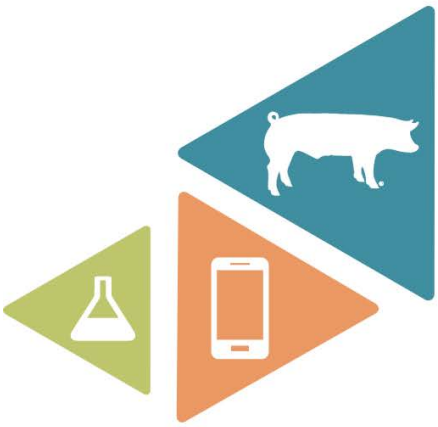

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Porcine pleuropneumonia (PPP) caused by Actinobacillus pleuropneumoniae (App)

Updated September 2021

Introduction

Porcine pleuropneumonia (PPP) is an infectious respiratory disease of swine caused by the bacteria *Actinobacillus pleuropneumoniae* (APP). The disease occurs throughout the world and results in significant economic losses due to mortality, growth retardation, veterinary costs and slaughter condemnations. It is rather well controlled in North America, but PPP remains a major concern in Latin-American, Australasian and most European countries.

Objectives

- Explain how APP serotype differences can result in a wide range of clinical signs
- Provide additional factors that may influence APP infections
- Describe the transmission, lung lesions and diagnosis of APP
- Discuss various treatment, control and prevention strategies

Cause and Distribution

APP has different serotypes and not all are equally dangerous.
APP is a bacterial organism that can be grown via laboratory culture. After culturing the organism, a single colony or isolate can be further characterized. APP isolates belong to one of 18 variations of the bacteria, called serotypes. Serotypes are determined by the isolated bacteria's capsule (protector shields that surrounds the bacteria). The bacterium has another important structural component called lipopolysaccharides (LPS). Some serotypes have the same LPS components. For example, serotypes 1, 9 and 11, 3, 6, 8 and 15 and 4 and 7, share similar LPS components. Since the LPS is used in some blood tests, cross-reactions may occur due to these serotype similarities. Composition of the capsule and LPS of serotypes 16, 17 and 18 (recently described) are still poorly known.

The virulence (ability to cause disease) of APP isolates varies greatly and is usually related to the serotype involved. This variation results in a large spectrum of clinical situations from absence of clinical signs to acute and chronic infections. The basis for APP virulence is not completely understood. To cause disease, the bacteria secrete toxins that lead to severe lung damage and destruction of the host's defense cells. These toxins are called Apx and there are four types: ApxI, ApxII, ApxIII and ApxIV. The first three are extremely harmful to pigs and some serotypes produce combinations of these three toxins. The ApxIV toxin is produced by all serotypes and is used for diagnostic testing. Presence of antibodies (immune response) against this toxin in the blood of an animal means that it has been infected by APP. The role of ApxIV in causing disease is still unknown.

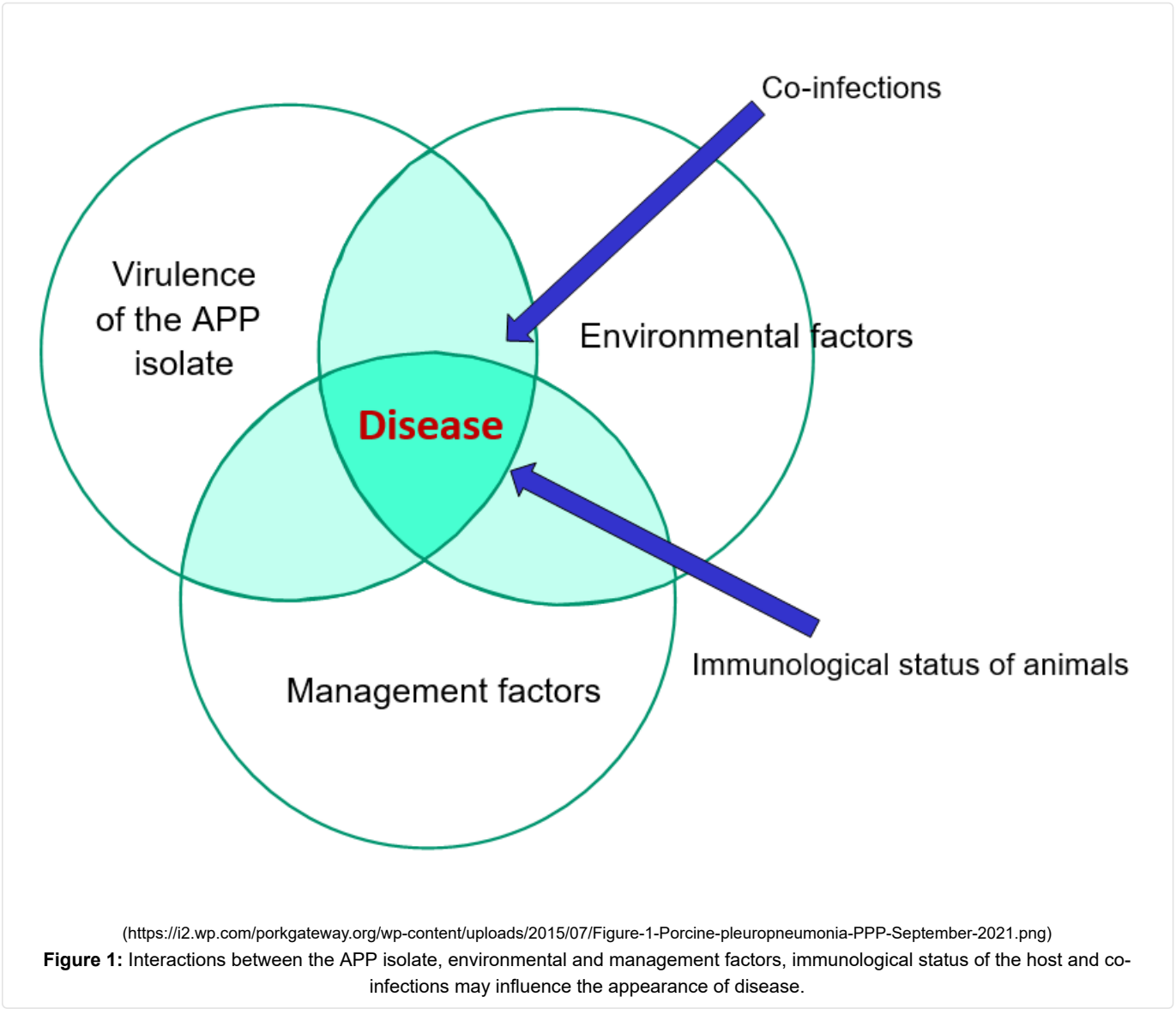
In North America, serotypes 1, 5 and 7 are the most virulent. However, serotype 1 has been nearly completely eradicated during recent years. Serotypes 12, 6, 8, 13, 15 and 17 are considered to have intermediate virulence, causing clinical disease mainly in high health status herds or when other cofactors are involved. Serotypes 2, 3, 4 and 10 are rarely recovered from diseased pigs and serotypes 9, 11, 14, 16 and 18 have never been reported in North America.

Transmission of APP between herds occurs mainly through the introduction of healthy appearing animals carrying the bacteria. The main route of spread is by direct contact from pig to pig or by air droplets transmitted over short distances. In sudden outbreaks, sick animals will secrete large amounts of pathogenic bacteria; therefore, APP also has the potential to be transmitted several hundred meters by air. Other animal species have not been shown to carry or spread the bacteria to pigs. APP survival in the environment is usually of short duration. However, when protected with organic matter, such as feces, it can survive for several days. This may explain why fomites and people are occasionally involved in the transmission of APP.

The cycle of infection usually starts with chronically infected sows who transmit the bacteria to their offspring. Only a few piglets become infected from their mother during lactation. Antibodies provided by the mother through the milk may reduce and/or prevent the bacterial colonization of piglets. The later the age of weaning, the greater the chance piglets could be infected. Infected piglets spread the bacteria to other pigs after weaning as immunity from their mother declines. The presence of antibodies from the mother may last 2 to 10 weeks, depending on their initial level of antibodies in the sow and the amount of colostrum consumed by the pigs.

Presence of APP in a herd does not automatically mean presence of clinical signs and death.
Most herds are infected by one or more low or mildly virulent serotype(s) without clinical signs or clear lung lesions at slaughter. Some herds may even be infected with virulent serotypes without causing death, clinical signs or even lung lesions. However, many of these herds will experience occasional clinical outbreaks when cofactors help APP cause disease. Examples of these cofactors are (Figure 1):

- Co-infections with *Mycoplasma hyopneumoniae*, swine influenza virus, and/or virulent *Pasteurella multocida*. A possible synergism between APP and the Porcine reproductive and respiratory syndrome virus (PRRSV) has yet to be scientifically confirmed
- Environmental stressors such as overcrowding, poor ventilation, rapid temperature changes and/or high humidity
- Management-related factors such as mixing pigs from different sources, continuous flow production and/or weaning piglets >21 days of age
- Immunity status of the animals i.e. presence or absence of antibodies (vaccinated animals would have more protection, while animals from herds free of all APP serotypes would be more susceptible to infection)

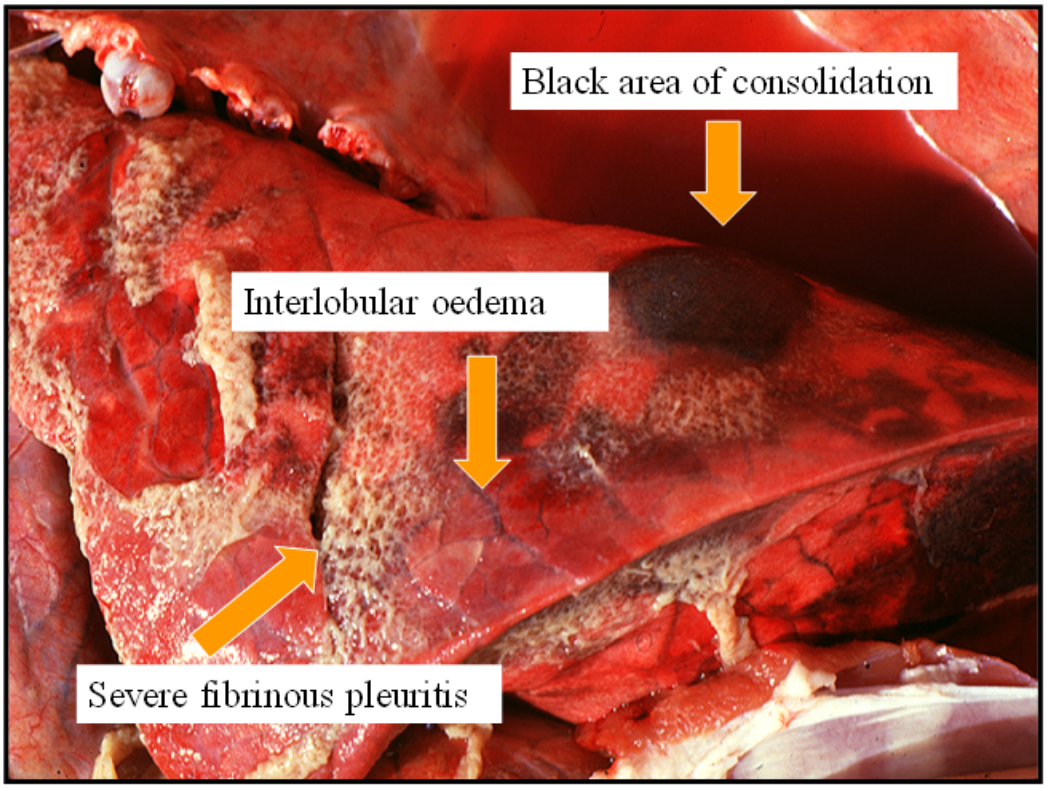


(https://i2.wp.com/porkgateway.org/wp-content/uploads/2015/07/Figure-1-Porcine-pleuropneumonia-PPP-September-2021.png)
Figure 1: Interactions between the APP isolate, environmental and management factors, immunological status of the host and co-infections may influence the appearance of disease.

Clinical Signs and Lesions

In the peracute or extremely sudden form, it is common to find one or more animals dead without observing clinical signs and with a characteristic foamy blood tinged nasal discharge. In the acute or sudden form, usually many pigs are sick with elevated body temperatures of 105–106˚F (40.5–41˚C). Animals are depressed and reluctant to rise, eat and/or drink. They may show difficulty breathing, coughing, blood from the nose, and sometimes open mouth breathing. The course of the disease differs from animal to animal, depending on the extent of the lung lesions and the time of initial treatment. In the subacute or not sudden form, animals have mild to no fever and cough with variable (usually low) intensity and duration; these signs are not easily distinguished from respiratory disease caused by other pathogens. Appetite may be reduced or intermittent, which is hard to appreciate where ad-lib feeding systems are in use, and this may contribute to decreased rate of weight gain. In the chronic form, there is little to no fever, and a spontaneous or irregular cough of varying intensity develops. Feed consumption may be reduced, and affected animals may not tolerate exercise. When forced to move they remain behind the group and struggle minimally when restrained. Mortality is seldom a feature of this form of infection, although an increase in cull pigs many be seen.

Lung inflammation (pneumonia) may be in one or both lungs, with major involvement of the top of the lung(s) near the diaphragm. There are usually areas of lung consolidation that are firm to the touch and are colored dark red to black (necrotic and easy to break apart), the lungs will also accumulate fluid (interlobular edema), and fibrin deposits will be present on the lung surface (fibrinous pleuritis) (Image 1). The chest cavity usually contains a blood-tinged fluid. In healthy appearing animals with chronic disease, the dark red or black color of the early lung lesion becomes lighter in color and remains firm in the worst affected areas of the lung. In many cases, the lung lesion resolves, and only an area of thickened scar tissue (fibrous adhesion) remains. A high occurrence of membrane inflammation (pleuritis) at slaughter is strongly suggestive of PPP. These lesions may result in slaughter condemnations or trimming of affected tissue.



(https://i1.wp.com/porkgateway.org/wp-content/uploads/2015/07/Image-1-Porcine-pleuropeumonia-September-2021.png)
Image 1: Typical lung lesions in pigs with acute APP infection: dark areas of firm consolidation, fluid accumulation (interlobular edema or oedema), and fibrin deposits on the lung surface (fibrinous pleuritis). Source: Dr. Robert Desrosiers.

Diagnosis

Even if lung lesions are suggestive of PPP, it is recommended to confirm the diagnosis through culture of the organism. Bacterial colony growth from pigs with sudden pneumonia is usually easy, except if the animal has been recently treated with antibiotics. Identification of the serotype is recommended for surveillance when vaccination is being considered. Serotyping should be conducted in appropriate diagnostic laboratories to prevent misidentification. Antimicrobial susceptibility testing is also available to support veterinarian's antibiotic selection.

Chronic PPP infections are more difficult to diagnose since it is usually hard to grow APP from chronic lung lesions, such as those observed at slaughter. Recently, a new bacterium (*Glaesserella australis*) has been isolated in Australia and France from lung lesions similar to those caused by APP. Detection of APP from tonsils in healthy animals carrying the bacteria is even more difficult and should be attempted only in certain cases by highly experienced diagnostic laboratories.

Detection of antibodies in blood samples is the most powerful tool for the diagnosis of later staged infections; presence of antibodies shows that animals have been or still are infected with APP. Many blood tests have been developed, but only two major categories of tests are currently used: a) tests that will identify the serotype/serogroup and b) a test that is specific for APP (all serotypes). The most useful tests able to identify serotypes/serogroups are based on the LPS and they will give a clear answer of which serotypes/serogroups are present (remember that some serotypes are more virulent than others). These tests can identify serogroups/serotypes as follows: 1, 9 and 11 (serotypes 9 and 11 are absent in the U.S.); 2; 3, 6 and 8; 4 and 7; 5 and 10; and 12. Commercial kits based on these antigens are available and used in several diagnostic laboratories. The second type of test, which is also commercially available, detects antibodies against the ApxIV toxin, which is produced by all isolates of APP. The test only detects APP antibodies; it cannot differentiate serotypes. Moreover, field data suggests that it is less likely to identify animals with APP than LPS-based tests. The test may be useful for the routine surveillance of herds believed to be free of all serotypes. However, its use in typical herds is questionable. Indeed, most herds are infected with one or more APP serotype(s) of low to mild virulence and many positive results are expected with this test. Interestingly, in an infected herd, the percentage or prevalence of infected animals depends on the serotype. Indeed, some serotypes are highly contagious, but poorly virulent and will not cause clinical disease (e.g., serotypes 6, 8, 12 and 15) whereas the opposite is true for others which infect less animals, but cause disease (e.g., serotypes 1 and 5). This should be taken into consideration when selecting pigs for blood testing.

Control

Antibiotic treatment is effective in clinically affected animals only in the initial phase of disease. The antibiotic selected should be based on research evidence, veterinary experience, clinical response and laboratory antimicrobial susceptibility testing. APP isolates are naturally resistant or may become resistant to certain antibiotics. Antimicrobials should be given by injection as affected animals often do not eat or drink normally. The success of treatment depends mainly on prompt treatment following the early detection of clinical signs. Medicated water or feed may be used to treat affected groups of pigs if food and water intake have not decreased significantly.

Feed and water medication can also be used in anticipation of acute outbreaks if the disease is recurrent. However, other measures should be implemented to control the disease and reduce the use of antibiotics and the antimicrobial susceptibility of the organism should be monitored as resistance may develop. Strategic medication should be targeted at periods of high risk, which can be identified through clinical examinations, post-mortem examinations, and blood sampling. Antibiotic treatments alone may control clinical signs but will not eliminate the bacteria.

Prevention

When stocking a new farm, it is strongly recommended to choose breeding stock free of all or at least the most virulent APP serotypes. This requires that the supplier has implemented a health program that includes regular blood testing and necropsies, as well as an effective biosecurity program. Open communication between the veterinarians of the supplier and the buyer is also essential to ensure that all health requirements are addressed. By contrast, introduction of naïve animals (free of all APP serotypes) into an infected herd is also dangerous and measures such as vaccination should be considered and applied to avoid clinical disease.

In herds facing clinical signs, the priority must be to control economic losses. Vaccination may be effective to reduce disease, death, antibiotic treatments, carcass condemnations and improve overall growth performance. The decision to vaccinate should be carefully evaluated. It is recommended that animals should not receive the primary vaccination before 8 weeks of age to avoid interference with antibodies from the mother. Gilts and sows can be vaccinated during pregnancy to improve passive immunity in piglets, although higher levels of antibodies may increase the risk of interference with piglet vaccination. Replacement animals can be vaccinated before their introduction into an infected herd.

APP vaccines fall into two main categories: those based on killed organisms (called “bacterins”) and the subunit toxin-based vaccines. All commercial vaccines are currently available in the U.S., along with mechanisms to create autogenous vaccines. Autogenous vaccines are created from cultures of organisms collected from a herd. These bacteria are then used to make a vaccine to be used in the same herd. Under experimental conditions, vaccination with bacterins only protects against the serotype(s) in the vaccine (serotype specific). Proof of autogenous vaccine effectiveness under field conditions is still lacking. Subunit vaccines composed mainly of the three major toxins (ApxI, ApxII and ApxIII) as well as toxin-bacterin combination vaccines also exist. Contrary to bacterins, these vaccines are supposed to provide protection against most serotypes due to the immune response against the toxins. Overall, vaccination of animals does not prevent infection and live bacteria may remain in the tonsils despite high levels of protective antibodies.

Eradication

Eradication of the organism may be attempted in infected herds. Several successful eradication procedures have been reported, but a careful economic evaluation is recommended before starting an eradication program. Depopulation and restocking with pigs from certified APP-free herds is probably the most effective means. This method is usually implemented in commercial herds that are facing severe PPP problems as well as other health issues (e.g., PRRSV, *Mycoplasma hyopneumoniae*, etc.). Nucleus breeding herds must use other methods to preserve their bloodlines. One approach that has succeeded in the past is medicated early weaning (MEW).

Summary

Even if PPP is generally well controlled in North America, it remains a concern for some producers. Losses result from increased mortality, reduced growth, veterinary costs and slaughter condemnations. Knowledge of the disease has greatly improved in the last 20 years and more efficient tools to diagnose and control the infection are now available. New herds should preferably be stocked with pigs from sources certified free of all or at least the most virulent APP serotypes. In infected herds, the disease can be partially prevented by vaccination or controlled by medication. Effective control of infectious (such as *Mycoplasma hyopneumoniae*) and non-infectious cofactors (such as overcrowding) is also extremely important.

Additional Resources

1. Gottschalk, M. 2015. The challenge of detecting herds sub-clinically infected with *Actinobacillus pleuropneumoniae*, Vet. J. 206:30–38.
2. Gottschalk, M., A. Broes. 2019. Actinobacillosis, in: Zimmerman JJ, Karriker LA, Ramirez A, Schwartz KJ, Stevenson GW, Zhang J, eds., Diseases of Swine, 11th ed., Hoboken, NJ: Wiley-Blackwell, pp. 749–766.
3. Loera-Muro A, C. Angulo. 2018. New trends in innovative vaccine development against *Actinobacillus pleuropneumoniae*. Vet. Microbiol. 217:66–75.
4. Sassu, E.L., J.T. Bossé, T.J. Tobias, M. Gottschalk, P.R. Langford PR, I. Hennig-Pauka. 2018. Update on *Actinobacillus pleuropneumoniae*-knowledge, gaps and challenges. Transb. Emerg. Dis. 65 (Suppl. 1):72–90.

Related Resources

(https://porkgateway.org/resource/evaluation-of-the-recrudescence-of-actinobacillus-pleuropneumonia-in-growing-pigs-following-pulmotil-treatments-in-nursery-and-grower/)
Evaluation of the Recrudescence of Actinobacillus pleuropneumonia in Growing Pigs Following Pulmotil Treatments in Nursery and Grower
(https://porkgateway.org/resource/evaluation-of-the-recrudescence-of-actinobacillus-pleuropneumonia-in-growing-pigs-following-pulmotil-treatments-in-nursery-and-grower/)
Purdue University 1999 Swine Research Report. Pulmotil is effective at preventing clinical outbreaks of respiratory disease caused by Actinobacillus pleuropneumoniae (App) and Pasteurella multocida (Pm) when fed at 200-400
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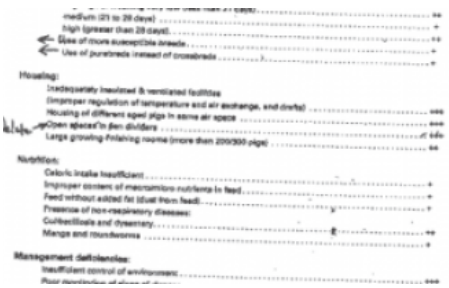

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Mycoplasmal Pneumonia of Swine
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Introduction

The bacteria, *Streptococcus suis* (*S. suis*) causes significant disease in swine operations worldwide. In fact, it is now the most common cause of systemic disease in nursery piglets. Furthermore, there has been a marked increase in *S. suis* cases recently, mainly in herds who have reduced and/or eliminated the use of preventive medications (Poeta Silva, 2021). *S. suis* colonizes the tonsils of most pigs and is capable of causing disease in the brain via meningitis (brain barrier inflammation) and through septicemia (bloodstream infection), thus reaching other organs. While *S. suis* is most often associated with meningitis, other manifestations include arthritis, polyserositis (widespread membrane inflammation), endocarditis (heart valve inflammation) and, as a secondary cause, pneumonia (lung inflammation). *S. suis* is also a zoonotic agent able to cause serious disease (mostly meningitis) in people working with pigs or pork-derived products.

Objectives

- *S. suis* may be a primary or secondary cause of disease. There are many strains of *S. suis,* some that are considered commensal or opportunistic while others have the potential to cause severe disease.
- Diagnostic testing and bacterial subtyping are critical to determine which strains are present in a herd and to differentiate from *Glaeserella parasuis* infection
- Describe various factors that influence disease development including co-infections, environmental stress, management variables and immune status.
- *S. suis* disease is not always associated with meningitis. The development of septicemia can affect many organs and can result in sudden death.
- Discuss *S. suis* presence, transmission, treatment, control and prevention strategies

Cause and Distribution

There are many different S. suis serotypes: a good way to identify who is who…
S. suis is a highly varied species, containing 35 variations of the bacteria, called serotypes. Serotypes are determined by the isolated bacteria's capsule (protective shield that surrounds the bacteria). In North America, most strains recovered from diseased pigs belong to serotypes 1 through 8 (including serotype 1/2, which is a mix of serotypes 1 and 2) and 14. Internationally, serotype 2 is described as the most virulent (disease causing) serotype affecting pigs (and rarely, humans). Serotype 9 is also frequently recovered from ill pigs in several countries, although strains from Europe have more virulent potential. Serotyping is an essential procedure done in experienced laboratories to reveal if one or more serotypes are affecting pigs in a herd, which is very common in Canada and the U.S.

Almost 100% of farms (and almost 100% of pigs within farms) are colonized by *S. suis*, mainly in the tonsils ("carrier state" – pigs are healthy, but carry the bacteria). It has been shown that pigs can carry *S. suis* for more than 500 days and medication does not eliminate the carrier state. Most of these strains are not disease causing since they are a part of the normal bacteria in the host, although potentially virulent strains may also be present. Invasion of the host by virulent (disease causing) strains usually occurs through the respiratory tract. Some reports indicate that the intestine may also be a reservoir and, from there, *S. suis* may invade the bloodstream of animals. However, this has not yet been confirmed and, so far, attempts to reproduce disease orally in a research setting have been unsuccessful.

Transmission of *S. suis* between herds occurs mainly through the introduction of healthy carrier animals. The main route of spread is by direct contact from pig to pig or by air droplets transmitted over short distances. *S. suis* has been isolated from many other animal species, although their role in the carry and spread of the bacteria has not been proven. Carcasses of dead pigs has been suggested to be a likely source of infection for other pen mates. Thus, proper disposal of infected carcasses by burning, burial or removal from the premises is recommended. *S. suis* survives 1-2 weeks in water at 39 °F (4 ºC), but only 10 minutes at 140 °F (60 ºC). At typical nursery environmental temperatures of 72–77 °F (22–25 ºC), the organism could survive about 8 days in feces, but less than 24 hours in dust. The bacteria survives in pig carcasses for 6 weeks at 39 °F (4 ºC) and for 12 days at 72–77 °F (22–25 ºC), providing a potential source for spread by birds, rats, mice, dogs or other species (still not confirmed). Commonly used disinfectants can kill *S. suis* in less than 1 minute, even at concentrations less than those recommended by the manufacturers.

The cycle of infection usually starts with the bacteria being naturally present in sows leading to piglet transmission during the (nose-to-nose contact) lactation phase. Maternal antibodies (immune response component) are normally present since most sows possess high amounts of antibodies against *S. suis*. Antibodies provided in the dam's colostrum usually protect piglets against disease during the lactation phase, but do not prevent bacterial colonization. Transmission between piglets also occurs pre and post weaning. The persistence of antibodies from the damn may protect piglets until 3-5 weeks of age, depending on the sow's immunity. Usually a low proportion of pigs are affected by this pathogen pending control measures, including PRRSV co-infections, all in and all out management, and appropriate cleaning and disinfection protocols. Still, outbreaks are common when these measures are not considered, novel strains enter a herd, or due to environmental or management stressors.

S. suis may be a primary or secondary cause of disease

The presence of a disease causing *S. suis* strain in tonsils does not guarantee appearance of clinical signs and, sometimes, clinical signs can be observed in the absence of such strains on the tonsils. On the other hand, some strains are highly virulent and may cause disease when present in a herd. This is still one of the major challenges we face with the diagnosis of *S. suis* infections: how to determine if a given strain that was isolated from a diseased animal is, in fact, really responsible for the health problems observed on a farm.

Many factors can contribute to the development of disease by *S. suis* (Table 1). Among the predisposing factors, Porcine reproductive and respiratory syndrome virus (PRRSV) infections play a major role: *S. suis* is one the most common secondary complications of this virus. Clinical disease caused by *S. suis* significantly increases in PRRSV affected herds. However, the disease is sometimes present in PRRSV-free, high health status farms that have both good hygiene and management practices.

Clinical Signs

S. suis disease outbreaks occur most frequently in 2-6 week old pigs. These pigs are affected because the level of maternal antibodies drastically decreases at weaning. In general, levels of antibodies generated by the piglet will not increase until around 8 weeks of age and will be optimized as an adult. This explains why clinical cases in late nursery, grow finish pigs are not frequent, normally only found as sudden death cases due to endocarditis. Sometimes pigs are found dead without previous clinical signs of disease. However, pigs with *S. suis* meningitis usually go through a progression of loss of appetite, reddening of the skin, fever, depression, loss of balance, lameness, paralysis, paddling (Image 1), shaking and convulsions. Blindness and deafness may also occur. Septicemia and arthritis without meningitis is less noticeable and sometimes will not be recognized. The role of *S. suis* as a cause of pneumonia is not clear: it may contribute to lung disease in the presence of viral co-infections (PRRSV, swine influenza virus), but is rarely the primary cause of lung disease.

(https://i2.wp.com/porkgateway.org/wp-content/uploads/2015/07/Strep-suis-Image-1-12.7.2021.png)
Image 1: Typical paddling of a piglet affected by meningitis caused, in this case, by *S. suis*

Diagnosis

Diagnosis of *S. suis* infections cannot be determined by clinical signs and necropsy (comparable to autopsy) findings alone, since it is difficult to differentiate *S. suis* from similar disease processes (*Glaeserella parasuis*, Shiga toxin-producing *Escherichia coli,* salt intoxication, etc.). Samples from animals that were not treated with antibiotics must be taken and sent to a diagnostic laboratory for complete diagnosis. To prevent the spread of non-virulent *S. suis* strains to organs, when possible, diseased animals should be euthanized just before taking samples. Type of samples to be sent will depend on the clinical signs observed (brain, cerebrospinal fluid, joints, spleen). Lungs should not be sent unless respiratory clinical signs only are present. In cases of septicemia, it is not uncommon to find one serotype that causes the disease (recovered from most tissues) and another non-related one in lungs.

Serotyping is essential to clearly identify if *S. suis* is a primary problem or a secondary problem due to cofactors (Table 1). It is recommended to send samples from at least three pigs at three different time points to the laboratory. After bacterial growth, isolation and serotyping, usually one of the following situations will occur:

1. Repeated identification of a few serotypes, which may indicate that mostly virulent strains are present;
2. Isolation of several different serotypes (most of them once), which may indicate that cofactors are making normally present strains in the tonsils more virulent, leading to disease. Approaches to control theses situations may vary.

Environmental factors	Management factors	Co-infections/cofactors	Susceptibility of the host
↑ Temperature changes	↑ Cross fostering	PRRSV	4-9 weeks of age with low levels of maternal antibodies
↓ Ventilation	Overcrowding	Swine influenza virus[a]	
↑ Humidity	Tail docking, teeth clipping, ear notching	↑ Mycotoxins (?)	
↑ Dust and ammonia	Weighing, vaccinating (stress)	↓ Vitamin E (?)	
Inadequate sanitation	Mixing pigs of different age		
	Poor adaptation to solid feed (?)		

[a] Only confirmed for serotypes 1, 2, 1/2 and 14.
(?): Still to be confirmed

(https://i2.wp.com/porkgateway.org/wp-content/uploads/2015/07/Strep-suis-Table-1-12.7.2021.png)

Table 1: Factors (other than the virulence of the strain) that may influence the appearance of clinical disease due to *S. suis*

More recently, the use of novel sequencing technologies such as whole genome sequencing has been applied to North American strains. Although there are no universal and accepted virulence markers that clearly identify virulent strains, data from genome sequencing may help to improving pig and gilt sourcing and tracking antimicrobial resistance.

There are no recognized methods to detect carrier animals who may have virulent strains. First, there is no clear definition of a “highly virulent” strain. Secondly, since *S. suis* is normally found in the tonsils, it is extremely hard to diagnose which type of strains are present in a given animal/herd. No validated blood test exists.

Treatment and control

Prior to learning the antimicrobial susceptibility of the *S. suis* organism affecting the animals, sick piglets may be treated individually with injectable antibiotics and given supportive care. Most strains are susceptible to penicillin, amoxicillin, ceftiofur, enrofloxacin, florfenicol and ampicillin. In general, most *S. suis* strains are resistant to tetracyclines. Antimicrobial use should be discussed and determined by working with a veterinarian, utilizing available susceptibility data and accounting for local regulations.

Early treatment prevents death and may result in complete recovery. If a pig is down or convulsing, it should be removed from the pen. These pigs are disabled and may be shedding high amounts of bacteria that could infect other pigs in the same pen. Additional treatment approaches may include anti-inflammatory drugs, supportive fluids or electrolytes. Affected pigs should be kept comfortable, warm and propped up on their sternum when possible. Treatments aimed at the rest of the group must also be considered, especially when other pigs become affected or flow history reveals an increased likelihood of *S. suis* outbreak. Alteration of management approaches to help minimize stress is a key factor in the control of this disease. In PRRSV unstable farms, control measures should be considered to reduce secondary clinical disease by *S. suis*.

Prevention

In seriously affected farms, strategic use of antibiotics in the feed, prior to known periods of heightened risk (during the nursery period) has been beneficial, but sometimes results in the shift of clinical cases to occur later in the production cycle. Restrictions in the use of antibiotics brings, among other consequences, an increase of clinical disease due to *S. suis* infections in post-weaned piglets. One of the most important challenges for the swine industry is to control such disease in the absence of medication. Good hygiene, environmental and management practices may help to prevent and reduce clinical cases caused by *S. suis*.

Since the hypothesis of a possible infection through the intestinal route (not yet proven), the use of non-antibiotic feed additives (lauric acid, cinnamon, oregano, rosemary, peppermint, etc.) has become popular. However, there is currently no scientific evidence supporting additive use for the control and prevent *S. suis* infections.

S. suis vaccine use has been a controversial topic. Currently, there is no commercially available vaccine that confers protection against all strains of *S. suis*. Thus, production systems and swine veterinarians have explored the use of autogenous vaccines. Autogenous vaccines are created from cultures of organisms collected from a herd and then used in the same herd. Although *S. suis* is easy to culture, the complexity of strains from diseased pigs within the same farm can difficult selection of the adequate strain(s) to be included in the vaccine. Therefore, isolation and further strain characterization of many strains from diseased animals should be carried out to make the correct choice. Strains isolated from lungs should usually not be included in autogenous vaccines. The efficacy of autogenous vaccines is difficult to evaluate and evidence of vaccine protection under controlled conditions and in the field is mostly unknown. The choice of adjuvant (an agent added to vaccines to improve the body's immune response) is highly important for the success of a *S. suis* vaccine.

Furthermore, there is no clear data on the optimal timing of autogenous vaccine application, and there are minimal scientific studies available. In the field, autogenous vaccines are used mostly in sows, sometimes in piglets or in both. Although levels of antibodies against *S. suis* are already very high in sows, vaccination before farrowing might increase that level. Being less costly than piglet vaccination, it represents an attractive economical alternative. Available results indicate pre-farrowing vaccination of sows results in increased sow antibodies, which are passed on to the piglets through milk and start to wane at 3-5 weeks of age, thus only protecting piglets at the beginning of the high-risk nursery period. Vaccination of young animals (with or without sow vaccination), such as suckling piglets, has the concern of possible interference with maternal antibodies. Vaccination of older piglets (for example, at 3 and 5 weeks of age) may not induce a booster of antibody production early enough to protect piglets in the nursery – there is clearly a problem due to window of vaccination. Oral reports of field autogenous vaccine use are contradictory, being highly effective for some practitioners but not for others. More scientific data is needed before determining the real value of this intervention.

Eradication of *S. suis* so far is not feasible for commercial farms, and medicated early weaning (MEW) is not effective since this bacteria is a early colonizer. Cesarean section (C-section) can be used to obtain pigs free of *S. suis*. However, this option is cost prohibitive. Since there are no validated diagnostic tools to detect carrier animals with virulent strains, and to avoid introduction of *S. suis* in uninfected herds, it is recommended to ask the source of new stock or replacement gilts whether there is a clinical problem in the herd and to understand the disease status of the herd.

Zoonotic *S. suis* (Infection in Humans)

Some types of *S. suis* can also affect humans (mainly serotypes 2 and 14), producing septicemia, meningitis and septic shock. It is an uncommon occurrence in North America with less than 20 cases described to date, all related to swine production. Significantly higher numbers of *S. suis* cases have been described in Europe and it is an endemic disease in humans in some Asian countries. Meat industry workers are at greatest risk. In addition, farmers, veterinarians, food preparers and anyone else who handles uncooked pork or is in contact with live and mainly ill pigs may be

exposed. Butchers and others become infected through cuts and abrasions while handling and cutting pig carcasses. The disease may manifest itself as a “flu-like” disease followed by meningitis. Approximately, 60% of the human cases that recover have permanent hearing loss. The use of gloves and correct disinfection after handling ill animals (with clinical signs that may be associated to *S. suis*) should be implemented.

Summary

S. suis is one of the most important pig diseases, commonly affecting nursery aged pigs. There is a high assortment of serotypes and strains, which makes diagnosis difficult. It can be a primary problem, causing disease alone. However, in many farms, clinical disease is mainly observed in the presence of cofactors such as coinfections with other pathogens (mainly PRRSV), as well as environmental and management problems. Disease can be well treated and even prevented with the use of antibiotics. However, those antibiotics that have high success rates are regulated in the animal industry, given their importance to both human and veterinary medicine. Restrictions in the use of antibiotics brings, among other consequences, an increase in *S. suis*-related cases. As the complexity of *S. suis* distribution increases in swine (multiple strains, multiple serotypes), field reports describing difficulties in disease control and management are common. Autogenous vaccines are largely used, but their impact on the control of the disease should be further evaluated.

References

1. Poeta Silva, AP, Schwartz, K, Arruda, B, Burrough, E, Santos, J, Macedo, N, Sahin, O, Harmon, K, Siepker C, Gauger, P, Sitthicharoenchai, P, Rahe, M, Magstadt, D, Michael, A, Pineyro, P, Derscheid, R, Main, R, Fano, E, Clavijo, M, 2021. Diagnostic trends of five swine endemic bacterial pathogens using data from the Iowa State University Veterinary Diagnostic Laboratory (2010-2019). In proceedings: 52nd Annual Meeting of the American Association of Swine Veterinarians (AASV), Atlanta, Georgia, USA., San Francisco, California, USA (Virtual meeting).

Additional Resources

1. Gottschalk, M., M. Segura M. Streptococcosis. 2015. In: Zimmerman J, et al., eds. Diseases of Swine. 11th ed. Hoboken, NJ: Wiley; 934–950.
2. Gottschalk, M., J. Xu, C. Calzas, M. Segura. *Streptococcus suis*: a new emerging or an old neglected zoonotic pathogen?. Future Microbiol. 5:371-391.
3. Goyette-Desjardins, G., J.P. Auger, J. Xu, M. Segura, M. Gottschalk. 2014. *Streptococcus suis*, an important pig pathogen and emerging zoonotic agent-an update on the worldwide distribution based on serotyping and sequence typing. Emerg Microbes Infect. 3:e45.
4. Segura, M. 2015. *Streptococcus suis* vaccines: candidate antigens and progress. Expert Rev Vaccines. 14:15871608.
5. Vötsch, D., M. Willenborg, Y.B. Weldearegay, P. Valentin-Weigand. 2018. *Streptococcus suis* – The “Two Faces” of a Pathobiont in the Porcine Respiratory Tract. Front Microbiol.;9:480.

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