



pork industry handbook

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Atrophic Rhinitis

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History

Rhinitis is an inflammation of the mucous membranes that line the pig's nose. Atrophic rhinitis (AR) is a transmissible disease of swine that is characterized by both rhinitis and wasting away or lack of growth of the turbinate bones in the nose. The turbinate bones are small, scroll-like, mucous membrane-covered structures in each nostril. They condition the air by warming, moistening and filtering it during the inspiration phase of respiration.

Other forms of rhinitis include necrotic rhinitis (bull nose) and a virus disease, inclusion body rhinitis. These diseases cause inflammation without turbinate atrophy. This discussion will be limited to atrophic rhinitis.

There are extensive reports in the veterinary literature regarding atrophic rhinitis. The oldest of these reports date back to a German scientist's 1831 description of atrophic rhinitis. The disease was first described in the United States and Canada in 1942-43. It had undoubtedly existed on this continent much earlier.

Incidence

Atrophic rhinitis is widespread among the U.S. swine population. Various authors report that 25-75% of all slaughter swine have evidence of atrophic rhinitis. Herd incidence has been estimated at 40-90%.

Cause

A bacterium, *Bordetella bronchiseptica*, has been proved to be a widely distributed causative agent of AR in the U.S. Other bacteria, especially *Pasteurella*, may be involved as secondary invaders that intensify the disease. Irritating gases in the pig's environment and concurrent diseases such as diarrhea or chronic pneumonia have been suggested but not verified as factors causing intensification of atrophic rhinitis.

The problem is complicated by the fact that *B. bronchiseptica* can be carried in respiratory tracts of many mammals such as cats, dogs, mice, rats, and even man.

The following facts explain how *B. bronchiseptica* bacteria survive and infect pigs:

1. In a dry area exposed to the sun, most of the organisms will die in 5-6 weeks.

2. In a cool, shaded moist area, the organisms may survive up to 4 months.

3. All of the common disinfectants and fumigants are effective against the organism if they come in contact with it.

4. The organism lives and multiplies almost exclusively in the respiratory tract but is not confined to the respiratory tract of only swine. Control and eradication plans would be much more successful if *B. bronchiseptica* were exclusively an organism of swine.

5. It has been recovered from the respiratory tracts of a wide range of mammals including man, cats, dogs, rodents, and many others. It may occur with a much lower frequency in the respiratory tract of birds, but at present birds are not considered to have very much potential for spreading the disease.

6. The organism has been recovered from flies and ants. The possible insect spread of the infection must receive additional study.

7. Not all strains of the organism have equal disease-producing ability (virulence) for swine. There are as yet no practical laboratory tests to tell the degree of virulence of an individual strain of the organism for the pig, but it appears that many of the nonswine-origin strains may have low pathogenicity for pigs. Herds infected from nonswine sources frequently require a few years of pig-to-pig passage before turbinate damage becomes apparent. On the other hand some swine herds infected by the introduction of clinically infected pigs will have clinical disease in pigs farrowed within a few weeks of the introduction of the infected pigs.

8. SPF pigs inoculated with *B. bronchiseptica* at 1-11 days of age had advanced turbinate atrophy 3 weeks after inoculation. Comparison with noninfected litter mates

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followed to slaughter, however, showed that infection in this case did not result in the development of a lasting, growth-retarding form of atrophic rhinitis. This emphasizes the important role of secondary invaders and environmental management in the atrophic rhinitis complex.

Spread

Atrophic rhinitis is spread in three common ways.

1. By an infected shedder sow infecting her litter shortly after birth.
2. Through the air in farrowing houses and nurseries.
3. By exposure to nonswine sources such as cats.

Losses

It has been estimated that atrophic rhinitis causes a production loss of 5-10% in the average herd. Losses can be much higher than this when atrophic rhinitis becomes severe and is complicated by poor management. Mortality is low; the losses are mainly in slowed growth and poorer feed efficiency. Some production losses may occur in herds that have minimal clinical signs of the disease.

Clinical Signs

Clinical signs are those of a nasal infection. The signs vary with the strain of bacterium involved, secondary bacterial and viral invaders, health status and age of the pigs involved.

Sneezing is the most common sign. Sniffing, snorting and coughing are usually observed in pigs with atrophic rhinitis. A moist crescent-shaped area on the face below the eye caused by excessive tearing is commonly present.

A clear to yellowish-white discharge from the nostrils is frequently observed. This discharge may contain blood in severe cases. Twisting and/or shortening and thickening of the nose may occur. The shortening of the upper jaw may cause malalignment of the teeth. Pneumonias are common, and occasionally brain involvement may occur.

All ages of swine can become infected with *B. bronchiseptica*, but the most severe lesions require that the pig be infected during the first few weeks of life.

Environmental stresses such as overcrowding and cold, damp, drafty quarters can intensify a rhinitis problem. Other health problems such as diarrhea, pneumonias or parasitic infections also magnify the problem.

It cannot be overemphasized that the severity of atrophic rhinitis is frequently related to the level of management existing in the herd.

Diagnosis

Diagnosis is by clinical signs and by observing the turbinate atrophy during postmortem examination. The turbinates are examined by carefully sawing with a fine-toothed saw across the nose at the level of the second premolar tooth. The first premolar tooth erupts at about 1 year of age; therefore, the first visible jaw tooth in pigs younger than 1 year is the second premolar. Bacterial cultures of the nasal cavity may result in the isolation of *B. bronchiseptica*.

Control

Several methods are used to limit atrophic rhinitis. These are:

1. Cull visibly affected animals on a continual basis—a simple procedure but the least effective.
2. Use the "all-in all-out" system in the farrowing house and in the nursery rooms.
3. Maintain older breeding stock and keep the percentage of replacements to a minimum.

4. Isolate sows and their litters until after weaning. Continue to isolate each litter for 1 month after weaning. Select breeding stock from litters that are free of clinical signs and also react negatively to nasal swabbing tests for bordetella.

5. Caesarean-derive pigs, and rear in isolation—SPF procedure.

Bordetella can survive in the soil for several weeks. Six weeks in the summer and 3 months in the winter are considered to be the minimal "down time" necessary to repopulate a premise that was previously contaminated. It is strongly recommended that repopulation be done during the summer months.

To maintain a bordetella-free herd:

1. Quarantine the herd from exposure to other swine.
2. Use the closed herd concept.
3. Add only boars that have had 3 consecutive negative swabbing tests to bordetella with no positives, or add only SPF laboratory-derived pigs.
4. Control cat and rodent populations.
5. Monitor the herd by nasal swabbing and culturing a portion of each new crop of weaned pigs.

It has been reported that there are cyclic high and low populations of bordetella organisms, even when no treatments are being used.

Treatment

Treatment is based upon drugs that are secreted into the nasal cavity. The two drugs of choice that fit this category are sulfamethazine and sulfathiazole. Because these drugs work by blocking the nutrient intake of bacteria, treatment must take place over a considerable period to be effective. It is usually recommended that a sulfa level of 100 gm. per ton of feed be administered to sows for 3-5 weeks. This is done shortly before farrowing. Pigs receive the same level of sulfa in their feed for a 6-week or longer period starting at preweaning and continuing until the pigs reach 50-75 lb.

An alternative is to treat both the sows and pigs with the water-soluble form of sulfathiazole in the drinking water at the level of 1 lb./1,000 gal. Sows are treated for 3 weeks prior to farrowing until 1 or 2 weeks after farrowing. Baby pigs are offered medicated water from the first day of life until they reach 50-75 lb. Great care must be used to insure that sulfa-medicated feed or water is not fed to market-weight hogs unintentionally by contamination of their feed or water with sulfa-medicated feed or water. Always observe the recommended preslaughter withdrawal time.

When chronic pneumonias are associated with atrophic rhinitis, treatment levels of broad spectrum antibiotics should be added to the ration for 10 days or longer.

Vaccines

Vaccination is a new and useful aid in the control of *B. bronchiseptica* infection. A federally licensed and approved vaccine is now available. The approved procedure for the use of this vaccine is the administration of a dose to the pig at 7 days of age. A second dose is given at 28 days of age. Sows should be vaccinated 4-5 weeks and again 2-3 weeks before farrowing.

Vaccination is a valuable aid in atrophic rhinitis control, but it should be supplemented with the other control procedures previously mentioned.

The local practicing veterinarian is the best resource person in the community to determine which control methods will be the most effective for a particular swine production unit. A veterinarian should also monitor the progress of an atrophic rhinitis control program.