are important sources of infection. For instance, in a study conducted in New Brunswick, Canada, healthy pigs were infected within 24 hours exposure after they were moved into pens previously inhabited by scabies-infected pigs. The healthy pigs were moved into the contaminated pens 6 days after the infected pigs were moved out, suggesting that *Sarcoptic mange* is capable of surviving at least for six days outside the host on contaminated fomites (Smith, 1986).

4.1.2. Environmental survival and susceptibility to disinfectants

Sarcoptes scabiei are very fragile in the environment and can be killed within a few minutes when exposed to direct sunlight. The mites may survive in the environment for 1 to 2 weeks in a cool and humid condition under the shade, but laboratory experiments indicate that the mites could

not survive for more than 24 hours at a temperature between 24 and 30°C. Survival at temperatures >30°C was less than an hour (Losson and Mignon, 2011). As discussed above, the mites were able to infect pigs that moved into an infected pen 6 days after removal of the infected pigs, implicating the survival of the parasite in the environment for about a week. However, another study conducted by Cargill and Dobson in Australia and cited by (Greve and Davies, 2012) reported that infection could not occur in pigs transferred into a room where the bedding had been contaminated with the parasites three days before. These contrasting findings may be results of the different environmental conditions that exist in Australia and Canada.

Despite for a short period of time, the ability of the mites to survive outside the host suggests that appropriate biosecurity measures are required to avoid introduction of the parasite into disease-free herds by fomites, including transport vehicles. The parasite are highly sensitive to a wide variety of chemicals (acaricides) used for the treatment of ectoparasites. Although the majority of the acaricides are to be used as an injection to treat infected animals, there are also acaricides which can be used as a spray that may help to disinfect contaminated fomites, including transport vehicles. However, acaricides are highly toxic and must be used with caution if they are going to be used as a spray to disinfect contaminated fomites and vehicles, to avoid adverse environmental impacts and a toxic effect on personnel.

4.2. Ascariasis

Ascariasis is the most prevalent and most economically important internal parasite of swine. It affects all age groups of pigs, but it is more severe in young, growing pigs. The parasite induces enteritis, which may or may not be associated with diarrhea, but the most significant damage is done by the migratory larvae that cause severe pneumonia and hepatitis (Neumann, 2014).

Ascariasis is widespread in all pig-raising countries, resulting in a significant economic loss to the pork industry due to reductions in average daily gain and feed efficiency. Furthermore, Ascariasis infestation results in economic losses due to liver condemnation at slaughter (Stewart and Hale, 1988).

4.2.1. Etiology and transmission

Ascariasis in pigs is caused by a nematode parasite called *Ascaris suum*, also known as large round worm of pigs. The parasite can grow up to 40 cm in length and 6 mm in thickness. Adult female parasites live in the intestines of the pig and can lay about 1.4 to 2 million eggs per day (Kelley and Smith, 1956; Olsen et al., 1958). The eggs are shed to the external environment with the feces, and contaminate feed and water. The eggs are very hardy, and can survive for years in the environment. Transmission is through ingestion of contaminated feed and water. Ingested eggs will hatch into larvae in the intestine of pigs. The larvae penetrate the intestinal wall and start migrating through the liver into the lungs. They will finally be coughed and swallowed, to return back in to the intestine and grow into adults. Adults stay in the intestine for months and lay eggs until they are finally expelled from the host body at around 40 to 45 weeks post-infection; however the highest egg production observed between week 10 to 13 post infection starts to dramatically decrease after 16 weeks of infection (Olsen et al., 1958).

4.2.2. Environmental resistance and susceptibility to disinfectants

The eggs of *Ascaris suum* are covered with a thick and resistant shell that helps them to withstand the effect of desiccation and toxic chemicals in the environment. It is generally assumed that, under suitable conditions, the eggs can survive for 10 to 15 years in the environment (Roepstorff and Murrell, 1997). However, experimental results coming from different studies on the

resistance of *A. suum* eggs to environmental factors are conflicting. On one study, 90% of the *A. suum* eggs inoculated into experimental test tubes containing pig slurry and kept in a wet and shade area survived for about 8 weeks. In contrast, 90% of the eggs died within 2 weeks when eggs were put in a sunny area with the temperature close to 25^{o}C (Gaasenbeek and Borgsteede, 1998). Similarly, an extensive review by Nansen and Roepstorff (1999) has shown that, despite the survival of the eggs of a closely related *Ascaris lumbricoides* for more than 6 years in the outdoor environment, there was high mortality of eggs of *A. suum* outside the host due to desiccation, which resulted in lack of transmission of the disease from egg-excreting dams to their piglets in an intensive Danish pig farm. According to this review, *A.suum* eggs can survive for a prolonged time at a temperature below 15^{o}C, but egg development and embryonation can only occur when temperature exceeds 15°C. As a result, infection rate in the temperate regions of the northern hemisphere is restricted to the summer period when the higher temperature at the beginning of June allows massive egg development and embryonation (Nansen and Roepstorff, 1999). A more recent study on the effect of environmental temperature on the development and embryonation of *A. suum* eggs reported that the eggs were able to survive for a long period of time at lower temperature but did not develop and germinate for a month at 5^{o}C. However, when the eggs were kept at higher temperature (25-35^{o}C) they were able to develop to the 8-cell infective stage within 5 to 6 days (Kim et al., 2012b). This findings prompted the authors to suggest that the absence of infection in the Danish study may not be due to the high mortality of the eggs reported in that study, but may be because the eggs were not able to grow into the 8-cell infective embryo due to an unfavorable microenvironment in the farm.

Ascaris suum eggs are highly resistant to common disinfectants, which makes it difficult to control and eradicate the parasitic disease. A study that tested the efficacy of 11 chemical disinfectants containing chlorine, phenol, cresol, sodium or potassium hydroxide, quaternary ammonium compounds, glutaraldehydes or paraformaldehyde concluded that none of the disinfectants were effective against the eggs of A. suum (vd Burg and Borgsteede, 1987). Similarly, a more recent study has shown the ineffectiveness of quaternary ammonium compounds and providone-iodine for the inactivation of *A.suum* eggs (Labare et al., 2013). Overall, chemical disinfections are ineffective in the fight against Ascariasis; however, as discussed above, the eggs are sensitive to direct sunlight. Therefore, other alternatives of inactivation such as UV irradiation should be the focus in developing effective biosecurity protocols against *A. suum* infections (Brownell and Nelson, 2006). Furthermore, *A. suum* eggs are highly susceptible to heating and drying; therefore, washing of contaminated equipment, including livestock trailers, with pressurized hot water followed by appropriate drying of the trailers can effectively eliminate the eggs from the surface of the vehicles or other equipment.

5. Disinfectants and Disinfection

Infectious diseases are of great economic importance to the swine industry, primarily because of losses in production and death of animals, but also because of the cost of treatment incurred to treat sick animals during disease outbreaks. Pathogens can be introduced into pig farms through different ways, and prevention of disease introduction into a farm by using appropriate biosecurity measures is the most effective and cheapest way of controlling disease outbreaks and reducing the costs associated with disease control measures. By compiling data from studies conducted between 1988 and 1999 in the UK, Gadd calculated the economic benefit from applying a good biosecurity protocol in pig farms to be between £2.10 and £8.80 per pig, cited by (Thomson et al., 2007). Therefore, properly applied disinfection protocols are becoming an integral part of a farm biosecurity regimen in order to prevent and control infectious diseases and maintain or increase profitability.

The objective of this chapter is, therefore, to provide information on the commonly used disinfectants in the livestock industry, to give a summarized overview of the mode of action of some chemical disinfectants, to list some factors to consider for effective disinfection, and to outline some essential steps for a successful disinfection procedure. But first we will start by defining some commonly used words as adapted and modified from the disinfection 101 document prepared by Center for Food Security and Public Health, Iowa State University (Dvorak, 2008a).

5.1. Definitions

Biocide or germicide is the term used to refer to chemical agents that kill (cidal) or inhibit the growth (static) of micro-organisms. It is a general term that includes antibiotics, antiseptics and disinfectants.

Disinfectant is a term used usually for a chemical agent that destroys or irreversibly inactivates bacteria and some viruses. Disinfectants are directly applied on inanimate surfaces to inactivate or destroy microorganisms. On the other hand, **Antiseptics** are chemicals applied on the surface of living organisms or tissues to destroy or inhibit growth of microorganism. One important point, however, is that most disinfectants and antiseptics are effective in killing vegetative bacteria but may not be as effective in killing bacterial spores.

Sterilization is a process used to destroy or eliminate all forms of life, especially microorganisms, including spores, by using either a chemical agent (disinfection) or physical means such as heat (moist or dry), ultraviolet light, radiations, and microwaves. Heating is one of the oldest methods of sterilization and uses either a dry heat at 160 to 170^{o}c for 2 to 4 hours or moist heat at 121^{o}C for 15 minutes. Moist heat is the most effective method of destroying microorganisms, including spore forming bacteria on a given surface. Chemical disinfection is typically used on surfaces if moist heat cannot be used.

The use of UV light, including direct sunlight, is also a physical method of disinfection for some sensitive viruses, bacteria, fungi and mycoplasma; however, the penetrating power of UV light on surfaces is very low, according to Shama, 1999 cited by (Keyser et al., 2008). Therefore, sterilization using UV or direct sunlight could be effective only on airborne or waterborne

organisms. In addition to this, some viruses such as adenoviruses were not able to be fully inactivated by the dose of UV used to inactivate most pathogens (Nwachuku et al., 2005). Their full inactivation required an increase in the dose of UV to be used, and this may have its own side effects on the quality of the material being disinfected as well as the health and safety of the personnel working with it. Finally, although not commonly practiced, other forms of physical disinfection including x-ray, gamma radiation, microwaves, and ozone could also be used as a possible means of physical sterilization.

The use of ozone (O_3) as a disinfectant, particularly in the food industry and water treatment plants, is widespread. Ozone is reported to inactivate a variety of pathogens including bacteria, viruses, fungi and protozoa through chemical interaction, primarily with the unsaturated lipid layers of bacterial cell walls and viral envelopes but also with protein, carbohydrate, and nucleic acids of all forms of microorganisms (Lillard, 2004). As discussed above for UV disinfection, some microorganisms such as Cryptosporidium parvum oocysts are not susceptible to the standard dose of ozone used to inactivate most pathogens. A combination of ozone and UV may help to increase the efficacy of physical disinfection on pathogens while decreasing the amount of UV and ozone used during the disinfection process (Meunier et al., 2006). A combination of ozone and chlorine was also reported to increase the disinfection efficacy against microsporidia spores in water (John et al., 2005).

5.2. Factors to consider during disinfection

The main objective of any disinfection protocol is to create a sterile surface free of any living microorganism and spores. The efficiency of any disinfection or sterilization protocol will depend on a number of factors.

5.2.1. Nature of the surface/material

The nature of the surface to be disinfected plays an important role. In general, rough surfaces are more difficult to clean and disinfect than smooth surfaces. It is easy to clean and disinfect smooth concrete floors and metal surfaces; however, some disinfectants such as chlorine, peracetic acid, and iodophors are corrosive to metallic surfaces (Gamage, 2003). Raw concrete and wooden surfaces may have porous, uneven, pitted, and cracked surfaces that make it difficult to effectively remove debris, and these can be important hiding places for microorganisms. Wooden surfaces can absorb the disinfectant and dilute its concentration (Dvorak, 2008a). For example, the concentration of citric acid required to completely inactivate FMD virus on a wooden surface was twice the concentration used on metallic surfaces, 2% and 1%, respectively (Krug et al., 2012; Krug et al., 2011). Therefore, it is important to use disinfectants compatible with the materials of the surface to be disinfected. Finally, it is important to note that there is no known safe and environmentally friendly disinfection protocol to decontaminate earth (dirt, sand or clay) surfaces (CFSPH, 2014).

5.2.2. Environmental factors

Factors including temperature, pH, relative humidity, water hardness, and contact time can determine the efficacy of a disinfectant and affect the disinfection process. In general, most disinfectants function better at a temperature higher than 20^{o}C, and efficacy may decrease under cold environmental conditions (Quinn and Markey, 2001). In one study, disinfectants which were effective at 20^{o}C lost their effectiveness when applied at 4^{o}C or/and 10^{o}C (Thomson et al., 2007). Similarly, out of seven different disinfectants tested to decontaminate trailer models against PRRSV, only Synergize (glutaraldehyde plus quaternary ammonium chloride) inactivated the virus

completely within 60 minutes at 4^{o}C; however, the same decontamination protocol did not work when applied at -20^{o}C (Dee et al., 2005b). Conversely, a temperature higher than 40^{o}C may decrease the efficacy of some disinfectants like chlorine and iodine (Quinn and Markey, 2001). Alkaline pH and high humidity are also optimal for most disinfectants. Another important environmental factor to consider is the nature of the water. Hard water contains positive ions like Ca^{2+} and Mg^{2+}, which can interact with the chemical composition of disinfectants and inhibit their effect on microorganisms. Quaternary ammonium compounds are known to be inactivated by hard water, and the efficacy of aldehydes can be reduced by hard water (Dvorak, 2008a). Therefore, using soft water or adding chelating agents like EDTA to the solution will be important to decrease the negative effect of hard water during disinfection. Finally, following the manufacturer's instructions on the optimal contact time for each disinfectant is very important for an effective disinfection protocol. Some disinfectants may kill pathogens instantly, but most disinfectants need 20 to 30 minutes of contact time (Ewart, 2001).

5.2.3. Organic matter

Dirt or/and organic matter on the surface of equipment or floor can interfere with the disinfection process. Organic matter in the form of blood, pus, body fluids/discharges, and feces contain proteins that can bind to the active ingredient of some disinfectants, such as chlorine and iodine, and rapidly reduce their efficacy. Organic matter can also completely inactivate disinfectants like quaternary ammonium compounds (Dvorak, 2008a). Furthermore, organic matter provides physical protection to the microorganism and inhibit contact between pathogens and disinfectants thereby slowing down their action (CDC, 2008). It is almost

impossible to disinfect dirt or surfaces covered with organic matter. Therefore, the first step in any disinfection process is proper cleaning of the surface to be decontaminated.

Cleaning includes dry and wet cleaning; dry cleaning refers to the physical removal of dirt such as manure, bedding, and feed by a combination of sweeping, scraping and vacuuming, to be followed by wet cleaning using water and detergents or soaps. Detergents are chemicals or organic substances used to disperse and remove organic matter from a surface before a disinfectant is applied. Detergents help to reduce surface tension and allow water to penetrate the organic matter for easy and fast removal of the dirt from the surface. The use of soaps or detergents can decrease washing time by about 12% (Hurnik, 2005).

Detergents can be **cationic** (positively charged chemicals), **anionic** (negatively charged compounds like soaps), and **nonionic** (uncharged). The most commonly used detergents are the anionic and nonionic compounds. However, the anionic compounds are highly foamy and not suitable for cleaning. Nonionic detergents are considered good detergents as they have more emulsifying power, less foaming properties, and are compatible to use with hard water (Ewart, 2001). Most commercially available detergents are a mixture of anionic and nonionic compounds. It is always important to check the compatibility of the detergent with the disinfectants to be used; some disinfectants like chlorine and quaternary ammonium compounds may not work and even can be inactivated when mixed with detergents (Dvorak, 2008a).

Steam (hot water around 95^{o}C) and high pressure ($\geq$ 1000 psi) washers are very useful for effective cleaning of surfaces such as room floors and trailers (Ewart, 2001), which can reduce the washing time by about 22% (Hurnik, 2005). Special attention has to be given to corners and

grooves during cleaning and washing of surfaces, particularly trailers. Finally, the surface has to be rinsed and properly dried before applying the disinfectant. Drying can be done naturally, but usually takes at least 24 hours. Using hot air fans may speed up the process of drying and decrease the down time to 2 to 8 hours. Proper cleaning and washing procedures have the ability to decrease the number of pathogens on the surface by up to 90% (Fotheringham, 1995) making the next disinfection step very effective.

5.2.4. Type of pathogens

Microorganisms vary in their susceptibility/resistance to various disinfectant chemicals, and the choice of the disinfectant always depends on the nature of the microorganism targeted for decontamination. Information on the susceptibility of each pathogen to different disinfectants and methods of disinfection is presented in detail under each disease. In general, obligate intracellular bacteria such as Mycoplasma are the most susceptible organisms to disinfection. The next groups of susceptible pathogens include Gram-positive and Gram-negative bacteria, lipophilic enveloped viruses, and fungal spores. These organisms are “tougher” than the intracellular bacteria, but are not considered to be resistant to disinfection. The resistant microorganisms include the hydrophobic non-enveloped viruses and mycobacteria. The most challenging organisms to inactivate with ordinary disinfection protocols are bacterial spores, protozoal oocytes and prions (Quinn and Markey, 2001).

There is no known single disinfection protocol for complete sterilization of prions, and therefore, combinations of different disinfection methods are recommended. An extended steam sterilization in the presence of chlorine-releasing compounds like sodium hypochlorite or autoclaving in the presence of high concentration of sodium hydroxide is reported to be effective

against prions (Taylor, 2000). Similarly, McDonnell and colleagues confirmed cleaning with alkaline formulation disinfectants in combination with steam sterilization to be an effective method of prion decontamination (McDonnell et al., 2013).

Another important challenge during disinfection is the ability of some microorganisms to form biofilms on the surface of materials like wood, glass, plastic or stainless steel. Biofilms are defined as structured microbial communities that are tightly attached to each other and to the surface by a self-produced thick polymer matrix known as extracellular polymeric substances (EPS). Microorganisms within the biofilms are believed to be 1000 times more resistant to disinfectants than their planktonic counterparts (Bridier et al., 2011). Most disinfectants are not effective against bacteria in biofilms; however, a combination of disinfection procedures such as acid pre-treatment followed by application of a concentrated bleach solution (Marion-Ferey et al., 2003), or sequential treatment with chlorine dioxide followed by rinsing with water and drying (Bang et al., 2014) are reported to be effective methods to inactivate bacteria within biofilms.

5.2.5. Nature of the disinfectant

A good disinfectant is the one which is effective against all pathogens (broad spectrum), cheap, non-toxic, non-corrosive, and environmentally friendly. However, no disinfectant is universally effective against all microorganisms and it is important to choose the right type and concentration of disinfectants to be used for the specific purpose. In general, the higher the concentration of the disinfectant, the greater is the efficacy and the shorter the contact time. The exception to this is the alcohols and iodophors that have an optimal concentration for greatest efficacy (Favero and Bond, 2001). Increasing concentration for some disinfectants may help to inactivate pathogens in the presence of organic matter when effective cleaning is not

possible; however, the health and safety of personnel and the environmental implications need to be considered when there is a need to use disinfectants at a concentration higher than the recommended concentration.

Disinfectants can be classified into different groups based on the chemical nature of their composition. Each group of disinfectants has its own unique characteristics such as mode of action, advantages, disadvantages, effect on various organisms, environmental factors that can affect its efficacy, and hazards/toxicity effects. In general, disinfectants target the cell wall and plasma membrane of bacteria, as well as the envelope and capsid of viruses. Disinfectants that specifically destroy the viral capsid or envelope without acting on the nucleic acid may not be the disinfectants of choice to inactivate viruses, as some free viral genomes can still be infectious (Nuanualsuwan and Cliver, 2003). It is beyond the scope of this review to discuss the detailed characteristics and mode of action of each group of disinfectants; however, a table which summarizes the most important characteristics of each group of disinfectants, adapted from a previously prepared document (Dvorak, 2008a), is included here in the appendix section. In addition to this, results from an extensive in vitro study conducted in the UK on the efficacy of selected disinfectants against bacterial pathogens of pigs (Thomson et al., 2007) are presented in a series of tables presented in appendix 2 and a brief summary of the results will be discussed in this section.

Actinobacillus pleuropneumoniae was sensitive to all disinfectants under low and high organic matter with the exception of quaternary ammonium compound, which was not effective at the highest concentration used (1:100), and an iodine compound that was effective only at a higher

concentration than the recommended dose. *Haemophilus parasuis*, the causative agent of Gläser's disease, showed good susceptibility to all but peracetic acid plus hydrogen peroxide and the iodine compound, which were not effective under high organic matter conditions. Furthermore, under low organic matter conditions, iodine was effective only when it was used at a concentration higher than the recommended dose. Under low organic matter, *Streptococcus suis* was susceptible to almost all disinfectants except for iodine, which showed some limitations at low temperature or shorter contact time. On the other hand, under high organic matter, *S. suis* was not susceptible to Iodine, peracetic acid plus hydrogen peroxide, and peroxygen. All strains of the *Salmonella typhimurium* isolates were not susceptible to any of the disinfectants when tested under high organic matter conditions; however, two preparations containing hydrogen peroxide (peracetic acid plus H_2O_2 and quaternary ammonium plus H_2O_2) showed varying efficacy under low organic matter conditions. *Brachyspira hyodysenteriae,* the causative agent of swine dysentery, was generally susceptible to all compounds except iodine and peroxygen, which required concentrations higher than the regular dose to inactivate the bacteria under high organic matter or low temperature (4^oC) conditions. Finally, the *E.coli* strain isolated from neonatal pigs during a colibacillosis outbreak was resistant to all disinfectants used except for the mixture preparation of quaternary ammonium and hydrogen peroxide that show efficacy when tested under low organic matter conditions. Taken together, the results of this extensive study have shown that low temperature, short contact time, and high organic matter decreased the efficacy of most of the disinfectants tested.

6. Animal Transport Biosecurity

The multi-site production technology in a modern livestock farming system necessitates animal transportation between sites. The Canadian Food inspection Agency (CFIA) estimated that more than 25 million pigs were transported in Canada in 2013, either to slaughter houses or for export purposes. In addition to the animal welfare issues of transportation, long-distance transportation of animals is a stressful process that may result in the emergence of several previously asymptomatic gastrointestinal and respiratory diseases. For instance, the prevalence of salmonella in feces increased from 18% before transport to 46% after 30 to 40 minutes of transporting the animals (Barham et al., 2002). Furthermore, several animal pathogens that shed from the animal body as a result of transportation stress are able to survive outside the host for a considerable period of time. The table below (Hurnik, 2005) shows examples of pig pathogens and their survival time in the environment, and thus in livestock transport vehicles. This information suggests the high risk of disease dissemination during animal transportation.

Survival times of common pig pathogens outside the Host

Pathogenic agent	Survival in environment
Mycoplasma Hyponeumoniae	Up to 7 days in organic matter
Actinobacillus Pleuropneumoniae	few days in organic matter
Pasteurella Multocida	8 days in water ; 6 days in liquid manure
Hemophilus parasuis	short
Streptococcus suis	25 days @ 9 ^{o}C ; 100 days @ 0 ^{o}C
Salmonella sp	years in manure, 115 days in water 120 days in soil
Serpulina Hyodysenteriae	61 days @ 5 ^{o}C ; 7 days @ 25 ^{o}C
E coli	11 weeks in manure
PRRSv	3 weeks in organic matter ; 11 days in water
Pseudorabies virus	18 days on steel, manure 2 days, urine 14 days, well water 7 days,
TGE/PRCV	low summer, stable when frozen
Influenza virus	24 - 48 hours
Ascaris suum	years

Source: Daniel Hurnik – London Swine conference, 2005

Vehicles used to transport livestock, their products or by-products have long been considered an important risk factor for disease spread among farms, either through contact of naïve pigs with the interiors of contaminated transport vehicles or through pathogens carried by the tire treads of the vehicle (Poumian, 1995). Some of the economically important swine diseases reported to be disseminated by transport vehicles include African swine fever (Mur et al., 2012), classical swine fever (Elbers et al., 2001), foot and mouth disease (Muroga et al., 2013), porcine reproductive and respiratory syndrome (Dee et al., 2004b), porcine epidemic diarrhea (Lowe et al., 2014), post-weaning multisystem wasting syndrome (Kristensen et al., 2009), *Actinobacillus plueropneumoniae* (Fussing et al., 1998), Streptococcus suis (Dee and Corey, 1993), swine salmonellosis (Fedorka-Cray et al., 1997), and many other enteric and respiratory diseases.

To prevent the spread of swine diseases between farms or provinces, and to limit the importation of exotic diseases, the Canadian Swine Health Board has set a number of transport biosecurity measures applicable at different levels of the industry. The standard protocol for washing and disinfection of vehicles is discussed briefly in section 2.5.4 of this document and presented in detail in the protocol "Live Hog Transportation Vehicle Wash/Disinfect/Dry" prepared by the Canadian Swine Health Board (CSHB, 2011). Despite the important role the current system is playing in reducing disease spread between farms, a study conducted in association with the current threat of Porcine Epidemic Diarrhea virus (PEDv) has identified a number of gaps in the present regulations and technologies (PAMI, 2014). Therefore, the focus of this section will be to discuss some of the limitation of the current system and recent advances in addressing these limitations.

As stated above the two main focuses of truck washing and sanitization processes include the physical removal of the manure and bedding from the trailer and inactivation/elimination of pathogens of concern. These two important steps of the truck biosecurity process use manual labor that has its own inherent problems for cleaning ease and consistency. The quality of cleaning, disinfection, and the methods to verify the cleanliness or safety of the trailer prior to reuse will depend on the training of the personnel, which may vary from individual to individual. Another challenge the current system is imposing on the hog industry is the time required to finish the whole process of washing and disinfection, which forces the transport trailer out of service for prolonged periods of time. Washing times may vary from 30 to 120 minutes depending on category of animals transported, amount of bedding used and season. Drying at room temperature may take up to 24 hours; however, this could be even longer depending on the pathogen under consideration with studies suggesting that trailers would require one week of drying post-washing and disinfection at room temperature to inactivate PEDV (Thomas et al., 2015). Nevertheless, it is widely accepted that in today's large scale commercial hog production systems, trailer sanitation programs that require time periods greater than 2 hours are not economically feasible.

Some advances have been made recently to shorten the time required for drying and effective inactivation of pathogens. The North America Pig Improvement Company (PIC; Franklin, Kentucky) has introduced a new system known as the Thermo-Assisted Drying and Decontamination (TADD) protocol, which fully inactivates pathogens in less than 30 minutes. The system uses high velocity hot air applied to the trailer interior to bring the surface temperature to $\geq$ 71^{o}C for at least 30 minutes and was reported to completely inactivate PRRS virus in less

than 60 minutes (Dee et al., 2005c; Dee et al., 2007) and the PED virus in 10 minutes (Thomas et al., 2015). With forced hot air at $\geq$ 71^{o}C capable of inactivating most pathogens in less than 30 minutes, the whole process of washing and decontamination can be finished in less than 2 hours, making this new system technically effective and economically feasible for the swine industry. However, it requires testing in different weather conditions as the effect during summer seasons could different from conditions encountered in the Canadian winters.

Another challenge in the current standard protocol for vehicle washing and decontamination is the environmental issues associated with the waste (manure, bedding and water) produced and released to the environment during the cleaning and washing procedures. It is hypothesized here that a vacuum system combined with high pressure wash (hydrovac) may help to solve this problem. The excavation industry commonly uses a vacuum system combined with high pressure water to excavate soil from around buried infrastructure. This has successfully achieved excavation even in frozen soils. This system minimizes water use and collects almost 100% of the water and soil. It would appear that such technology (hydrovac) could be adapted to remove manure and bedding from livestock transport trailers effectively with minimal water use and efficient collection of the resulting slurry for processing and/or disposal.

7. Conclusions

Preventing the introduction of pathogens into a farm, region or country will remain a continuous challenge for all stakeholders including pig farmers, veterinarians and policy makers. Biosecurity has become the center of attention in the swine industry as a means to prevent the Introduction of new pathogens into a farm. The preliminary route of disease introduction into a farm is via an infected animal, and most of the current biosecurity protocols are focused on avoiding the introduction of infected pigs. However, most swine viral, bacterial and parasitic pathogens can also be spread to distant farms, regions and even across the border through other routes of diseases transmission, such as animal transport vehicles. In this document, we tried to discuss important swine diseases and their modes of transmission, with the ultimate goal of designing better biosecurity protocols to be used in animal transport vehicles.

The current biosecurity protocol being applied on transport vehicles is based on washing, disinfection and drying. While this protocol has helped the pork industry to minimize the introduction of pathogens into farms, and save the industry from huge economic losses that could have happened due to disease outbreaks, it is being challenged by the emergence of new porcine pathogens such as the PED virus and by the cost and time it requires to completely disinfect a truck. Therefore, additional research is required to develop a new vehicle biosecurity protocol that is more efficient, time and cost oriented, and compatible with new emerging diseases. We hope the information compiled in this document will provide the background information that may help in the design of a new biosecurity protocol for livestock transportation trailers.

8. Appendices

8.1. Summary of characteristics of disinfectants

Characteristics of Selected Disinfectants

For More Information, see the 'Disinfection 101' document at www.cfsph.iastate.edu

Disinfectant Category	Alcohols	Aldehydes	Biguanides	Halogens: Hypochlorites	Halogens: Iodine Compounds	Oxidizing Agents	Phenols	Quaternary Ammonium Compounds (QAC)
Sample Trade Names	Ethyl alcohol Isopropyl alcohol	Formaldehyde Glutaraldehyde	Chlorhexidine Nolvasan® Virosan®	Bleach	Betadyne® Providone®	Hydrogen peroxide Peracetic acid Virkon S® Oxy-Sept 333®	One-Stroke Environ® Pheno-Tek II® Tek-Trol®	Roccal® DiQuat® D-256®
Mechanism of Action	•Precipitates proteins •Denatures lipids	•Denatures proteins •Alkylates nucleic acids	•Alters membrane permeability	•Denatures proteins	•Denatures proteins	•Denature proteins and lipids	• Denatures proteins • Alters cell wall permeability	• Denatures proteins • Binds phospholipids of cell membrane
Advantages	•Fast acting •Leaves no residue	•Broad spectrum	•Broad spectrum	•Broad spectrum •Short contact time •Inexpensive	•Stable in storage •Relatively safe	•Broad spectrum	• Good efficacy with organic material • Non-corrosive • Stable in storage	• Stable in storage • Non-irritating to skin • Effective at high temperatures and high pH (9-10)
Disadvantages	•Rapid evaporation •Flammable	•Carcinogenic •Mucous membranes and tissue irritation •Only use in well ventilated areas	•Only functions in limited pH range (5–7) •Toxic to fish (environmental concern)	•Inactivated by sunlight •Requires frequent application •Corrodes metals •Mucous membrane and tissue irritation	•Inactivated by QACs •Requires frequent application •Corrosive •Stains clothes and treated surfaces	•Damaging to some metals	• Can cause skin and eye irritation	
Precautions	Flammable	Carcinogenic		Never mix with acids; toxic chlorine gas will be released			May be toxic to animals, especially cats and pigs	
Vegetative Bacteria	Effective	Effective	Effective	Effective	Effective	Effective	Effective	YES—Gram Positive Limited—Gram Negative
Mycobacteria	Effective	Effective	Variable	Effective	Limited	Effective	Variable	Variable
Enveloped Viruses	Effective	Effective	Limited	Effective	Effective	Effective	Effective	Variable
Non-enveloped Viruses	Variable	Effective	Limited	Effective	Limited	Effective	Variable	Not Effective
Spores	Not Effective	Effective	Not Effective	Variable	Limited	Variable	Not Effective	Not Effective
Fungi	Effective	Effective	Limited	Effective	Effective	Variable	Variable	Variable
Efficacy with Organic Matter	Reduced	Reduced	?	Rapidly reduced	Rapidly reduced	Variable	Effective	Inactivated
Efficacy with Hard Water	?	Reduced	?	Effective	?	?	Effective	Inactivated
Efficacy with Soap/ Detergents	?	Reduced	Inactivated	Inactivated	Effective	?	Effective	Inactivated

? Information not found

Disclaimer: The use of trade names does not in any way signify endorsement of a particular product. For additional product names, please consult the most recent Compendium of Veterinary Products.

References: Linton AH, Hugo WB, Russel AD. Disinfection in Veterinary and Farm Practice. 1987. Blackwell Scientific Publications; Oxford, England; Quinn PJ, Markey BK. Disinfection and Disease Prevention in Veterinary Medicine, In: Block SS, ed., Disinfection, Sterilization and Preservation. 5th edition. 2001. Lippincott, Williams and Wilkins: Philadelphia.

the Center for Food Security & Public Health
Iowa State University
www.cfsph.iastate.edu

Source: Disinfection 101 by Dvorak, 2008 from the centre for food security and public health Iowas State University

8.2. Effect of different disinfectants against various pig pathogens

Table 1. Bacterial isolates used in the disinfectant study.

Organism	Reference number	Source of isolate
E.coli	NCTC 10418	Reference strain used for establishing and standarising techniques
Actinobacillus pleuropneumoniae	P4681/3/06	Porcine lung lesions during pleuropneumonia outbreak
Bordetella bronchiseptica	P50977/3/04	Porcine nasal turbinates during rhinitis/respiratory disease outbreak
Brachyspira hyodysenteriae	P3695/6B/04	Porcine colon from a case of swine dysentery
E. coli (Abbotstown)	P5297/06	Porcine intestine; neonatal colibacillosis outbreak
Haemophilus parasuis	P600194/1/06	Porcine lung; case of Glasser's disease
Pasteurella multocida	P502613/2/06	Porcine lung lesions; pneumonia outbreak
Salmonella derby	P502102/2/06	Porcine intestine; diarrhoea outbreak
Salmonella enterica Typhimurium	P502512/06	Porcine intestine; diarrhoea outbreak
Salmonella enterica Typhimurium	P502485/06	Porcine intestine; diarrhoea outbreak
Salmonella enterica Typhimurium	P502573/2/06	Porcine intestine; diarrhoea outbreak
Staphylococcus hyicus	P502515/1/06	Porcine skin; outbreak of Greasy pig disease.
Streptococcus suis serotype I/II	P50257/2	Porcine meninges; meningitis outbreak
Yersinia enterocolitica	P5341/06	Porcine intestine; diarrhoea outbreak

Table 2. Disinfectant compounds used in the study.

Key	Active compound	Recommended dilution range
A	Iodine (acidic based)	1:125 – 1:600
B	Glutaraldehyde plus quaternary ammonium	1:50 – 1:190 (SVD 1:250)
C	Peracetic acid plus hydrogen peroxide	1:100 – 1:200
D	Iodine	1:200
E	Quaternary ammonium plus hydrogen peroxide	1:100 – 1:200
F	Quaternary ammonium	1:50 – 1:100
G	Peroxygen	1:100 – 1:200

Table 3. Evaluation of the bacteriocidal activity of seven chemical disinfectants against three respiratory bacterial pathogens of pigs using British Standard Method BSEN 1656:2000 (phase 2 /step 1). Results are given as the lowest effective concentration of product giving at least 10^5 reduction in viable bacterial count under the stated test conditions.

Bacteria	Disinfect ~	Test conditions											
		Low organic matter						High organic matter					
		4° C		10° C		20° C		4° C		10° C		20° C	
		30 min	60min	30 min	60min	30 min	60min	30 min	60min	30 min	60min	30 min	60min
APP^	A	1/1000	1/1000	1/1000	1/1000	1/1000	1/1000	1/800	1/800	1/800	1/800	1/800	1/800
	B	1/200	1/200	1/200	1/200	1/200	1/200	1/100	1/100	1/100	1/100	1/100	1/100
	C	1/1000	1/1000	1/1000	1/1000	1/1000	1/1000	1/800	1/800	1/800	1/800	1/800	1/800
	D	1/100	1/100	1/100	1/100	1/100	1/100	1/100	1/100	1/100	1/100	1/100	1/100
	E	1/10000	1/10000	1/10000	1/10000	1/10000	1/10000	1/1000	1/5000	1/1000	1/5000	1/1000	1/5000
	F	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100
	G	1/800	1/800	1/800	1/800	1/800	1/800	1/100	1/100	1/400	1/400	1/400	1/400
BB*	A	1/200	1/400	1/400	1/400	1/400	1/800	1/200	1/200	1/200	1/200	1/200	1/200
	B	1/200	1/200	1/200	1/200	1/200	1/200	1/100	1/100	1/100	1/100	1/100	1/100
	C	1/200	1/200	1/200	1/200	1/200	1/200	1/100	1/100	1/100	1/100	1/100	1/100
	D	1/200	1/200	1/200	1/400	1/200	1/400	1/100	1/100	1/100	1/200	1/100	1/200
	E	1/400	1/800	1/400	1/1000	1/800	1/1000	1/100	1/100	1/100	1/100	1/100	1/100
	F	1/200	1/200	1/200	1/200	1/200	1/200	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100
	G	1/100	1/100	1/100	1/100	1/100	1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100
PM#	A	1/1000	1/1000	1/1000	1/1000	1/1000	1/1000	1/400	1/400	1/400	1/400	1/400	1/400
	B	1/200	1/200	1/200	1/200	1/200	1/200	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100
	C	NE 1/100	NE 1/100	NE 1/100	NE 1/100	1/100	1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100
	D	NE 1/100	NE 1/100	NE 1/100	NE 1/100	1/100	1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	1/100	1/100
	E	1/1000	1/5000	1/1000	1/5000	1/1000	1/5000	1/100	1/200	1/200	1/200	1/200	1/200
	F	1/100	1/100	1/100	1/100	1/100	1/200	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100
	G	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100

^*Actinobacillus pleuropneumoniae* - field isolate P4681/3/06. Isolated from porcine lung (pleuropneumonia outbreak)
**Bordetella bronchiseptica* - field isolate P50977/3/04. Isolated from porcine nasal turbinates (rhinitis/respiratory disease outbreak)
#*Pasteurella multocida* - field isolate P502613/2/06. Isolated from pneumonic porcine lung tissue (respiratory disease outbreak)
~ The key for the disinfectant compounds is given in Table 2
NE 1/100 = Not effective at the highest concentration tested (1/100)

Table 4. Evaluation of the bacteriocidal activity of seven chemical disinfectants against three bacterial pathogens of pigs using British Standard Method BSEN 1656:2000 (phase 2 /step 1). Results are given as the lowest effective concentration of product giving at least 10^5 reduction in viable bacterial count under the stated test conditions.

Bacteria	Disinfect ~	Test conditions											
		Low organic matter						High organic matter					
		4° C		10° C		20° C		4° C		10° C		20° C	
		30 min	60min	30 min	60min	30 min	60min	30 min	60min	30 min	60min	30 min	60min
HP^	A	1/1000	1/1000	1/1000	1/1000	1/1000	1/1000	1/100	1/100	1/100	1/100	1/100	1/100
	B	1/1000	1/1000	1/1000	1/1000	1/1000	1/1000	1/100	1/100	1/100	1/100	1/100	1/100
	C	1/100	1/100	1/100	1/100	1/100	1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100
	D	1/100	1/100	1/100	1/100	1/100	1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100
	E	1/400	1/400	1/400	1/400	1/400	1/400	1/200	1/200	1/200	1/200	1/200	1/200
	F	1/800	1/800	1/800	1/800	1/800	1/800	1/200	1/200	1/200	1/200	1/400	1/400
	G	1/5000	1/5000	1/5000	1/5000	1/5000	1/5000	1/1000	1/1000	1/1000	1/1000	1/1000	1/1000
SH*	A	1/400	1/800	1/800	1/800	1/800	1/800	NE 1/100	NE 1/100	1/100	1/200	1/100	1/200
	B	1/100	1/200	1/100	1/200	1/100	1/200	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100
	C	1/100	1/100	1/100	1/100	1/100	1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100
	D	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100
	E	1/100	1/100	1/100	1/100	1/100	1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100
	F	NE 1/100	NE 1/100	1/100	1/100	1/100	1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100
	G	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100
SS#	A	1/800	1/800	1/800	1/1000	1/1000	1/1000	1/400	1/800	1/400	1/800	1/400	1/800
	B	1/400	1/400	1/800	1/800	1/800	1/800	1/400	1/400	1/400	1/400	1/400	1/400
	C	1/200	1/200	1/200	1/200	1/200	1/400	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100
	D	1/100	1/100	1/100	1/200	1/100	1/200	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100
	E	1/1000	1/1000	1/1000	1/1000	1/1000	1/1000	1/400	1/400	1/400	1/400	1/400	1/800
	F	1/200	1/800	1/400	1/800	1/400	1/800	1/100	1/100	1/100	1/100	1/200	1/200
	G	1/100	1/100	1/100	1/200	1/100	1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100

^ *Haemophilus parasuis* - field isolate P600194/1/06. Isolated from porcine lung (from a case of Glasser's disease)
* *Staphylococcus hyicus* - field isolate P502515/1/06. Isolated from porcine skin (greasy pig disease outbreak)
# *Streptococcus suis* - field isolate P50257/2 (serotype I/II). Isolated from porcine meninges (meningitis outbreak)
~ The key for the disinfectant compounds is given in Table 2
NE 1/100 = Not effective at the highest concentration tested (1/100)

Table 5. Evaluation of the bacteriocidal activity of seven chemical disinfectants against three *Salmonella enterica* Typhimurium isolates from pigs using British Standard Method BSEN 1656:2000 (phase 2 /step 1). Results are given as the lowest effective concentration of product giving at least 10^5 reduction in viable bacterial count under the stated test conditions.

Bacteria	Disinfect ~	Test conditions											
		Low organic matter						High organic matter					
		4° C		10° C		20° C		4° C		10° C		20° C	
		30 min	60min	30 min	60min	30 min	60min	30 min	60min	30 min	60min	30 min	60min
ST (1)^	A	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100
	B	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100
	C	1/100	1/100	1/100	1/100	1/100	1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100
	D	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100
	E	1/100	1/100	1/100	1/100	1/100	1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100
	F	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100
	G	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100
ST (2)*	A	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100
	B	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100
	C	NE 1/100	NE 1/100	1/100	1/100	1/100	1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100
	D	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100
	E	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100
	F	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100
	G	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100
ST (3)#	A	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100
	B	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100
	C	NE 1/100	NE 1/100	1/100	1/100	1/100	1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100
	D	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100
	E	NE 1/100	NE 1/100	NE 1/100	NE 1/100	1/100	1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100
	F	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100
	G	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100

^ *Salmonella enterica* Typhimurium (1) - field isolate P502512/06. Isolated from porcine intestine (diarrhoea outbreak)
* *Salmonella enterica* Typhimurium (2) - field isolate P502485/06. Isolated from porcine intestine (diarrhoea outbreak)
\# *Salmonella enterica* Typhimurium (3) - field isolate P502573/2/06. Isolated from porcine intestine (diarrhoea outbreak)
~ The key for the disinfectant compounds is given in Table 2
NE 1/100 = Not effective at the highest concentration tested (1/100)

Table 6. Evaluation of the bacteriocidal activity of seven chemical disinfectants against three enteric pathogens of pigs using British Standard Method BSEN 1656:2000 (phase 2 /step 1). Results are given as the lowest effective concentration of product giving at least 10^5 reduction in viable bacterial count under the stated test conditions.

Bacteria	Disinfect ~	Test conditions											
		Low organic matter						High organic matter					
		4° C		10° C		20° C		4° C		10° C		20° C	
		30 min	60min	30 min	60min	30 min	60min	30 min	60min	30 min	60min	30 min	60min
BH^	A	1/400	1/400	1/800	1/800	1/800	1/800	1/200	1/200	1/200	1/200	1/400	1/400
	B	1/800	1/800	1/800	1/800	1/800	1/800	1/200	1/200	1/200	1/200	1/200	1/200
	C	1/800	1/800	1/1000	1/1000	1/1000	1/1000	1/400	1/400	1/800	1/800	1/800	1/800
	D	1/100	1/100	1/200	1/200	1/400	1/400	1/100	1/100	1/100	1/100	1/100	1/100
	E	1/200	1/200	1/800	1/800	1/800	1/800	1/200	1/200	1/400	1/400	1/800	1/800
	F	1/800	1/800	1/800	1/800	1/1000	1/1000	1/200	1/200	1/200	1/200	1/200	1/200
	G	1/200	1/200	1/200	1/200	1/400	1/400	NE 1/100	NE 1/100	1/100	1/100	1/100	1/100
SD*	A	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100
	B	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100
	C	NE 1/100	1/100	NE 1/100	1/100	NE 1/100	1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100
	D	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100
	E	NE 1/100	NE 1/100	1/100	1/100	1/100	1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100
	F	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100
	G	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100
YE#	A	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100
	B	NE 1/100	NE 1/100	NE 1/100	NE 1/100	1/200	1/200	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100
	C	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100
	D	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100
	E	NE 1/100	NE 1/100	NE 1/100	NE 1/100	1/100	1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100
	F	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100
	G	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100

^ *Brachyspira hyodysenteriae* - field isolate P3695/6B/04. Isolated from porcine colon (from a case of swine dysentery)
* *Salmonella derby* - field isolate P502102/2/06. Isolated from porcine intestine (diarrhoea outbreak)
\# *Yersinia enterocolitica* - field isolate P5341/06. Isolated from porcine intestine (diarrhoea outbreak)
~ The key for the disinfectant compounds is given in Table 2
NE 1/100 = Not effective at the highest concentration tested (1/100)

Table 7. Evaluation of the bacteriocidal activity of seven chemical disinfectants against *E.coli* (Abbotstown strain) and E.coli NCTC 10418 (Type strain) using British Standard Method BSEN 1656:2000 (phase 2 /step 1). Results are given as the lowest effective concentration of product giving at least 10^5 reduction in viable bacterial count under the stated test conditions.

Bacteria	Disinfect ~	Test conditions											
		Low organic matter						High organic matter					
		4° C		10° C		20° C		4° C		10° C		20° C	
		30 min	60min	30 min	60min	30 min	60min	30 min	60min	30 min	60min	30 min	60min
EC (A)^	A	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100
	B	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100
	C	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100
	D	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100
	E	1/200	1/200	1/200	1/200	1/100	1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100
	F	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100
	G	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100
EC (T)*	A	1/400	1/400	1/400	1/400	1/400	1/400	1/100	1/100	1/100	1/100	1/100	1/100
	B	1/800	1/800	1/800	1/800	1/800	1/800	1/200	1/200	1/200	1/200	1/200	1/200
	C	1/1000	1/1000	1/1000	1/1000	1/1000	1/1000	1/200	1/200	1/200	1/200	1/200	1/200
	D	1/200	1/400	1/200	1/400	1/400	1/400	NE 1/100	NE 1/100	NE 1/100	NE 1/100	1/100	1/100
	E	1/400	1/800	1/800	1/800	1/1000	1/1000	1/400	1/400	1/400	1/400	1/400	1/400
	F	1/800	1/800	1/800	1/800	1/1000	1/1000	1/100	1/100	1/100	1/200	1/200	1/200
	G	NE 1/100	1/100	1/100	1/100	1/100	1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100	NE 1/100

^ *E.coli* (Abbotstown strain) - field isolate P5297/06. Isolated from porcine intestine (diarrhoea outbreak)
* *E.coli* NCTC 10418 (Type strain)
~ The key for the disinfectant compounds is given in Table 2
NE 1/100 = Not effective at the highest concentration tested (1/100)

Green shading indicates the concentration of disinfectant product that was found to be effective against the specified pathogen under the stated conditions, and that this result is covered by the recommended dilution range for general disinfection purposes.

Yellow shading indicates that the effective concentration of the disinfectant product was higher than that recommended for general disinfection purposes.

Red shading indicates that the disinfectant product was not effective at the stated concentration.

Source: Thomson et al, 2007.

References

Alexandersen, S., Knowles, N.J., Dekker, A., Belsham, G.J., Zhang, Z., Koenen, F. 2012. Picornaviruses. In Diseases of Swine (10th Edition). Zimmerman, J., Karriker, L., Ramirez, A., Schwartz, K.J., Stevenson, G.W., eds. (Hoboken, NJ, USA, Willey-Blackwell).

Allan, G.M., Ellis, J.A., 2000. Porcine circoviruses: a review. J Vet Diagn Invest 12, 3-14.

Alonso, C., Goede, D.P., Morrison, R.B., Davies, P.R., Rovira, A., Marthaler, D.G., Torremorell, M., 2014. Evidence of infectivity of airborne porcine epidemic diarrhea virus and detection of airborne viral RNA at long distances from infected herds. Vet Res 45.

Alsop, J.E., 2005. An outbreak of salmonellosis in a swine finishing barn. J Swine Health Prod 13, 265-268.

Alvarez-Ordonez, A., Martinez-Lobo, F.J., Arguello, H., Carvajal, A., Rubio, P., 2013. Swine dysentery: aetiology, pathogenicity, determinants of transmission and the fight against the disease. International journal of environmental research and public health 10, 1927-1947.

Amano, H., Shibata, M., Kajio, N., Morozumi, T., 1994. Pathologic observations of pigs intranasally inoculated with serovar 1, 4 and 5 of Haemophilus parasuis using immunoperoxidase method. The Journal of veterinary medical science / the Japanese Society of Veterinary Science 56, 639-644.

Amass, S.F., 2004. Diagnosing disinfectant efficacy. J Swine Health Prod 12, 82-83.

Amass, S.F., Clark, L.K., 1999. Biosecurity considerations for pork production units. Swine Health Prod 7, 217-228.

Amass, S.F., Stevenson, G.W., Anderson, C., Grote, L.A., Dowell, C., Vyverberg, B.D., Kanitz, C., Ragland, D., 2000a. Investigation of people as mechanical vectors for porcine reproductive and respiratory syndrome virus. Swine Health Prod 8, 161-166.

Amass, S.F., Vyverberg, B.D., Ragland, D., Dowell, C.A., Anderson, C.D., Stover, J.H., Beaudry, D.J., 2000b. Evaluating the efficacy of boot baths in biosecurity protocols. Swine Health Prod 8, 169-173.

Anonymous 2013. Impact of Inluenza A on Pork Production. In The Pig Site.

Aragon, V., segales, J., Oliveira, S. 2012. Glässer's Disease. In Diseases of Swine, 10th edition, Zimmerman, J., Karriker, L., Ramirez, A., Schwartz, K.J., Stevenson, G.W., eds. (West Sussex, UK, Willey-Blackwell), 760-769.

Assavacheep, P., Rycroft, A.N., 2013. Survival of Actinobacillus pleuropneumoniae outside the pig. Res Vet Sci 94, 22-26.

Bachrach, H.L., Breese, S.S., Jr., Callis, J.J., Hess, W.R., Patty, R.E., 1957. Inactivation of foot-and-mouth disease virus by pH and temperature changes and by formaldehyde. Proc Soc Exp Biol Med 95, 147-152.

Bang, J., Hong, A., Kim, H., Beuchat, L.R., Rhee, M.S., Kim, Y., Ryu, J.H., 2014. Inactivation of Escherichia coli O157:H7 in biofilm on food-contact surfaces by sequential treatments of aqueous chlorine dioxide and drying. International journal of food microbiology 191, 129-134.

Barber, D.A., Bahnson, P.B., Isaacson, R., Jones, C.J., Weigel, R.M., 2002. Distribution of Salmonella in swine production ecosystems. Journal of food protection 65, 1861-1868.

Barham, A.R., Barham, B.L., Johnson, A.K., Allen, D.M., Blanton, J.R., Jr., Miller, M.F., 2002. Effects of the transportation of beef cattle from the feedyard to the packing plant on prevalence levels of Escherichia coli O157 and Salmonella spp. Journal of food protection 65, 280-283.

Batista, L., Pijoan, C., Ruiz, A., Utrera, V., Dee, S., 2004. Assessment of transmission of Mycoplasma hyopneumoniae by personnel. J Swine Health Prod 12, 75-77.

Benfield, D.A., Nelson, E., Collins, J.E., Harris, L., Goyal, S.M., Robison, D., Christianson, W.T., Morrison, R.B., Gorcyca, D., Chladek, D., 1992. Characterization of swine infertility and respiratory syndrome (SIRS) virus (isolate ATCC VR-2332). J Vet Diagn Invest 4, 127-133.

Bharti, A.R., Nally, J.E., Ricaldi, J.N., Matthias, M.A., Diaz, M.M., Lovett, M.A., Levett, P.N., Gilman, R.H., Willig, M.R., Gotuzzo, E., Vinetz, J.M., Leptospirosis, P.-U.S., 2003. Leptospirosis: a zoonotic disease of global importance. Lancet Infect Dis 3, 757-771.

Biberstein, E.L., White, D.C., 1969. A proposal for the establishment of two new Haemophilus species. Journal of medical microbiology 2, 75-78.

Bloemraad, M., de Kluijver, E.P., Petersen, A., Burkhardt, G.E., Wensvoort, G., 1994. Porcine reproductive and respiratory syndrome: temperature and pH stability of Lelystad virus and its survival in tissue specimens from viraemic pigs. Veterinary microbiology 42, 361-371.

Boye, M., Baloda, S.B., Leser, T.D., Moller, K., 2001. Survival of Brachyspira hyodysenteriae and B-pilosicoli in terrestrial microcosms. Veterinary microbiology 81, 33-40.

Bridier, A., Briandet, R., Thomas, V., Dubois-Brissonnet, F., 2011. Resistance of bacterial biofilms to disinfectants: a review. Biofouling 27, 1017-1032.

Brooke, C.J., Riley, T.V., 1999. Erysipelothrix rhusiopathiae: bacteriology, epidemiology and clinical manifestations of an occupational pathogen. Journal of medical microbiology 48, 789-799.

Brown, T.T., Jr., 1981. Laboratory evaluation of selected disinfectants as virucidal agents against porcine parvovirus, pseudorabies virus, and transmissible gastroenteritis virus. American journal of veterinary research 42, 1033-1036.

Brownell, S.A., Nelson, K.L., 2006. Inactivation of single-celled Ascaris suum eggs by low-pressure UV radiation. Applied and environmental microbiology 72, 2178-2184.

Cardona, A.C., Pijoan, C., Dee, S.A., 2005. Assessing Mycoplasma hyopneumoniae aerosol movement at several distances. The Veterinary record 156, 91-92.

Cargill, C.F., Dobson, K.J., 1979. Experimental Sarcoptes Scabiei Infestation in Pigs .2. Effects on Production. Veterinary Record 104, 33-36.

Carlson, S.A., Barnhill, A.E., Griffith, R.W. 2012. Salmonellosis. In Diseases of Swine. 10th edition, Zimmerman, J.J., Karriker, L.A., Ramirez, A., Schwartz, K.J., Stevenson, G.W., eds. (Hoboken, NJ, USA, Willey-Blackwell).

CAST 2008. Fate and Transport of Zoonotic Bacterial, Viral, and Parasitic Pathogens During Swine Manure Treatment, Storage, and Land Application Special Publication. In PorkCheckoff (Council for Agricultural Science and Technology).

Castrucci, M.R., Donatelli, I., Sidoli, L., Barigazzi, G., Kawaoka, Y., Webster, R.G., 1993. Genetic reassortment between avian and human influenza A viruses in Italian pigs. Virology 193, 503-506.

CDC 2008. Guideline for disinfection and sterilization in healthcare facilities. At http://www.cdc.gov (Centers for Diseases Control and Prevention).

CFIA 2015. Canadain Food Insepection Agency Investigation into Feed as a Possible Sources of Porcine Epidemic Diarrhea (CFIA).

CFSPH 2014. Cleaning and Disinfection of Premises. In Just in Time Training At http://www.cfsph.iastate.edu/Emergency-response (Ames, USA, Iowa State University, College of Veteinary Medicine).

Chae, C., 2012. Commercial porcine circovirus type 2 vaccines: Efficacy and clinical application. Veterinary Journal 194, 151-157.

Chapman, P.A., Siddons, C.A., Gerdan Malo, A.T., Harkin, M.A., 1997. A 1-year study of Escherichia coli O157 in cattle, sheep, pigs and poultry. Epidemiol Infect 119, 245-250.

Chasey, D., Cartwright, S.F., 1978. Virus-Like Particles Associated with Porcine Epidemic Diarrhea. Res Vet Sci 25, 255-256.

Chen, Q., Li, G.W., Stasko, J., Thomas, J.T., Stensland, W.R., Pillatzki, A.E., Gauger, P.C., Schwartz, K.J., Madson, D., Yoon, K.J., Stevenson, G.W., Burrough, E.R., Harmon, K.M., Main, R.G., Zhang, J.Q., 2014. Isolation and Characterization of Porcine Epidemic Diarrhea Viruses Associated with the 2013 Disease Outbreak among Swine in the United States. J Clin Microbiol 52, 234-243.

Chia, S.P., Taylor, D.J., 1978. Factors Affecting Survival of Treponema-Hyodysenteriae in Dysenteric Pig Feces. Veterinary Record 103, 68-70.

Christensen, L.S., Mousing, J., Mortensen, S., Soerensen, K.J., Strandbygaard, S.B., Henriksen, C.A., Andersen, J.B., 1990. Evidence of long distance airborne transmission of Aujeszky's disease (pseudorabies) virus. The Veterinary record 127, 471-474.

Clavijo, A., Sanchez-Vazquez, M.J., Buzanovsky, L.P., Martini, M., Pompei, J.C., Cosivi, O., 2015. Current Status and Future Prospects to Achieve Foot-and-Mouth Disease Eradication in South America. Transboundary and emerging diseases.

Clifton-Hadley, F.A., Enright, M.R., 1984. Factors affecting the survival of Streptococcus suis type 2. The Veterinary record 114, 584-586.

Cornick, N.A., Helgerson, A.F., 2004. Transmission and infectious dose of Escherichia coli O157:H7 in swine. Applied and environmental microbiology 70, 5331-5335.

Cornick, N.A., Vukhac, H., 2008. Indirect transmission of Escherichia coli O157:H7 occurs readily among swine but not among sheep. Applied and environmental microbiology 74, 2488-2491.

Corona-Barrera, E., Smith, D.G.E., Murray, B., Thomson, J.R., 2004. Efficacy of seven disinfectant sanitisers on field isolates of Brachyspira pilosicoli. Veterinary Record 154, 473-474.

Cote, C., Quessy, S., 2005. Persistence of Escherichia coli and Salmonella in surface soil following application of liquid hog manure for production of pickling cucumbers. Journal of food protection 68, 900-905.

CSHB, C.S.h.B. 2011. LIVE HOG TRANSPORT VEHICLE WASH / DISINFECT / DRY PROTOCOLS (Sherbrooke, Quebec, M2 Laboratories).

Davies, P.R., 1995. Sarcoptic Mange and Production Performance of Swine - a Review of the Literature and Studies of Associations between Mite Infestation, Growth-Rate and Measures of Mange Severity in Growing Pigs. Veterinary parasitology 60, 249-264.

Dawood, F.S., Jain, S., Finelli, L., Shaw, M.W., Lindstrom, S., Garten, R.J., Gubareva, L.V., Xu, X., Bridges, C.B., Uyeki, T.M., 2009. Emergence of a novel swine-origin influenza A (H1N1) virus in humans. The New England journal of medicine 360, 2605-2615.

Dee, S., Batista, L., Deen, J., Pijoan, C., 2005a. Evaluation of an air-filtration system for preventing aerosol transmission of Porcine reproductive and respiratory syndrome virus. Canadian journal of veterinary research = Revue canadienne de recherche veterinaire 69, 293-298.

Dee, S., Clement, T., Schelkopf, A., Nerem, J., Knudsen, D., Christopher-Hennings, J., Nelson, E., 2014. An evaluation of contaminated complete feed as a vehicle for porcine epidemic diarrhea virus infection of naive pigs following consumption via natural feeding behavior: proof of concept. Bmc Vet Res 10.

Dee, S., Deen, J., Burns, D., Douthit, G., Pijoan, C., 2004a. An assessment of sanitation protocols for commercial transport vehicles contaminated with porcine reproductive and respiratory syndrome virus. Canadian journal of veterinary research = Revue canadienne de recherche veterinaire 68, 208-214.

Dee, S., Deen, J., Burns, D., Douthit, G., Pijoan, C., 2005b. An evaluation of disinfectants for the sanitation of porcine reproductive and respiratory syndrome virus-contaminated transport vehicles at cold temperatures. Canadian journal of veterinary research = Revue canadienne de recherche veterinaire 69, 64-70.

Dee, S., Otake, S., Oliveira, S., Deen, J., 2009a. Evidence of long distance airborne transport of porcine reproductive and respiratory syndrome virus and Mycoplasma hyopneumoniae. Vet Res 40, 39.

Dee, S., Pitkin, A., Deen, J., 2009b. Evaluation of alternative strategies to MERV 16-based air filtration systems for reduction of the risk of airborne spread of porcine reproductive and respiratory syndrome virus. Veterinary microbiology 138, 106-113.

Dee, S., Torremorell, M., Thompson, B., Deen, J., Pijoan, C., 2005c. An evaluation of thermo-assisted drying and decontamination for the elimination of porcine reproductive and respiratory syndrome virus from contaminated livestock transport vehicles. Canadian journal of veterinary research = Revue canadienne de recherche veterinaire 69, 58-63.

Dee, S.A. 2014. Mycoplasma Pneumonia in Pigs. In The Merck Veterinary Manual., Allen, D.G., Constable, P.D., eds.

Dee, S.A. 2014 Swine Influenza In The Merck Veterinary Manual (Whitehouse Station, N.J., U.S.A., Merck Sharp & Dohme Corp).

Dee, S.A., Corey, M.M., 1993. The survival of Streptococcus suis on farm and veterinary equipment. Swine Health Prod 1, 17-20.

Dee, S.A., Deen, J., Otake, S., Pijoan, C., 2004b. An experimental model to evaluate the role of transport vehicles as a source of transmission of porcine reproductive and respiratory syndrome virus to susceptible pigs. Canadian journal of veterinary research = Revue canadienne de recherche veterinaire 68, 128-133.

Dee, S.A., Torremorell, M., Thompson, R., Cano, J.P., Deen, J., Pijoan, C., 2007. Evaluation of the thermo-assisted drying and decontamination system for sanitation of a full-size

transport vehicle contaminated with porcine reproductive and respiratory syndrome virus. J Swine Health Prod. 15, 12-18.
Desrosiers, R., Moore, C., 1998. Indirect transmission of Actinobacillus pleuropneumoniae. Swine Health Prod 6, 263-265.
Dvorak, G. 2008a. Disinfection 101. In The center for Food Security and Public Health. At www.cfsph.iastate.edu, J., R., Amass, S., eds. (Ames, IA, USA, The center for Food Security and Public Health, Iowa State University).
Dvorak, G. 2008b. Disinfection 101 At: http://www.cfsph.iastate.edu/Disinfection/Assets/Disinfection101.pdf, Roth, J., Amass, S., eds. (Ames, IA, Center for Food Security and Public Health, Iowa state University).
EAZWV 2011. Transmissible Disease Fact Sheet 004: African Swine Fever

In Transmissible Diseases Handbook (European Association of Zoo and Wildlife veterinarians).
Edwards, S., 2000. Survival and inactivation of classical swine fever virus. Veterinary microbiology 73, 175-181.
Elbers, A.R., Moser, H., Ekker, H.M., Crauwels, P.A., Stegeman, J.A., Smak, J.A., Pluimers, F.H., 2001. Tracing systems used during the epidemic of classical swine fever in the Netherlands, 1997-1998. Rev Sci Tech 20, 614-629.
Ellis, W.A. 2012. Leptospirosis. In Diseases of Swine. 10th edition, Zimmerman, J.J., Karriker, L.A., Ramirez, A., Schwartz, K.J., Stevenson, G.W., eds. (Hoboken, NJ, USA, Willey-Blackwell).
Ellis, W.A., 2015. Animal leptospirosis. Current topics in microbiology and immunology 387, 99-137.
Ellis, W.A., Mcparland, P.J., Bryson, D.G., Cassells, J.A., 1986a. Boars as Carriers of Leptospires of the Australis Serogroup on Farms with an Abortion Problem. Veterinary Record 118, 563-563.
Ellis, W.A., Mcparland, P.J., Bryson, D.G., Thiermann, A.B., Montgomery, J., 1986b. Isolation of Leptospires from the Genital-Tract and Kidneys of Aborted Sows. Veterinary Record 118, 294-295.
Enright, M.R., Alexander, T.J., Clifton-Hadley, F.A., 1987. Role of houseflies (Musca domestica) in the epidemiology of Streptococcus suis type 2. The Veterinary record 121, 132-133.
Ewart, S.L., 2001. Disinfectants and control of environmental contamination., 3rd edition. Edition. Mosby, St. Louis.
Fairbrother, J.M., Gyles, C.L. 2012. Colibacillosis, In: Zimmerman, J., Karriker, L., Ramirez, A., Stevenson, G. (Eds.) Diseases of Swine 10th edition. Willy-Blackwell, West Sussex, UK.
Fano, E., Pijoan, C., Dee, S., 2005. Dynamics and persistence of Mycoplasma hyopneumoniae infection in pigs. Canadian journal of veterinary research = Revue canadienne de recherche veterinaire 69, 223-228.
FAO 2007. FAO Biosecurity Toolkit (Rome, Food and Agricuklturall Organization of the United Nations).
Favero, M., Bond, W., 2001. Chemical disinfection of medical and surgical materials. Lippincott Williams & Wilkins, Philadelphia.

Fedorka-Cray, P.J., Hogg, A., Gray, J.T., Lorenzen, K., Velasquez, J., Von Behren, P., 1997. Feed and feed trucks as sources of salmonella contamination in swine. Swine Health Prod 5, 189-193.

Fidalgo, S.G., Longbottom, C.J., Rjley, T.V., 2002. Susceptibility of Erysipelothrix rhusiopathiae to antimicrobial agents and home disinfectants. Pathology 34, 462-465.

Floegel, G., Wehrend, A., Depner, K.R., Fritzemeier, J., Waberski, D., Moennig, V., 2000. Detection of Classical Swine Fever virus in semen of infected boars. Veterinary microbiology 77, 109-116.

Fotheringham, V.J.C., 1995. Disinfection of Livestock Production Premises. Rev Sci Tech Oie 14, 191-205.

Fouchier, R.A., Munster, V., Wallensten, A., Bestebroer, T.M., Herfst, S., Smith, D., Rimmelzwaan, G.F., Olsen, B., Osterhaus, A.D., 2005. Characterization of a novel influenza A virus hemagglutinin subtype (H16) obtained from black-headed gulls. Journal of virology 79, 2814-2822.

Francis, D.H., 1999. Colibacillosis in pigs and its diagnosis. Swine Health and Production. At https://www.aasv.org/shap/issues/v5n5/v5n5p189.pdf 7, 241-244.

Fu, Z.F., Hampson, D.J., Blackmore, D.K., 1989. Detection and survival of group A rotavirus in a piggery. The Veterinary record 125, 576-578.

Fussing, V., Barfod, K., Nielsen, R., Moller, K., Nielsen, J.P., Wegener, H.C., Bisgaard, M., 1998. Evaluation and application of ribotyping for epidemiological studies of Actinobacillus pleuropneumoniae in Denmark. Veterinary microbiology 62, 145-162.

Gaasenbeek, C.P.H., Borgsteede, F.H.M., 1998. Studies on the survival of Ascaris suum eggs under laboratory and simulated field conditions. Veterinary parasitology 75, 227-234.

Gamage, B. 2003. Selection and use of disinfectants, Control, B.C.f.D., ed. (BC Centre for Disease Contrrol).

Gloster, J., Freshwater, A., Sellers, R.F., Alexandersen, S., 2005. Re-assessing the likelihood of airborne spread of foot-and-mouth disease at the start of the 1967-1968 UK foot-and-mouth disease epidemic. Epidemiol Infect 133, 767-783.

Gloster, J., Sellers, R.F., Donaldson, A.I., 1982. Long distance transport of foot-and-mouth disease virus over the sea. The Veterinary record 110, 47-52.

Goodwin, R.F.W., 1972. Survival of Mycoplasma-Suipneumoniae in Liquid Medium, on Solid Medium and in Pneumonic Tissue. Res Vet Sci 13, 203-&.

Goodwin, R.F.W., 1985. Apparent Reinfection of Enzootic-Pneumonia-Free Pig Herds - Search for Possible Causes. Veterinary Record 116, 690-694.

Gottschalk, M. 2012. Actinobacillosis. In Diseases of Swine, 10th Edition., Zimmerman, J., Karriker, L., Ramirez, A., Stevenson, G., eds. (Hoboken, NJ, USA, Wiley-Blackwell).

Gottschalk, M., Segura, M., Xu, J., 2007. Streptococcus suis infections in humans: the Chinese experience and the situation in North America. Animal health research reviews / Conference of Research Workers in Animal Diseases 8, 29-45.

Gough, P.M., Jorgenson, R.D., 1983. Identification of porcine transmissible gastroenteritis virus in house flies (Musca domestica Linneaus). American journal of veterinary research 44, 2078-2082.

Gray, J.T., Fedorka-Cray, P.J., 2001. Survival and infectivity of Salmonella choleraesuis in swine feces. Journal of food protection 64, 945-949.

Greatorex, J.S., Digard, P., Curran, M.D., Moynihan, R., Wensley, H., Wreghitt, T., Varsani, H., Garcia, F., Enstone, J., Nguyen-Van-Tam, J.S., 2011. Survival of influenza A(H1N1) on materials found in households: implications for infection control. Plos One 6, e27932.

Greve, J.H., Davies, T. 2012. External Parasites. In Diseases of Swine, 10th edition, Zimmerman, J., Karriker, L., Ramirez, A., Schwartz, K.J., Stevenson, G.W., eds. (West Sussex, UK, Willey-Blackwell), 885 -894.

Grubman, M.J., Baxt, B., 2004. Foot-and-mouth disease. Clinical microbiology reviews 17, 465-493.

Gutierrez, C.B., Rodriguez Barbosa, J.I., Suarez, J., Gonzalez, O.R., Tascon, R.I., Rodriguez Ferri, E.F., 1995. Efficacy of a variety of disinfectants against Actinobacillus pleuropneumoniae serotype 1. American journal of veterinary research 56, 1025-1029.

Haas, B., Ahl, R., Bohm, R., Strauch, D., 1995. Inactivation of viruses in liquid manure. Rev Sci Tech 14, 435-445.

Halbur, P.a.O., T. 2006. Practical management of PCV2-associated diseases. In: The American experience. In proceedings of the American Association of Swine Veterinarians Annual Meeting.

Hampson, D.J. 2012. Brachyspiral Colitis. In Diseases of Swine, 10th Edition., Zimmerman, J., Karriker, L., Ramirez, A., Stevenson, G., eds. (Hoboken, NJ, USA, Wiley-Blackwell).

Harding, J.C. 1996. Postweaning Multisystemic Wasting Syndrome: Preliminary epidemiology and clinical findings. In: Proc. West. Can. Assoc. Swine. Pract., 21.

Harris, D.L.H. 2013. Transmissible Gastroenteritis in Pigs. In The Merck Veterinary Manual

Hege, R., Zimmermann, W., Scheidegger, R., Stark, K.D., 2002. Incidence of reinfections with Mycoplasma hyopneumoniae and Actinobacillus pleuropneumoniae in pig farms located in respiratory-disease-free regions of Switzerland--identification and quantification of risk factors. Acta Vet Scand 43, 145-156.

Holley, R.A., Arrus, K.M., Ominski, K.H., Tenuta, M., Blank, G., 2006. Salmonella survival in manure-treated soils during simulated seasonal temperature exposure. Journal of environmental quality 35, 1170-1180.

Holtkamp, D.J., Kliebenstein, J.B., Neumann, E.J., Zimmerman, J.J., Rotto, H.F., Yoder, T.K., Wang, C., Yeske, P.E., Mowrer, C.L., Haley, C.A., 2013. Assessment of the economic impact of porcine reproductive and respiratory syndrome virus on United States pork producers. J Swine Health Prod 21, 72-84.

Huang, Y.W., Dickerman, A.W., Pineyro, P., Li, L., Fang, L., Kiehne, R., Opriessnig, T., Meng, X.J., 2013. Origin, evolution, and genotyping of emergent porcine epidemic diarrhea virus strains in the United States. mBio 4, e00737-00713.

Huong, V.T., Ha, N., Huy, N.T., Horby, P., Nghia, H.D., Thiem, V.D., Zhu, X., Hoa, N.T., Hien, T.T., Zamora, J., Schultsz, C., Wertheim, H.F., Hirayama, K., 2014. Epidemiology, clinical manifestations, and outcomes of Streptococcus suis infection in humans. Emerging infectious diseases 20, 1105-1114.

Hurnik, D. 2005. Investigations into optimal washing and disinfection techniques for pig pens. In London Swine Conference- Production at the Leading Edge (London).

Isaacson, R.E., Firkins, L.D., Weigel, R.M., Zuckermann, F.A., DiPietro, J.A., 1999. Effect of transportation and feed withdrawal on shedding of Salmonella typhimurium among experimentally infected pigs. American journal of veterinary research 60, 1155-1158.

ISU 2015a. Mycoplasmal Pneumonia (Enzootic Pneumonia). In Veterinary Diagnostics and Production Animal Medicine (Ames, Iowa, USA, Iowa State University, College of Veterinary Medicine).

ISU 2015b. Salmonellosis. In Veterinary Diagnostics and Production Animal Medicine (Ames, Iowa, USA, Iowa State University, College of Veterinary Medicine).

Ito, T., Couceiro, J.N., Kelm, S., Baum, L.G., Krauss, S., Castrucci, M.R., Donatelli, I., Kida, H., Paulson, J.C., Webster, R.G., Kawaoka, Y., 1998. Molecular basis for the generation in pigs of influenza A viruses with pandemic potential. Journal of virology 72, 7367-7373.

Jobert, J.L., Savoye, C., Cariolet, R., Kobisch, M., Madec, F., 2000. Experimental aerosol transmission of Actinobacillus pleuropneumoniae to pigs. Canadian Journal of Veterinary Research-Revue Canadienne De Recherche Veterinaire 64, 21-26.

John, D.E., Haas, C.N., Nwachuku, N., Gerba, C.P., 2005. Chlorine and ozone disinfection of Encephalitozoon intestinalis spores. Water Res 39, 2369-2375.

Kao, R.R., 2003. The impact of local heterogeneity on alternative control strategies for foot-and-mouth disease. Proceedings. Biological sciences / The Royal Society 270, 2557-2564.

Kelley, G.W., Smith, L.J., 1956. The Daily Egg Production of Ascaris-Suum and the Inability of Low Levels of Aureomycin to Affect Egg Production and Embryonation. J Parasitol 42, 587-587.

Kemenes, F., Suveges, T., 1976. Leptospira-induced repeated abortion in sows. Acta veterinaria Academiae Scientiarum Hungaricae 26, 395-403.

Keyser, M., Muller, I.A., Cilliers, F.P., Nel, W., Gouws, P.A., 2008. Ultraviolet radiation as a non-thermal treatment for the inactivation of microorganisms in fruit juice. Innov Food Sci Emerg 9, 348-354.

Khairani-Bejo, S., Bahaman, A.R., Zamri-Saad, M., Mutalib, A.R., 2004. The Survival of Leptospira interrogans Serovar Hardjo in the Malaysian Environment. Journal of Animal and Veterinary Advances, 123-129.

Khan, S.U., Atanasova, K.R., Krueger, W.S., Ramirez, A., Gray, G.C., 2013. Epidemiology, geographical distribution, and economic consequences of swine zoonoses: a narrative review. Emerg Microbes Infec 2.

Kielstein, P., Rapp-Gabrielson, V.J., 1992. Designation of 15 serovars of Haemophilus parasuis on the basis of immunodiffusion using heat-stable antigen extracts. J Clin Microbiol 30, 862-865.

Kim, H.B., Lyoo, K.S., Joo, H.S., 2009. Efficacy of different disinfectants in vitro against porcine circovirus type 2. Veterinary Record 164, 599-600.

Kim, J., Diao, J., Shepherd, M.W., Jr., Singh, R., Heringa, S.D., Gong, C., Jiang, X., 2012a. Validating thermal inactivation of Salmonella spp. in fresh and aged chicken litter. Applied and environmental microbiology 78, 1302-1307.

Kim, J.S., Oh, D.S., Ahn, K.S., Shin, S.S., 2012b. Effects of kimchi extract and temperature on embryostasis of Ascaris suum eggs. The Korean journal of parasitology 50, 83-87.

Kingscote, B.F., 1986a. Leptospirosis in red foxes in Ontario. Journal of wildlife diseases 22, 475-478.

Kingscote, B.F., 1986b. Leptospirosis outbreak in a piggery in southern alberta. Can Vet J 27, 188-190.

Klinkenberg, D., de Bree, J., Laevens, H., de Jong, M.C.M., 2002. Within- and between-pen transmission of Classical Swine Fever Virus: a new method to estimate the basic reproduction ratio from transmission experiments. Epidemiol Infect 128, 293-299.

Knight-Jones, T.J.D., Rushton, J., 2013. The economic impacts of foot and mouth disease - What are they, how big are they and where do they occur? Preventive veterinary medicine 112, 161-173.

Knowles, N.J., He, J., Shang, Y., Wadsworth, J., Valdazo-Gonzalez, B., Onosato, H., Fukai, K., Morioka, K., Yoshida, K., Cho, I.S., Kim, S.M., Park, J.H., Lee, K.N., Luk, G., Borisov, V., Scherbakov, A., Timina, A., Bold, D., Nguyen, T., Paton, D.J., Hammond, J.M., Liu, X., King, D.P., 2012. Southeast Asian foot-and-mouth disease viruses in Eastern Asia. Emerging infectious diseases 18, 499-501.

Kramer, A., Schwebke, I., Kampf, G., 2006. How long do nosocomial pathogens persist on inanimate surfaces? A systematic review. BMC infectious diseases 6, 130.

Kristensen, C.S., Baekbo, P., Bille-Hansen, V., Botner, A., Vigre, H., Enoe, C., Larsen, L.E., 2009. Induction of porcine post-weaning multisystemic wasting syndrome (PMWS) in pigs from PMWS unaffected herds following mingling with pigs from PMWS-affected herds. Veterinary microbiology 138, 244-250.

Krug, P.W., Larson, C.R., Eslami, A.C., Rodriguez, L.L., 2012. Disinfection of foot-and-mouth disease and African swine fever viruses with citric acid and sodium hypochlorite on birch wood carriers. Veterinary microbiology 156, 96-101.

Krug, P.W., Lee, L.J., Eslami, A.C., Larson, C.R., Rodriguez, L., 2011. Chemical disinfection of high-consequence transboundary animal disease viruses on nonporous surfaces. Biologicals : journal of the International Association of Biological Standardization 39, 231-235.

Labare, M.P., Soohoo, H., Kim, D., Tsoi, K., Liotta, J.L., Bowman, D.D., 2013. Ineffectiveness of a quaternary ammonium salt and povidone-iodine for the inactivation of Ascaris suum eggs. American journal of infection control 41, 360-361.

Larochelle, R., Bielanski, A., Muller, P., Magar, R., 2000. PCR detection and evidence of shedding of porcine circovirus type 2 in boar semen. J Clin Microbiol 38, 4629-4632.

Laude, H., 1981. Thermal inactivation studies of a coronavirus, transmissible gastroenteritis virus. The Journal of general virology 56, 235-240.

Lee, J.Y.S., Wilson, M.R., 1979. Review of Pseudorabies (Aujeszkys Disease) in Pigs. Can Vet J 20, 65-69.

Leforban, Y., Gerbier, G., 2002. Review of the status of foot and mouth disease and approach to control/eradication in Europe and Central Asia. Rev Sci Tech Oie 21, 477-492.

Letellier, A., Messier, S., Lessard, L., Quessy, S., 2000. Assessment of various treatments to reduce carriage of Salmonella in swine. Canadian journal of veterinary research = Revue canadienne de recherche veterinaire 64, 27-31.

Levett, P.N., 2001. Leptospirosis. Clinical microbiology reviews 14, 296-326.

Levis, D.G., Baker, R.B. 2011. Biosecurity of Pigs and Farm Security (University of Nebraska - Lincoln Extension, IANR pub.), 1-28.
Lillard, S. 2004. How Ozone Affects Bacteria, Fungus, Molds And Viruses.
Lobova, D., Cizek, A., 2004. Bactericidal efficacy of two disinfectants against Brachyspira hyodysenteriae and one feed supplement against B-hyodysenteriae and B-pilosicoli. Vet Med-Czech 49, 156-160.
Losson, B.J., Mignon, B. 2011. Mange in Pigs. In The Merck Veterinary Manual (Whitehouse Station, NJ. USA, Merck Sharp & Dohme).
Lowe, J., Gauger, P., Harmon, K., Zhang, J.Q., Connor, J., Yeske, P., Loula, T., Levis, I., Dufresne, L., Main, R., 2014. Role of Transportation in Spread of Porcine Epidemic Diarrhea Virus Infection, United States. Emerging infectious diseases 20, 872-874.
Ma, W., Richt, J.A., 2010. Swine influenza vaccines: current status and future perspectives. Animal health research reviews / Conference of Research Workers in Animal Diseases 11, 81-96.
Ma, Y., Zhang, Y., Liang, X., Lou, F., Oglesbee, M., Krakowka, S., Li, J., 2015. Origin, evolution, and virulence of porcine deltacoronaviruses in the United States. mBio 6.
MacInnes, J.I., Gottschalk, M., Lone, A.G., Metcalf, D.S., Ojha, S., Rosendal, T., Watson, S.B., Friendship, R.M., 2008. Prevalence of Actinobacillus pleuropneumoniae, Actinobacillus suis, Haemophilus parasuis, Pasteurella multocida, and Streptococcus suis in representative Ontario swine herds. Canadian Journal of Veterinary Research-Revue Canadienne De Recherche Veterinaire 72, 242-248.
Madec, F., Eveno, E., Morvan, P., Hamon, L., Blanchard, P., Cariolet, R., Amenna, N., Morvan, H., Truong, C., Mahe, D., Albina, E., Jestin, A., 2000. Post-weaning multisystemic wasting syndrome (PMWS) in pigs in France: clinical observations from follow-up studies on affected farms. Livest Prod Sci 63, 223-233.
Marion-Ferey, K., Pasmore, M., Stoodley, P., Wilson, S., Husson, G.P., Costerton, J.W., 2003. Biofilm removal from silicone tubing: an assessment of the efficacy of dialysis machine decontamination procedures using an in vitro model. The Journal of hospital infection 53, 64-71.
McClurkin, A.W., Stark, S.L., Norman, J.O., 1970. Transmissible gastroenteritis (TGE) of swine: the possible role of dogs in the epizootiology of TGE. Canadian journal of comparative medicine. Revue canadienne de medecine comparee 34, 347-349.
McColl, K.A., Westbury, H.A., Kitching, R.P., Lewis, V.M., 1995. The persistence of foot-and-mouth disease virus on wool. Australian veterinary journal 72, 286-292.
McDonnell, G., Dehen, C., Perrin, A., Thomas, V., Igel-Egalon, A., Burke, P.A., Deslys, J.P., Comoy, E., 2013. Cleaning, disinfection and sterilization of surface prion contamination. The Journal of hospital infection 85, 268-273.
Medici, K.C., Barry, A.F., Alfieri, A.F., Alfieri, A.A., 2011. Porcine rotavirus groups A, B, and C identified by polymerase chain reaction in a fecal sample collection with inconclusive results by polyacrylamide gel electrophoresis. J Swine Health Prod 19, 146-150.
Meggison, D.L., Jr., Mueller, W.S., 1956. Effect of a quaternary ammonium germicide on electrophoretic mobility of Escherichia coli in various salt solutions. Applied microbiology 4, 119-121.

Meunier, L., Canonica, S., von Gunten, U., 2006. Implications of sequential use of UV and ozone for drinking water quality. Water Res 40, 1864-1876.
Meyns, T., Maes, D., Dewulf, J., Vicca, J., Haesebrouck, F., de Kruif, A., 2004. Quantification of the spread of Mycoplasma hyopneumoniae in nursery pigs using transmission experiments. Preventive veterinary medicine 66, 265-275.
Moennig, V., Floegel-Niesmann, G., Greiser-Wilke, I., 2003. Clinical signs and epidemiology of classical swine fever: A review of new knowledge. Veterinary Journal 165, 11-20.
Morozumi, T., Hiramune, T., 1982. Effect of temperature on the survival of Haemophilus parasuis in physiological saline. National Institute of Animal Health quarterly 22, 90-91.
Mur, L., Martinez-Lopez, B., Sanchez-Vizcaino, J.M., 2012. Risk of African swine fever introduction into the European Union through transport-associated routes: returning trucks and waste from international ships and planes. Bmc Vet Res 8, 149.
Muroga, N., Kobayashi, S., Nishida, T., Hayama, Y., Kawano, T., Yamamoto, T., Tsutsui, T., 2013. Risk factors for the transmission of foot-and-mouth disease during the 2010 outbreak in Japan: a case-control study. Bmc Vet Res 9, 150.
Myers, K.P., Olsen, C.W., Gray, G.C., 2007. Cases of swine influenza in humans: a review of the literature. Clinical infectious diseases : an official publication of the Infectious Diseases Society of America 44, 1084-1088.
Nansen, P., Roepstorff, A., 1999. Parasitic helminths of the pig: factors influencing transmission and infection levels. International journal for parasitology 29, 877-891.
Neumann, E.J. 2014. Swine Disease Manual, 4th edition.
Neumann, E.J., Kliebenstein, J.B., Johnson, C.D., Mabry, J.W., Bush, E.J., Seitzinger, A.H., Green, A.L., Zimmerman, J.J., 2005. Assessment of the economic impact of porcine reproductive and respiratory syndrome on swine production in the United States. Journal of the American Veterinary Medical Association 227, 385-392.
Ngwai, Y.B., Adachi, Y., Ogawa, Y., Hara, H., 2006. Characterization of biofilm-forming abilities of antibiotic-resistant Salmonella typhimurium DT104 on hydrophobic abiotic surfaces. Journal of microbiology, immunology, and infection = Wei mian yu gan ran za zhi 39, 278-291.
Nims, R.W., Gauvin, G., Plavsic, M., 2011. Gamma irradiation of animal sera for inactivation of viruses and mollicutes--a review. Biologicals : journal of the International Association of Biological Standardization 39, 370-377.
Nuanualsuwan, S., Cliver, D.O., 2003. Infectivity of RNA from inactivated poliovirus. Applied and environmental microbiology 69, 1629-1632.
Nwachuku, N., Gerba, C.P., Oswald, A., Mashadi, F.D., 2005. Comparative inactivation of adenovirus serotypes by UV light disinfection. Applied and environmental microbiology 71, 5633-5636.
Ochiai, S., Adachi, Y., Mori, K., 1997. Unification of the genera Serpulina and Brachyspira, and proposals of Brachyspira hyodysenteriae comb nov, Brachyspira innocens comb nov and Brachyspira pilosicoli comb nov. Microbiol Immunol 41, 445-452.
OIE 2008. PRRS: the disease, its diagnosis, prevention and control. In Report of The OIE Ad Hock Group on Porcine Reproductive and Respiratory Syndromme (Paris, OIE).
OIE 2012. Terrestrial Animal Health Code, Health, W.O.f.A., ed. (Paris, World Organization for Animal Health).

Oie, S., Kamiya, A., Tomita, M., Katayama, A., Iwasaki, A., Miyamura, S., 1999. Efficacy of disinfectants and heat against Escherichia coli O157:H7. Microbios 98, 7-14.
Ojeh, C.K., Cusack, T.M., Yolken, R.H., 1995. Evaluation of the Effects of Disinfectants on Rotavirus Rna and Infectivity by the Polymerase Chain-Reaction and Cell-Culture Methods. Mol Cell Probe 9, 341-346.
Ojkic, D., Hazlett, M., Fairles, J., Marom, A., Slavic, D., Maxie, G., Alexandersen, S., Pasick, J., Alsop, J., Burlatschenko, S., 2015. The first case of porcine epidemic diarrhea in Canada. Can Vet J 56, 149-152.
Okolo, M.I., 1986. Isolation of Erysipelothrix rhusiopathiae from apparently healthy pigs reared under intensive and free range systems of management. Microbios 47, 29-35.
Oliveira, S., Galina, L., Blanco, I., Canals, A., Pijoan, C., 2003. Naturally-farrowed, artificially-reared pigs as an alternative model for experimental infection by Haemophilus parasuis. Canadian journal of veterinary research = Revue canadienne de recherche veterinaire 67, 146-150.
Oliveira, S., Pijoan, C., 2004. Haemophilus parasuis: new trends on diagnosis, epidemiology and control. Veterinary microbiology 99, 1-12.
Olsen, L.S., Kelley, G.W., Sen , H.G., 1958. Longivity and Egg-Production of Ascaris suum. Transactions of the American Microscopical Society 77, 380 - 383.
Opriessnig, T., Wood, R.L. 2012. Erisipelas. In Diseases of Swine, 10th Edition, Zimmerman, J., Karriker, L., Ramirez, A., Schwartz, K.J., Stevenson, G.W., eds. (West Sussex, UK, Willey-Blackwell).
Otake, S., Dee, S., Corzo, C., Oliveira, S., Deen, J., 2010. Long-distance airborne transport of infectious PRRSV and Mycoplasma hyopneumoniae from a swine population infected with multiple viral variants. Veterinary microbiology 145, 198-208.
Otake, S., Dee, S.A., Jacobson, L., Torremorell, M., Pijoan, C., 2002a. Evaluation of aerosol transmission of porcine reproductive and respiratory syndrome virus under controlled field conditions. The Veterinary record 150, 804-808.
Otake, S., Dee, S.A., Moon, R.D., Rossow, K.D., Trincado, C., Pijoan, C., 2004. Studies on the carriage and transmission of porcine reproductive and respiratory syndrome virus by individual houseflies (Musca domestica). The Veterinary record 154, 80-85.
Otake, S., Dee, S.A., Rossow, K.D., Joo, H.S., Deen, J., Molitor, T.W., Pijoan, C., 2002b. Transmission of porcine reproductive and respiratory syndrome virus by needles. The Veterinary record 150, 114-115.
Otake, S., Dee, S.A., Rossow, K.D., Moon, R.D., Pijoan, C., 2002c. Mechanical transmission of porcine reproductive and respiratory syndrome virus by mosquitoes, Aedes vexans (Meigen). Canadian journal of veterinary research = Revue canadienne de recherche veterinaire 66, 191-195.
Oura, C. 2013. The Merck Veterinary Manual. In Overview of African Swine Fever.
PAMI 2014. Preliminary Investigation of a Reliable and Effective Swine Transportation Sanitization System. In Sask Pork Forum, Browne, K., ed. (Saskatoon, SK, Saskatchewan Pork Development Board).
Parker, J., Walker, M., 2011. Survival of a pathogenic Leptospira serovar in response to combined in vitro pH and temperature stresses. Veterinary microbiology 152, 146-150.

Pasick, J., Berhane, Y., Ojkic, D., Maxie, G., Embury-Hyatt, C., Swekla, K., Handel, K., Fairles, J., Alexandersen, S., 2014. Investigation into the role of potentially contaminated feed as a source of the first-detected outbreaks of porcine epidemic diarrhea in Canada. Transboundary and emerging diseases 61, 397-410.

Paster, B.J., Dewhirst, F.E., 2000. Phylogenetic foundation of spirochetes. Journal of molecular microbiology and biotechnology 2, 341-344.

Patterson, A.R., Baker, R.B., Madson, D.M., Pintar, A.L., Opriessnig, T., 2011a. Disinfection protocols reduce the amount of porcine circovirus type 2 in contaminated 1:61 scale model livestock transport vehicles. J Swine Health Prod 19, 156-164.

Patterson, A.R., Madson, D.M., Halbur, P.G., Opriessnig, T., 2011b. Shedding and infection dynamics of porcine circovirus type 2 (PCV2) after natural exposure. Veterinary microbiology 149, 225-229.

Penrith, M.L., Vosloo, W., 2009. Review of African swine fever: transmission, spread and control. Journal of the South African Veterinary Association 80, 58-62.

Pensaert, M.B., de Bouck, P., 1978. A new coronavirus-like particle associated with diarrhea in swine. Archives of virology 58, 243-247.

Perry, B.D., Kalpravidh, W., Coleman, P.G., Horst, H.S., McDermott, J.J., Randolph, T.F., Gleeson, L.J., 1999. The economic impact of foot and mouth disease and its control in South-East Asia: a preliminary assessment with special reference to Thailand. Rev Sci Tech 18, 478-497.

Pieper, U., Kapadia, G., Zhu, P.P., Peterkofsky, A., Herzberg, O., 1995. Structural evidence for the evolutionary divergence of mycoplasma from gram-positive bacteria: the histidine-containing phosphocarrier protein. Structure 3, 781-790.

Pitkin, A., Deen, J., Dee, S., 2009a. Use of a production region model to assess the airborne spread of porcine reproductive and respiratory syndrome virus. Veterinary microbiology 136, 1-7.

Pitkin, A., Deen, J., Otake, S., Moon, R., Dee, S., 2009b. Further assessment of houseflies (Musca domestica) as vectors for the mechanical transport and transmission of porcine reproductive and respiratory syndrome virus under field conditions. Canadian journal of veterinary research = Revue canadienne de recherche veterinaire 73, 91-96.

Pitkin, A., Otake, S., Dee, S. 2009c. Biosecurity Measures for the Prevention of Spread of PRRS virus. In AASV.

Pohl, S., Bertschinger, H.U., Frederiksen, W., Mannheim, W., 1983. Transfer of Hemophilus-Pleuropneumoniae and the Pasteurella-Haemolytica-Like Organism Causing Porcine Necrotic Pleuropneumonia to the Genus Actinobacillus (Actinobacillus-Pleuropneumoniae Comb Nov) on the Basis of Phenotypic and Deoxyribonucleic-Acid Relatedness. Int J Syst Bacteriol 33, 510-514.

Pointon, A.M., Byrt, D., Heap, P., 1985. Effect of Enzootic Pneumonia of Pigs on Growth-Performance. Australian veterinary journal 62, 13-18.

PorkCheckoff 2013. Evaluation of Stalosan F disinfectant to inactivate Porcine Epidemic Diarrhea Virus and Porcine Reproductive and Respiratory Virus when applied to commercial hog trailers. In PEDV Reaseasrch Update 2013 (Iowa State University).

PorkCheckoff 2015. Disinfectant Expands Options to Fight PEDV. In PorkCheckoff update reports (National Pork Board).

Pospischil, A., Stuedli, A., Kiupel, M., 2002. Update on porcine epidemic diarrhea. J Swine Health Prod 10, 81-85.
Poumian, A.M., 1995. Disinfection of Trucks and Trailers. Rev Sci Tech Oie 14, 171-176.
Proux, K., Cariolet, R., Fravalo, P., Houdayer, C., Keranflech, A., Madec, F., 2001. Contamination of pigs by nose-to-nose contact or airborne transmission of Salmonella Typhimurium. Vet Res 32, 591-600.
Quinn, P.J., Markey, B.K., 2001. Disinfection and disease prevention in veterinary medicine., 5th Edition. Lippincott Williams & Wilkins, Philadelphia
Ramos, A.C., Souza, G.N., Lilenbaum, W., 2006. Influence of leptospirosis on reproductive performance of sows in Brazil. Theriogenology 66, 1021-1025.
Ramos, A.P., Stefanelli, C.C., Linhares, R.E., de Brito, B.G., Santos, N., Gouvea, V., de Cassia Lima, R., Nozawa, C., 2000. The stability of porcine rotavirus in feces. Veterinary microbiology 71, 1-8.
Rapp-Gabrielson, V.J., Gabrielson, D.A., 1992. Prevalence of Haemophilus parasuis serovars among isolates from swine. American journal of veterinary research 53, 659-664.
Razin, S., Yogev, D., Naot, Y., 1998. Molecular biology and pathogenicity of mycoplasmas. Microbiology and molecular biology reviews : MMBR 62, 1094-1156.
Reboli, A.C., Farrar, W.E., 1989. Erysipelothrix rhusiopathiae: an occupational pathogen. Clinical microbiology reviews 2, 354-359.
Reed, W.M., Olander, H.J., Thacker, H.L., 1986. Studies on the Pathogenesis of Salmonella-Typhimurium and Salmonella-Choleraesuis Var Kunzendorf Infection in Weanling Pigs. American journal of veterinary research 47, 75-83.
Restaino, L., Frampton, E.W., Hemphill, J.B., Palnikar, P., 1995. Efficacy of ozonated water against various food-related microorganisms. Applied and environmental microbiology 61, 3471-3475.
Ribbens, S., Dewulf, J., Koenen, F., Laevens, H., de Kruif, A., 2004a. Transmission of classical swine fever. A review. Vet Quart 26, 146-155.
Ribbens, S., Dewulf, J., Koenen, F., Laevens, H., Mintiens, K., De Kruif, A., 2004b. An experimental infection (II) to investigate the importance of indirect classical swine fever virus transmission by excretions and secretions of infected weaner pigs. J Vet Med B 51, 438-442.
Roberts, M., 1995. Evaluation of Optimal Size of Restriction Zones in Disease Control with Particular Reference to Classical Swine Fever. Society for Veterinary Epidemiology and Preventive Medicine, Proceedings, 119-130.
Robertson, I.D., Blackmore, D.K., Hampson, D.J., Fu, Z.F., 1991. A Longitudinal-Study of Natural Infection of Piglets with Streptococcus-Suis Type-1 and Type-2. Epidemiol Infect 107, 119-126.
Rodriguez Ferri, E.F., Martinez, S., Frandoloso, R., Yubero, S., Gutierrez Martin, C.B., 2010. Comparative efficacy of several disinfectants in suspension and carrier tests against Haemophilus parasuis serovars 1 and 5. Res Vet Sci 88, 385-389.
Roepstorff, A., Murrell, K.D., 1997. Transmission dynamics of helminth parasites of pigs on continuous pasture: Ascaris suum and Trichuris suis. International journal for parasitology 27, 563-572.

Romero, C.H., Meade, P.N., Shultz, J.E., Chung, H.Y., Gibbs, E.P., Hahn, E.C., Lollis, G., 2001. Venereal transmission of pseudorabies viruses indigenous to feral swine. Journal of wildlife diseases 37, 289-296.

Rose, N., Opriessnig, T., Grasland, B., Jestin, A., 2012. Epidemiology and transmission of porcine circovirus type 2 (PCV2). Virus research 164, 78-89.

Royer, R.L., Nawagitgul, P., Halbur, P.G., Paul, P.S., 2001. Susceptibility of porcine circovirus type 2 to commercial and laboratory disinfectants. J Swine Health Prod 9, 281-284.

Scheidt, A.B., Rueff, L.R., Grant, R.H., Teclaw, R.F., Hill, M.A., Meyer, K.B., Clark, L.K., 1991. Epizootic of pseudorabies among ten swine herds. Journal of the American Veterinary Medical Association 199, 725-730.

Segales, J., Sitjar, M., Domingo, M., Dee, S., Del Pozo, M., Noval, R., Sacristan, C., De las Heras, A., Ferro, A., Latimer, K.S., 1997. First report of post-weaning multisystemic wasting syndrome in pigs in Spain. Veterinary Record 141, 600-601.

Singla, R., Goel, H., Ganguli, A., 2014. Novel synergistic approach to exploit the bactericidal efficacy of commercial disinfectants on the biofilms of Salmonella enterica serovar Typhimurium. Journal of bioscience and bioengineering 118, 34-40.

Sinkala, Y., Simuunza, M., Pfeiffer, D.U., Munang'andu, H.M., Mulumba, M., Kasanga, C.J., Muma, J.B., Mweene, A.S., 2014. Challenges and economic implications in the control of foot and mouth disease in sub-saharan Africa: lessons from the zambian experience. Veterinary medicine international 2014, 373921.

Smith, G.W. 2013. Overview of Actinobacillos. In The Merck, Allen, D.G., Constable, P.D., eds.

Smith, H.J., 1986. Transmission of Sarcoptes scabiei in Swine by Fomites. Can Vet J 27, 252-254.

Smith, T.F., Burgert, E.O., Dowdle, W.R., Noble, G.R., Campbell, R.J., Vanscoy, R.E., 1976. Isolation of Swine Influenza-Virus from Autopsy Lung-Tissue of Man. New Engl J Med 294, 708-710.

Solano-Aguilar, G.I., Pijoan, C., Rapp-Gabrielson, V., Collins, J., Carvalho, L.F., Winkelman, N., 1999. Protective role of maternal antibodies against Haemophilus parasuis infection. American journal of veterinary research 60, 81-87.

Spickler, A. 2005a. Salmonellosis (Nonthyphoidal). In Swine Diseases and Resources. At http://www.cfsph.iastate.edu/DiseaseInfo/factsheets.php (The Center for Food Security and Public Health).

Spickler, A. 2005b. Streptococcosis. In Swine Diseases and Resources. At http://www.cfsph.iastate.edu/DiseaseInfo/factsheets.php. (The Center for Food Security and Public Health).

Spickler, A. 2006. Aujeszky's Disease. In Swine Diseases and Resources. At http://www.cfsph.iastate.edu/DiseaseInfo/factsheets.php. (The Center for Food Security and Public Health).

Spickler, A. 2010. African Swine Fever. In Swine Diseases and Resources. At http://www.cfsph.iastate.edu/DiseaseInfo/factsheets.php. (The Center for Food Security and Public Health).

Spickler, A. 2013. Leptospirosis. In Swine Diseases and Resources. At http://www.cfsph.iastate.edu/DiseaseInfo/factsheets.php

Spickler, A. 2014. Foot and Mouth Disease. In Swine Diseases and Resources. At http://www.cfsph.iastate.edu/DiseaseInfo/factsheets.php. (The Center for Food Security and Public Health).

Staats, J.J., Feder, I., Okwumabua, O., Chengappa, M.M., 1997. Streptococcus suis: past and present. Veterinary research communications 21, 381-407.

Stegeman, A., Elbers, A., de Smit, H., Moser, H., Smak, J., Pluimers, F., 2000. The 1997-1998 epidemic of classical swine fever in the Netherlands. Veterinary microbiology 73, 183-196.

Stevenson, G.W., Hoang, H., Schwartz, K.J., Burrough, E.R., Sun, D., Madson, D., Cooper, V.L., Pillatzki, A., Gauger, P., Schmitt, B.J., Koster, L.G., Killian, M.L., Yoon, K.J., 2013. Emergence of Porcine epidemic diarrhea virus in the United States: clinical signs, lesions, and viral genomic sequences. J Vet Diagn Invest 25, 649-654.

Stewart, T.B., Hale, O.M., 1988. Losses to internal parasites in swine production. Journal of animal science 66, 1548-1554.

Stone, S.S., Hess, W.R., 1973. Effects of some disinfectants on African swine fever virus. Applied microbiology 25, 115-122.

Suarez, D.L., Spackman, E., Senne, D.A., Bulaga, L., Welsch, A.C., Froberg, K., 2003. The effect of various disinfectants on detection of avian influenza virus by real time RT-PCR. Avian Dis 47, 1091-1095.

Sutmoller, P., Barteling, S.S., Olascoaga, R.C., Sumption, K.J., 2003. Control and eradication of foot-and-mouth disease. Virus research 91, 101-144.

Takahashi, T., Sawada, T., Muramatsu, M., Tamura, Y., Fujisawa, T., Benno, Y., Mitsuoka, T., 1987. Serotype, antimicrobial susceptibility, and pathogenicity of Erysipelothrix rhusiopathiae isolates from tonsils of apparently healthy slaughter pigs. J Clin Microbiol 25, 536-539.

Taylor, D.M., 2000. Inactivation of transmissible degenerative encephalopathy agents: A review. Vet J 159, 10-17.

Tellier, R., 2006. Review of aerosol transmission of influenza A virus. Emerging infectious diseases 12, 1657-1662.

Terpstra, F.G., van't Wout, A.B., Schuitemaker, H., van Engelenburg, F.A.C., Dekkers, D.W.C., Verhaar, R., de Korte, D., Verhoeven, A.J., 2008. Potential and limitation of UVC irradiation for the inactivation of pathogens in platelet concentrates. Transfusion 48, 304-313.

Thacker, E., Janke, B., 2008. Swine influenza virus: zoonotic potential and vaccination strategies for the control of avian and swine influenzas. The Journal of infectious diseases 197 Suppl 1, S19-24.

Thacker, E.L., 2004. Diagnosis of Mycoplasma hyopneumoniae. J Swine Health Prod 12, 252-254.

Thacker, E.L., Halbur, P.G., Ross, R.F., Thanawongnuwech, R., Thacker, B.J., 1999. Mycoplasma hyopneumoniae potentiation of porcine reproductive and respiratory syndrome virus-induced pneumonia. J Clin Microbiol 37, 620-627.

Thacker, E.L., Minion, F.C. 2012. Mycoplasmosis. In Diseases of Swine, Zimmerman, J., Karriker, L., Ramirez, A., Schwartz, K.J., Stevenson, G.W., eds. (Hoboken, NJ, USA, Willey-Blackwell).

Thomas, P., Karriker, L., Ramirez, A., Zhang, J., Ellingson, J., Holtkamp, D. 2014. Evaluation of Time and Temperature Sufficient to Inactivate PEDV in Swine Feces on Metal Surfaces. In Proceedings of the 23rd IPVS Congress. (Cancun, Mexico).

Thomas, P.R., Karriker, L.A., Ramirez, A., Zhang, J.Q., Ellingson, J.S., Crawford, K.K., Bates, J.L., Hammen, K.J., Holtkamp, D.J., 2015. Evaluation of time and temperature sufficient to inactivate porcine epidemic diarrhea virus in swine feces on metal surfaces. J Swine Health Prod 23, 84-90.

Thomas, Y., Vogel, G., Wunderli, W., Suter, P., Witschi, M., Koch, D., Tapparel, C., Kaiser, L., 2008. Survival of influenza virus on banknotes. Applied and environmental microbiology 74, 3002-3007.

Thomson, J.R., Bell, N.A., Rafferty, M., 2007. Efficacy of some disinfectant compounds against porcine bacterial pathogens. Pig Journal 60.

Tobias, T.J., Bouma, A., van den Broek, J., van Nes, A., Daemen, A.J.J.M., Wagenaar, J.A., Stegeman, J.A., Klinkenberg, D., 2014. Transmission of Actinobacillus pleuropneumoniae among weaned piglets on endemically infected farms. Preventive veterinary medicine 117, 207-214.

Turner, C., Williams, S.M., 1999. Laboratory-scale inactivation of African swine fever virus and swine vesicular disease virus in pig slurry. Journal of applied microbiology 87, 148-157.

Valdazo-Gonzalez, B., Timina, A., Scherbakov, A., Abdul-Hamid, N.F., Knowles, N.J., King, D.P., 2013. Multiple introductions of serotype O foot-and-mouth disease viruses into East Asia in 2010-2011. Vet Res 44, 76.

van Elsas, J.D., Semenov, A.V., Costa, R., Trevors, J.T., 2011. Survival of Escherichia coli in the environment: fundamental and public health aspects. The ISME journal 5, 173-183.

Vaughn, J.M., Chen, Y.S., Thomas, M.Z., 1986. Inactivation of Human and Simian Rotaviruses by Chlorine. Applied and environmental microbiology 51, 391-394.

vd Burg, W.P., Borgsteede, F.H., 1987. [Effects of various disinfectants on the development and survival possibilities of the pre-parasitic stages of Ostertagia ostertagi, Cooperia oncophora and Ascaris suum]. Tijdschr Diergeneeskd 112, 769-778.

Verreault, D., Letourneau, V., Gendron, L., Masse, D., Gagnon, C.A., Duchaine, C., 2010. Airborne porcine circovirus in Canadian swine confinement buildings. Veterinary microbiology 141, 224-230.

Vogeleer, P., Tremblay, Y.D., Mafu, A.A., Jacques, M., Harel, J., 2014. Life on the outside: role of biofilms in environmental persistence of Shiga-toxin producing Escherichia coli. Frontiers in microbiology 5, 317.

Wang, Q., Chang, B.J., Riley, T.V., 2010. Erysipelothrix rhusiopathiae. Veterinary microbiology 140, 405-417.

Webby, R.J., Swenson, S.L., Krauss, S.L., Gerrish, P.J., Goyal, S.M., Webster, R.G., 2000. Evolution of swine H3N2 influenza viruses in the United States. Journal of virology 74, 8243-8251.

Webby, R.J., Webster, R.G., 2001. Emergence of influenza A viruses. Philos T R Soc B 356, 1817-1828.

Weesendorp, E., Landman, W.J.M., Stegeman, A., Loeffen, W.L.A., 2008. Detection and quantification of classical swine fever virus in air samples originating from infected pigs and experimentally produced aerosols. Veterinary microbiology 127, 50-62.

Welch, J., Bienek, C., Gomperts, E., Simmonds, P., 2006. Resistance of porcine circovirus and chicken anemia virus to virus inactivation procedures used for blood products. Transfusion 46, 1951-1958.

Wieler, L.H., Ilieff, A., Herbst, W., Bauer, C., Vieler, E., Bauerfeind, R., Failing, K., Klos, H., Wengert, D., Baljer, G., Zahner, H., 2001. Prevalence of enteropathogens in suckling and weaned piglets with diarrhoea in southern Germany. Journal of veterinary medicine. B, Infectious diseases and veterinary public health 48, 151-159.

Wilkins, W., Rajic, A., Waldner, C., McFall, M., Chow, E., Muckle, A., Rosengren, L., 2010. Distribution of Salmonella serovars in breeding, nursery, and grow-to-finish pigs, and risk factors for shedding in ten farrow-to-finish swine farms in Alberta and Saskatchewan. Canadian journal of veterinary research = Revue canadienne de recherche veterinaire 74, 81-90.

Will, L.A., Paul, P.S., Proescholdt, T.A., Aktar, S.N., Flaming, K.P., Janke, B.H., Sacks, J., Lyoo, Y.S., Hill, H.T., Hoffman, L.J., Wu, L.L., 1994. Evaluation of Rotavirus Infection and Diarrhea in Iowa Commercial Pigs Based on an Epidemiologic-Study of a Population Represented by Diagnostic Laboratory Cases. J Vet Diagn Invest 6, 416-422.

Wills, R.W., 2000. Diarrhea in growing-finishing swine. The Veterinary clinics of North America. Food animal practice 16, 135-161.

Wittmann, G., 1985. Aujeszky's disease: factors important for epizootiology and control. Rev. sci. tech. Off. int. Epiz. 4 5-20.

Yaeger, M.J., 1995. Actinobacillus suis se˜ticemia: An emerging disease in high-health herds. Swine Health Prod 3, 209-210.

Yaeger, M.J., 1996. An outbreak of Actinobacillus suis septicemia in grow/finish pigs. J Vet Diagn Invest 8, 381-383.

Yang, J.S., Song, D.S., Kim, S.Y., Lyoo, K.S., Park, B.K., 2003. Detection of porcine circovirus type 2 in feces of pigs with or without enteric disease by polymerase chain reaction. J Vet Diagn Invest 15, 369-373.

Yuan, L.G., Stevenson, W., Saif, L.J. 2006. Rotavirus and reovirus., In: B. E. Straw, J. J.Zimmerman, S. D'Allaire, Taylor, D.J. (Eds.) Diseases of Swine, 9th ed. Blackwell Publishing, Ames, Iowa, 435–454.

Zimmerman, J.J., Benfield, D.A., Dee, S.A., Murtaugh, M.P., Stadejek, T., Stevenson, G.W., Torremorell, M. 2012. PRRS. In Diseases of Swine (10th Edition). , Zimmerman, J., Karriker, L., Ramirez, A., Schwartz, K.J., Stevenson, G.W., eds. (Hoboken, NJ, USA, Willey-Blackwell).

Zou, S., Guo, J., Gao, R., Dong, L., Zhou, J., Zhang, Y., Dong, J., Bo, H., Qin, K., Shu, Y., 2013. Inactivation of the novel avian influenza A (H7N9) virus under physical conditions or chemical agents treatment. Virology journal 10, 289.