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Swine Health: Respiratory Diseases of Swine

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Joe Munroe, Farm Quarterly

SWINE HEALTH



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RESPIRATORY DISEASES OF SWINE

James H. Bailey

Chronic pneumonia is said to be the most costly disease of swine in the world. Pneumonia causes considerable death loss in swine of all ages. It also causes poor performance, slow gains, and poor feed utilization in chronically infected animals. This condition is the number one problem of swine production today. Modern confinement rearing also contributes to the problem. Large numbers of hogs living in close proximity provide ideal conditions for the spread of infectious agents from one to another. Poorly managed swine facilities, with less than adequate ventilation "add fuel to the fire."

At the present time there is no known specific treatment for many of these respiratory diseases. We must rely on prevention rather than on treatment. Diagnosis plays a very important role in these conditions because many of them resemble each other. Your veterinarian should be consulted, and if necessary he may enlist the aid of a diagnostic laboratory.

There are many interactions between diseases. For example, swine that have atrophic rhinitis are more susceptible to pneumonia. The filtering capacity of the nasal passage is reduced due to damage to the turbinate bones. The presence of lung worms or migrating ascarid larvae can also predispose lungs to more severe infections.

This fact sheet touches briefly on some of the most common respiratory problems encountered in swine production. This list is by no means complete.

SWINE ENZOOTIC PNEUMONIA

Until recently, swine enzootic pneumonia (SEP) was known as Virus Pig Pneumonia. Researchers in the United States as well as in England, determined that this infection is not due to a virus as had been previously suggested but is caused by *Mycoplasma hyopneumoniae*.

Swine enzootic pneumonia is a chronic pneumonia with a high percentage of the herd involved but with relatively low death losses. The first signs of the disease are usually shown in pigs between 3 and 10 weeks of age. The incubation period is from 10 to 16 days following exposure. Diarrhea may be evident for 2 or 3 days followed by a dry cough. Suckling pigs may go through a period of sneezing that is not often seen in older pigs. These pigs show a characteristic cough particularly when they are roused in the morning. The cough may

be brought on by exercise and movement and may persist for 1 to 3 three weeks or it may be present indefinitely. Affected pigs may show no outward signs of lung involvement such as thumping or labored breathing. Pigs may continue to eat but fail to grow normally. Stunting and loss of condition may become evident. Pigs that appear to be fully recovered may relapse when about 4 months old.

Severe respiratory problems and clinical illness occurs when SEP is complicated with migrating ascarid larvae or when lungworms are present.

Spread of Infection

SEP is spread from one pig to another by direct contact or by inhalation of the airborne organism. It may be brought into the herd by addition of infected pigs or carriers which may show no evidence of the infection. Young pigs usually contract SEP directly from their mother. It may be spread when several litters are put together at weaning time.

Incidence of Infection

Surveys throughout the United States have shown SEP to be very widespread and is probably the most common type of pneumonia. The incidence of SEP in lungs has run from 30%-75% in samples gathered at various packing plants. Lesions of SEP may be confused with other types of pneumonia and diagnosis based on gross examination of the lungs may not be entirely accurate.

Diagnosis

This condition must be differentiated from swine influenza. A history of the herd is helpful. SEP is usually a chronic herd problem that may have been present for months or years. The infection may be passed from breeding stock to young pigs which may perpetuate the chronic condition. SEP may follow introduction of new breeding stock into a herd.

Isolation of *Mycoplasma hyopneumoniae* confirms the diagnosis but specialized techniques are necessary and few laboratories are equipped to do this. Microscopic examination of lung tissue sections is helpful to the diagnostician. Various blood tests have been used experimentally but must await further research. Fluorescent antibody studies have been conducted to confirm the presence of *M. hyopneumoniae* but here again, they must be perfected before they can be adapted to everyday laboratory work.

Treatment

M. hyopneumoniae has been shown to be susceptible to chlortetracycline. If chlortetracycline is administered before pigs are exposed to the organism, development of lesions can be prevented with drug levels of 400 grams per ton down to 50 grams per ton. If the drug is started after pigs are infected, only a partial suppression of lesions is seen. It does not eliminate organisms from the lungs and when taken off the drug, pigs develop lesions of the disease from which the organism can be isolated. Treated pigs can still spread the organism to contact pigs.

Control

The organism has been shown to persist up to 66 weeks in infected swine and thus presents a carrier problem which allows transmission from sow to offspring. Obtaining pigs by the SPF method is one way in which the cycle may be broken. SPF swine herds have made possible the repopulation of badly infected herds and provided a practical way to eliminate this costly chronic type of pneumonia.

Presently no vaccines are available to produce an immunity against the infection. Great care must be exercised when bringing new stock into a herd because of the prevalence of this infection and the great transmissibility to swine of all ages.

Management practices which will control stresses on SEP infected swine will be helpful in reducing the effect of the disease. Uncomplicated cases of SEP have less detrimental effects on performance than those cases that progress to more advanced stages due to bacterial complications.

ATROPHIC RHINITIS

Infectious atrophic rhinitis of swine is a transmissible disease that causes atrophy (shrinking) of the nasal turbinates. The turbinates are scroll-like, fine, bony structures in the nasal passages that are covered with the mucous membrane that lines the entire respiratory system. The turbinates are composed of two portions, the upper (dorsal) and lower (ventral) parts. The ventral portion is larger and in turn has two scrolls, the upper and the lower scroll. The turbinates serve several mechanical functions. All of the air that passes through the nasal passage travels over the turbinates where it is warmed and filtered before it reaches the lower portion of the respiratory system.

Atrophy of the turbinates reduces the filtering and tempering action and results in inhalation of particles into the lungs that otherwise would have been removed by normal turbinates.

History

Infectious atrophic rhinitis was recognized in Germany in 1830 and it was reported that affected swine did not fatten as they should. It was reported in the U. S. in 1941 and verified in 1944.

Cause

Researchers have reported that atrophic rhinitis in itself is not due to one single cause. There are many different infectious organisms and other irritants that can produce atrophy of the turbinates. Therefore, the term

infectious atrophic rhinitis is not specific and confusion exists as to the etiologic (causative) factor or factors involved. Various bacteria such as *Pasteurella* sp., *Trichomonas* sp., *Spherophorus necrophorus*, *Mycoplasma* sp. and *Bordetella bronchiseptica* acting singly or in combination have been incriminated at various times.

Recent work indicates that *Bordetella bronchiseptica* is the most likely cause because of the ability to reproduce typical lesions experimentally with this organism.

It is quite possible that rhinitis from several other causes may be involved in the condition that we are now terming "atrophic rhinitis."

Clinical Signs

Sneezing in baby pigs is usually the first sign of this condition. It may occur in pigs as early as one week of age. It should be pointed out, however, that just because baby pigs sneeze is no sure sign that they have infectious atrophic rhinitis. Irritants to the nasal passage such as dust, chemicals, poor ventilation, etc. can produce signs very similar to the early stages of atrophic rhinitis. Sneezing, sniffing, and snorting are attempts by the pig to get rid of an irritating substance. Excess nasal discharge may be evident and tears overflow the tear duct and cause a wet area beneath the eyes. This wet area traps dust and becomes black. Inflammation in the nasal passage may block the opening of the tear duct where it empties into the nasal passage, thus causing tears to back up and overflow.

Small amounts of clear discharge may come from the nose when sneezing. This may be followed by blood when the irritation becomes severe enough to erode blood vessels.

Wrinkles may form over the snout giving the nose a rough, thickened appearance. The nasal cavity may not develop uniformly and some distortion will cause the nose to be twisted to one side. There may be severe atrophy of the turbinates without any external signs of the disease.

Pneumonia is commonly present along with atrophic rhinitis because of the lack of filtration of air by atrophied turbinates. Most researchers agree that it is the secondary pneumonia and not so much the rhinitis that causes reduced gains and swine to do poorly.

Spread

The primary spread is from pig to pig by way of nasal discharges being expelled as an aerosol. Exposure may occur at any time in life, but turbinate atrophy usually occurs in the pigs that are exposed at a few days or weeks of age. Repeated exposure of young pigs in conditions that favor aerosol transmission usually results in a high incidence of severe lesions. Stresses such as dampness, chilling and poor nutrition seem to favor development of the disease.

Control

Several methods that will prevent young pigs from coming in contact with their dam as well as other swine have been used to break the cycle of infection. Probably the best known is the Specific Pathogen Free program. When properly applied and maintained, it apparently provides a satisfactory method of controlling the

spread of infectious atrophic rhinitis and swine enzootic pneumonia as well.

Switzer has found an organism causing AR to be sensitive to several of the sulfonamide compounds. However, recent surveys in Iowa have indicated an increasing number of the organisms are resistant to sulfas. This is causing some apprehension because the use of 100 grams of sulfamethazine per ton of complete feed has been quite effective in reducing the incidence of the disease. This level of sulfamethazine has been cleared by FDA to be used with aureomycin and penicillin in one combination and with tylosin for a second combination.

SWINE INFLUENZA

Swine influenza is an acute, infectious respiratory disease of swine caused by the bacterium *Hemophilus influenzae suis* and the swine influenza virus acting together. It generally appears each autumn with a rapid buildup of cases in October and November. The disease reaches a peak and the number of cases declines in December and January.

The bacterium *H. influenzae suis* can persist in the respiratory tract of both recovered and apparently normal swine. It has been found that the virus survives in the swine lungworm. This parasite has a complicated life cycle with its intermediate host being the common earthworm.

Clinical Signs

Swine influenza is generally a herd illness and single cases in a herd do not occur. The onset is sudden and practically all of the animals in the herd under 1 year of age are affected. Affected pigs go off feed, have a temperature of 105°-106° F. and are tucked up in the flanks and reluctant to move. They have a sharp cough and a peculiar type of abdominal breathing. The illness lasts for only a few days and recovery is very rapid except in cases complicated with pneumonia. During the first 3 or 4 days of illness the pigs appear to be very sick and recovery starts about the fifth day when appetite returns. Recovery is much more rapid than one would have imagined a few days earlier. The death rate in uncomplicated cases of influenza runs from 1% to 4%.

Post mortem examination of these pigs usually reveals a congested throat and windpipe with thick mucus extending into the lungs. The lungs are deep purplish-red in color with a definite line between the affected and the normal portions. The front lobes of the lungs are the most commonly affected. This is also quite characteristic of SEP. In fatal cases the lung contains a more fluid type of discharge and frequently there are some adhesions on the outer surface of the lungs.

Diagnosis

The history of an outbreak where nearly all swine are involved simultaneously with a respiratory infection is very indicative of swine influenza. Examination of individual animals will show high temperatures, prostration and signs of respiratory involvement. The bacterium *H. influenzae suis* can usually be isolated in pure culture from infected lungs.

Treatment

Careful nursing is of the greatest importance. Bedding animals in dry, nondrafty, comfortable quarters is necessary. Dust is detrimental and should be avoided. Sick animals should be handled as little as possible because of the respiratory embarrassment that may be brought on by any exertion. Mild expectorants may be indicated in the drinking water to assist in loosening any thick mucus that is in the respiratory tract.

Immunity

Swine that have recovered from swine influenza are immune to reinfection. It is thought that swine influenza is closely related to human influenza and it may be the same virus that caused the great human outbreak of influenza in 1918, the first year swine influenza was recognized in the U. S. More recent strains of the swine virus differ from the human strain, however.

Control

Due to the seasonal nature of this disease, and because the common earthworm plays an important role in the course of the disease, it is possible to reduce the incidence of swine influenza by preventing contact with earthworms.

Avoidance of stress from a cold, wet environment also plays an important part in prevention because stress seems necessary to trigger an outbreak. No vaccines are available for use against this disease.

BACTERIAL PNEUMONIA

There are several bacterial organisms that have been incriminated in cases of swine pneumonia. Some of these are capable of producing pneumonia by themselves, while others act as secondary invaders in cases of pneumonia already established by other infections. *Pasteurella multocida* and *Streptococcus* sp. are often secondary invaders. A recent survey of swine lungs showed the following:

Principal Bacteria Recovered from Pneumonic and Grossly Normal Lungs

Organisms Recovered	86 Pneumonic Lungs %	15 Grossly Normal Lungs %
<i>Mycoplasma</i> sp.	51.1	6.6
<i>Pasteurella multocida</i>	40.7	0
<i>Streptococcus</i> sp.	40.7	33.3

From this table it is apparent that while normal-appearing lungs may harbor disease producing bacteria, the pneumonic lungs contained a much higher percentage of bacterial infection.

PASTEURELLA PNEUMONIA

This infection rarely occurs as a primary disease but develops following lowered resistance of a pig by other disease processes. It may be a chronic problem or it may become an acute septicemia (blood poisoning). For example, a group of pigs may have swine enzootic pneumonia which lowers the resistance of the lungs and the vitality of the pig. This is the type of pig that would be apt to have complications with pasteurella pneu-

monia. The losses from pasteurellosis vary greatly depending on the virulence of the infecting organism and the conditions and circumstances under which the infection occurs. Good husbandry and nutrition play an important role in the control of this infection.

Occasionally, the virulence of the pasteurella organism may increase during an outbreak of the infection so that it will become potent enough to cause primary pasteurellosis and sudden death.

Clinical Signs

The lungs become filled with fluid and the affected pigs have difficulty breathing and show "thumps." The cough is usually dry and hacking in the early stages and becomes moist later as the disease progresses.

Subacute cases may live for a week or so and then die. In more chronic cases, infected animals live from 3 to 5 weeks with 30% to 40% of them recovering. During this period of time there is great loss of body weight and general weakness. There may be abscesses in the lungs from other bacteria invading the tissues.

In the acute or septicemic form, the onset is very sudden with extreme weakness and death in a high proportion of cases. The body temperature is 105°-107° F. Death results in about 10 to 36 hours. This is often designated as "quick pneumonia."

Often only a small number of pigs in a group are affected. Isolation of the pasteurella organisms by a laboratory is the only way to confirm presence of the organism. The lesions may be mistaken for other types of septicemia (blood poisoning).

Treatment

Treatment in the early stages with sulfonamides or large doses of injectable antibiotics may be of value. However, if the disease has progressed to an irreversible stage, treatment will not be effective under any conditions.

Good care and nursing with dry, draft-free quarters is very important during convalescence.

BORDETELLA PNEUMONIA

Bordetella pneumonia mainly affects young pigs from a few days to four or five weeks of age. The disease rarely affects older pigs but may be carried over from year to year in breeding animals.

Clinical Signs

Affected pigs are depressed, unthrifty and cough severely when exercised. The cough tends to be quite fluid and loose. A very high percentage of young pigs in a group are affected and there can be considerable death loss particularly if care and management are not good.

Diagnosis

The age of animals affected is quite often a key to the type of pneumonia. Bordetella pneumonia is about the only one that will involve pigs as young as a week of age.

The disease is progressive; that is, several stages of pneumonia can be present in the lung at one time. The infection must be differentiated from swine enzootic pneumonia. SEP will not cause as much involvement in pigs under 3 weeks of age.

Treatment

Tetracycline appears to be the most effective drug in treating this condition, however, sulfonamides are apparently effective also. The use of bordetella bacterins on baby pigs may have some merit but the early age at which the pigs are exposed from the dam may limit the effect of the bacterin. It takes 10-14 days to develop immunity following the use of a bacterin.

Control

The fact this organism is spread from dam to offspring makes it particularly difficult to control. This same organism has been incriminated by some to be the cause of infectious atrophic rhinitis. Methods that would control AR may be effective in controlling bordetella pneumonia.

VERMINOUS PNEUMONIA

Swine respiratory problems can be complicated by the presence of lung worms and also by the migrating larvae of the common round worm, *Ascaris suum*. Swine lung worms have their primary site of infection in the lungs. There are three different species of this parasite belonging to the genus *Metastrongylus*. The mature worms may plug the small air passages of the lungs, leading to pneumonia. Lungworms may also carry swine influenza virus and hog cholera virus. The presence of lung worms at the same time as infectious agents such as SEP will cause a much more severe form of pneumonia than either the lung worms or the infectious agent alone.

The migrating ascarid larvae reach the lungs by way of the venous circulation from the liver. They penetrate the lung walls, enter the air space and are coughed up and swallowed. The penetration of the lung walls causes an irritation that aggravates any lung infection that is present at the same time. Researchers claim there is 10 times more pneumonia damage in lungs infected with both SEP and ascarid larvae.

Control of both of these parasites depends on rigid sanitation. In the case of lung worms, the common earth worm acts as an intermediate host and must be present for the life cycle to be completed. Keeping swine on cement should prevent contact with earthworms. Recent experimental work shows tetramisol to be effective in removing lung worms from infected hogs.

Control of ascarids depends on sanitation and keeping away from dirt lots or pasture where ascarid eggs may have been deposited. Some eggs may remain infective for 8-10 years. Pregnant sows and gilts should be dewormed before going into the farrowing house as baby pigs may become infected with ascarid eggs from their mother's droppings as soon as they start to nurse.

This material was prepared by James H. Bailey, D.V.M., extension veterinarian, South Dakota State University at Brookings. It is based on a research review, "Swine Respiratory Diseases and Arthritis," sponsored by the National Pork Producers Council, the South Dakota State University Cooperative Extension Service and the Federal Extension Service. Additional copies of this fact sheet are available from the National Pork Producers Council, 3101 Ingersoll, Des Moines, Iowa 50312. The research review will be available at cost from the Department of Veterinary Science, South Dakota State University, Brookings, South Dakota 57006.
