Atrophic Rhinitis

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 but not 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 is an 1830 German 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 prior to that time.

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, is a widely distributed cause of AR in the U.S. Other bacteria, especially Pasteurella, are 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 intensifying atrophic rhinitis.

Transmission of AR 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 or 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, and rodents. 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. Laboratory tests are now available to differentiate toxigenic (toxin-producing) strains of Bordetella and Pasteurella from nontoxigenic strains. Toxigenic Bordetella alone causes mild AR, and toxigenic Pasteurella alone can cause moderate AR. However, the combination of both toxigenic strains is associated with development of severe AR.
8. Baby pigs infected with B. bronchiseptica at an early age may develop advanced turbinate atrophy within three weeks. These turbinates may undergo regeneration and partial restoration of the atrophied structures if secondary invaders such as toxigenic or nontoxigenic Pasteurella multocida, Hemophilus parasuis, or other secondary bacterial invaders or adverse environmental conditions are not present.
Spread
Atrophic rhinitis is spread in three common ways.
1. By an infected sows or gilts infecting her litter shortly after birth.
2. By the air in farrowing houses and nurseries.
3. By exposure to nonswine sources such as cats.

Losses
Atrophic rhinitis causes an estimated production loss of 5-10% in the average herd. Financial losses result from reduced rate of gain and inefficient feed conversion. Losses can be much higher than this when atrophic rhinitis becomes severe and is complicated by coexisting disease, mycoplasmal pneumonia and/or ascitis infestation in particular, or by wet, cold and poorly ventilated facilities, or by dietary inadequacies, or by overcrowding. The more these adverse factors are present the greater the losses. Mortality is low; the losses are mainly in slowly growing and poorer feed efficiency. Some production losses occur even 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. Snuffling, 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 misalignment of the teeth. Pneumonia could occur but are not a common sign of AR.

All ages of swine can become infected with bacteria associated with AR, 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, pneumonia, or parasitic infections also magnify the problem.

It must be emphasized that the severity of losses associated with AR is enhanced by concomitant infections, environmental stresses, inadequate ventilation, and overcrowding.

Diagnosis
Diagnosis is suggested by clinical signs and must be confirmed by observing turbinate atrophy during postmortem examination. The assistance of a diagnostic laboratory is highly desirable, particularly in mild cases of turbinate atrophy. 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 and toxigenic Pasteurella multocida type D or Hemophilus parasuis.

Treatment
Successful treatment of bacteria that cause AR involves more than drugs; it requires a team effort between a veterinarian versed in swine medicine and a producer willing to follow the veterinarian's directions.

Once AR has been diagnosed at a farm, the following elements are needed to treat it effectively:

1. Correction of deficiencies in the environment,
2. Enhancement of herd immunity against bacteria which cause AR, and
3. Initiation and maintenance of antimicrobial therapy.

The importance of improving the environment, when needed, must be emphasized. Temperature, humidity, and ventilation adequacy are paramount. Dust, drafts, wetness, excessive ammonia, and overcrowding must be controlled. Neither vaccines nor drugs can correct deficiencies in the environment.

Veterinary recommendations should be followed regarding vaccination. The types of vaccines needed vary among regions, even among herds in a region, and the vaccines should be tailored to a herd's specific needs.

For many years sulfonamides (sulfas) were the drugs of choice in treating pigs with AR. Unfortunately many strains of Bordetella have become resistant to sulfas and treatment with sulfas alone is not always as effective as it was in the past. Currently sulfas may be successfully used in combination with tetracycline and penicillin. Oxytetracycline has also proven effective.

Control
Control of AR is possible. Methods of control include:

1. Careful monitoring of feed of animals into the herd.
2. Control of all-in/all-out systems in the farrowing house and nursery are useful, but of greater importance is the purchase of boars and replacements free of AR. If you don't have it, don't buy it. The merits of SPF stock should not be underestimated.
3. Medications: The type of drug used, the route of administration and the period during which it is employed will vary. Medications may be administered orally (feed/water) by injection. The pigs' age and the incidence (high versus low) of AR in the herd influence the route of administration.
4. Judicious use of immunizations. Multivalent vaccines and autogenous bacteria may be used, depending on specific needs of a herd. Immunizations are part of but not a substitute for comprehensive control measures.
5. Periodic evaluation of progress. Routine slaughter checks, tabulation of feed efficiency and growth rates, and percentage of poor-doers and stunted hogs are all used, singly or in combination to assess the economic impact of control measures.

The bottom line is economic return: Is the control system cost-effective? Monitoring the days it takes to produce a 220-lb. hog, before and after an AR control program, is a common way to determine the return on this investment.

Vaccines
Vaccination is a useful aid in the control of AR. Several licensed interstate and unlicensed intrastate vaccines are now available. The typical procedure for the use of vaccines 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.

Several AR vaccines now available are polyvalent. They include a combination of Bordetella and Pasteurella strains. Some autogenous bacterins are also available.

The swine veterinarian practitioner, in cooperation with capable personnel at a diagnostic laboratory, is the best resource person to determine which control methods will be the most cost effective for a particular pork production unit. A veterinarian can also help monitor the progress of an atrophic rhinitis control program.

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