Atrophic Rhinitis – Pork Industry Handbook
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Atrophic Rhinitis

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Atrophic rhinitis (AR) is a widely prevalent, multifactorial disease of swine characterized by a degeneration and/or failure of growth of the nasal turbinate bones. Clinical signs include sneezing, nasal discharge or bleeding, and distortions of the snout.

The milder, nonprogressive form of AR is caused by a toxin-producing bacterium, *Bordetella bronchiseptica*. The more severe and progressive form is caused by the toxin-producing *Pasteurella multocida* bacterium, alone or in combination with other bacteria such as *Bordetella bronchiseptica*. The severity of the disease may be related to the age of the pig when infected, the dose of infectious organisms, the amount of toxin produced by the bacteria and environmental conditions. Although *B. bronchiseptica* and *P. multocida* have been demonstrated to cause AR, clinical disease cannot be attributed solely to infection with either one or both bacteria. Various environmental, management and husbandry factors contribute to the development of clinical disease. In some herds these bacteria are present, but outward signs of disease are not. In herds harboring *B. bronchiseptica* and *P. multocida*, there are periods from 2 months to 2 years or longer when no clinical evidence of disease exists.

Development of AR Caused by *Bordetella bronchiseptica* Infection

Bordetella bronchiseptica readily colonizes the lining of the respiratory tract. It commonly locates in the nasal passages and the tonsils. The most severe disease occurs in piglets that are infected during the first week of life. Disease is less severe in pigs that are a few weeks older; and by 9 weeks of age, pigs show almost no clinical signs after infection. In some cases, damage from the initial infection persists until slaughter, but in many cases of pure *B. bronchiseptica* infection, damage to the nose begins to heal within 4 weeks after the onset of infection.

Development of AR Caused by *Pasteurella multocida* Infection

Toxin-producing strains of *Pasteurella multocida* have a poor capability for establishing themselves in a healthy nasal cavity. Experimentally, however, *P. multocida* readily colonize a nasal cavity that has been pretreated with chemical irritants or infected with *B. bronchiseptica*. *Pasteurella multocida* also infects tonsils and lungs. Similar to *B. bronchiseptica* infection, pigs infected with *P. multocida* at an earlier age may show more severe clinical signs. However, in contrast to *B. bronchiseptica*, *P. multocida* is capable of damaging the nasal cavity in pigs up to 16 weeks of age, and reversal of damage is less likely. In fact, the toxin produced by *P. multocida* has been shown to affect not only the structure of the nasal passages, but also to damage the liver, kidneys, ends of the long bones and certain components of the blood.

Sources of Infectious Agents

Infection in a herd most likely occurs after the introduction of infected breeding stock or feeder pigs, although other animals such as rodents, dogs and cats also may be carriers. Bacteria can be harbored in the nasal passages or tonsils of apparently healthy pigs.

Bacteria are spread from one pig to another through droplets in expired air. Litters in the farrowing house may be

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infected by the sow, but generally the major spread occurs pig-to-pig after 2 to 3 weeks of age or after weaning.

Bordetella bronchiseptica has been found in most domestic and wild animals; however, the strains of *B. bronchiseptica* isolated from non-swine species have only limited ability to produce disease in pigs. Toxin-producing strains of *P. multocida* have been found in cattle, rabbits, dogs, cats, poultry, sheep, goats and humans; however, the possible role of these other species in the spread of disease has not been determined.

Other Factors that Contribute to Clinical Expression of AR

Severity of infection is closely associated with intensive methods of swine production. The following factors all have been identified as contributing to the level of AR in infected herds: large herd size, additions of pigs from various sources, high proportion of gilt replacements in the breeding herd, large farrowing units, large nurseries, frequent moving and mixing, high stocking density, poor ventilation and temperature control, poor hygiene, continuous flow of pigs, and excessive levels of gases and dust. On occasion it has been possible to control AR solely through improvement of housing and management practices.

At one time it was thought that calcium:phosphorus imbalance led to AR, but this has been discounted. However, an existing imbalance would retard healing of lesions that might otherwise resolve.

Clinical Signs of AR

Clinical signs usually appear at about 4 to 12 weeks of age. Initially there is noticeable sneezing and snuffling and pigs may have a watery or thick mucous discharge from the nose. Tear staining appears as dirty brown streaks below the eyes. Some pigs have nose bleeding, which can be severe enough to be seen on the walls of the pen or on the backs of other pigs. As the condition progresses, damage to the interior bony structure of the nose may appear as a shortening or bending of the snout. Some pigs may have damage to their nasal turbinates without having obvious external signs. Pigs that are severely affected experience a reduction in growth rate and feed efficiency, which is compounded by the performance-depressing effects of poor environment and ventilation.

Diagnosis of Infection

When AR is moderate to severe, a diagnosis can be made clinically, based on the typical signs of disease. However, in milder cases additional testing may be necessary. Samples for bacterial culture are taken from the nasal passages and tonsils of affected pigs to check for toxin-producing strains of *B. bronchiseptica* and *P. multocida*. Specific laboratory tests are conducted to identify the two types of *P. multocida* (A and D) that are most commonly involved in AR. Subsequent testing is done to demonstrate the production of toxin. Frequently, examination of nasal structures is made at slaughter when a number of pigs can be examined to estimate the prevalence and severity of turbinate atrophy in the herd. See PIH-93, *Slaughter Checks--An Aid To Better Herd Health.*

Control of AR

Effective control of AR requires a combination of strategies involving management, environment, medication and vaccination. The main goals of control are to reduce the load of bacteria to which pigs are exposed, to treat pigs that are affected and to improve the environment which maximizes pig resistance and minimizes bacterial spread.

Vaccination. Vaccination of sows with a combination *B. bronchiseptica*/*P. multocida* vaccine helps reduce the prevalence of AR in their offspring, but does not eliminate the condition. For greater efficacy, it is important that the vaccine contain the specific *P. multocida* toxin so that the sow will produce antibodies to protect against the effects of the toxin. In some cases, piglets also should be vaccinated. However, immunity requires 2 weeks to develop, and often piglets are infected before they can develop a protective level of antibody. Proper timing of immunization is necessary to optimize the immune response.

Medication. Medication may be used as a temporary measure to help bring severe cases to a controllable level, but it is not a long-term solution. To reduce shedding of bacteria, sows may be given feed containing sulfonamides or oxytetracycline during the last month of gestation. Nursing piglets are strategically medicated by injections of antibacterials up to 4 times during the first 3 to 4 weeks of life. Typically, oxytetracycline, penicillin/streptomycin, or potentiated sulfonamides (extra-label use requires veterinary/client relationship) are used. The choice of drug should be based on the results of drug sensitivity testing on bacteria recovered from infected pigs. Weaned pigs are sometimes medicated in the drinking water or feed.

Management. Vaccination and medication efforts may fail unless combined with appropriate improvements in housing and management. It is important to employ all-in, all-out movement of pigs, minimize the number of gilts in the herd compared to older sows, reduce the stocking density, maintain strict hygiene, correct ventilation rates, and eliminate chilling drafts. Modifications of medicated early weaning (MEW) and multiple site production help to reduce the prevalence and severity of disease. New animals brought into the herd should originate from herds of known status. Breeding companies and producers should know the AR status of their herds and work together to determine if health levels are comparable.

Elimination of AR

Once a herd is infected, it is impossible to eliminate the causative bacteria. However, good management practices can reduce the prevalence and severity of disease. Freedom from the severe detrimental effects of AR can be achieved through strict adherence to proper vaccination and/or management programs including all-in, all-out pig flow. Pigs free of AR may be produced by SPF (Specific Pathogen Free) techniques or possibly through medicated early weaning. Breeding companies that advertise freedom from AR should be routinely monitoring to verify the absence of AR through periodic nasal cultures and slaughter examinations.