

# Etiology and diagnosis of progressive atrophic rhinitis

Susan E. Turnquist, DVM, PhD

All diseases that result in turbinate atrophy in swine are called atrophic rhinitis (AR); however, not all cases of AR are economically significant. Economic losses are attributed to reduced weight gain and feed efficiency and the cost of treatment and prevention. There are two recognized forms of AR, which include the progressive form caused by toxigenic *Pasteurella multocida* and the nonprogressive form caused by a toxigenic *Bordetella bronchiseptica*. The form caused by *B. bronchiseptica* is not as severe as that caused by *P. multocida*, and the lesion is thought to be reversible.

*Bordetella bronchiseptica* colonizes the ciliated respiratory mucosa, tonsils, and intestines; transmission occurs through direct contact, droplets, or aerosols and the fecal-oral route. Breeding females maintain the infection. Passive antibodies derived from naturally infected sows do not prevent infection but do prevent the turbinate lesions.

*Pasteurella multocida* colonizes the tonsils and requires some unknown factor to aid colonization of the nasal mucosa. It has been shown that one cytotoxin produced by *B. bronchiseptica* induces optimum growth of toxigenic *P. multocida*, and that pigs pretreated with this cytotoxin harbor increased numbers of toxigenic *P. multocida* within the nasal cavity. Breeding females maintain the infection and piglets usually acquire the organism within 1 week of birth. Older pigs may become infected, and previously uninfected herds are usually infected by the addition of a carrier animal or animals. Toxigenic strains have been isolated from humans with tonsillitis, rhinitis, sinusitis, pleuritis, appendicitis, and septicemia, indicating zoonotic potential.

## Factors affecting transmission and maintenance of AR within a herd

Anything that increases close animal contact, such as high population density or overcrowding, will increase the transmission of AR. The duration of contact appears to be critical. Continuity of stocking, reduced or inadequate ventilation, and high levels of dust or noxious gases (such as ammonia) also contribute to the propagation of the disease. Genetics may play a role in the

susceptibility to infection, and nutritional factors (such as calcium : phosphorus imbalance) may influence the severity of disease. Cats and rats can transmit the infection, and the bacteria may also be transferred by equipment. Seasonal influences have been reported, with AR increasing in prevalence and severity in the spring and summer.

## Clinical signs and gross lesions

Clinical signs are rarely observed prior to 3–4 weeks of age. Inclusion body rhinitis (cytomegalovirus) should be considered in clinically affected pigs less than 3 weeks old. Nasal discharge and sneezing are usually the first signs observed in AR. Signs develop further in the nursery- and growing/finishing-aged pigs and consist of sneezing, serous to mucopurulent exudate, and staining of the medial canthus due to decreased lacrimal drainage or increased lacrimation. In severe cases, there may be epistaxis and shortening or lateral deviation of the snout (Figure 4). The snout skin may be wrinkled in cases where shortening of the snout has occurred. It is important to note that only a fraction (5%–15%) of affected pigs will develop clinical signs even when 70%–80% of the herd has lesions at slaughter check.

The ventral scrolls are usually affected first and, in mild cases, may be slightly shrunken to moderately atrophied (Figure 2). A normal snout is shown in Figure 1 for comparison. In severe cases, there may be complete atrophy of ventral and dorsal turbinates (Figure 3). This lesion may or may not be accompanied by septal deviation (Figure 3) and facial deformity (Figure 4).

## Turbinate assessment and sampling

The diagnosis of individual cases is relatively easy for the practitioner and diagnostic lab personnel; however, determining the extent of herd involvement is more challenging.

Examining snouts at slaughter is the only practical method for determining the prevalence and severity of atrophic rhinitis within a herd. A minimum of 20–30 animals should be sampled, and snouts should be sectioned vertically between the first and second upper pre-molar teeth. There are several methods to evaluate snouts. A commonly used grading system uses grades 0–5, with 0 being normal and 5 being most severely affected. Rigidly adhering to an established protocol is not necessary. Most methods are

College of Veterinary Medicine, University of Missouri-Columbia, 1600 East Rollins, Columbia, Missouri, 65205

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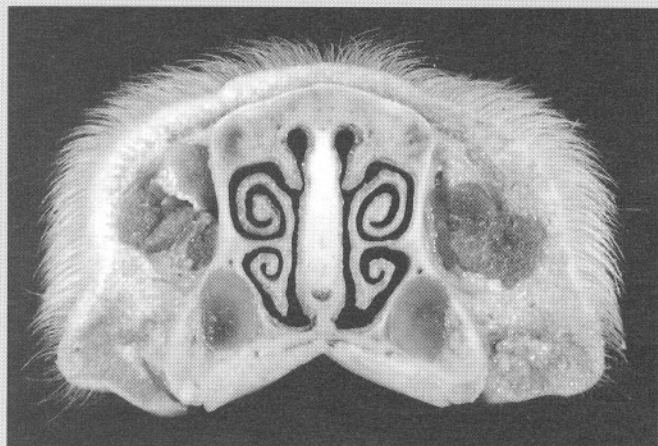
subjective, and the uniformity in evaluation (i.e., have one individual examine all of the animals in a group) is more important than the actual method used. The method can be as simple as assigning grades of severity such as MILD (only ventral scrolls affected), MODERATE (significant atrophy in ventral and dorsal scrolls), and SEVERE (dorsal and ventral scrolls severely atrophied, with or without facial deformity). If quantification is necessary, the distance from the ventral aspect of the ventral scrolls to the floor of the nasal meatus can be measured (the greater the distance, the more severe the atrophy).

The turbinates should be swabbed for bacterial culture prior to being immersed in the hot water tanks during processing. Whole tonsils or tonsil swabs may also be submitted for culture.

If slaughter checks are not feasible, live pigs may be cultured using the following method:

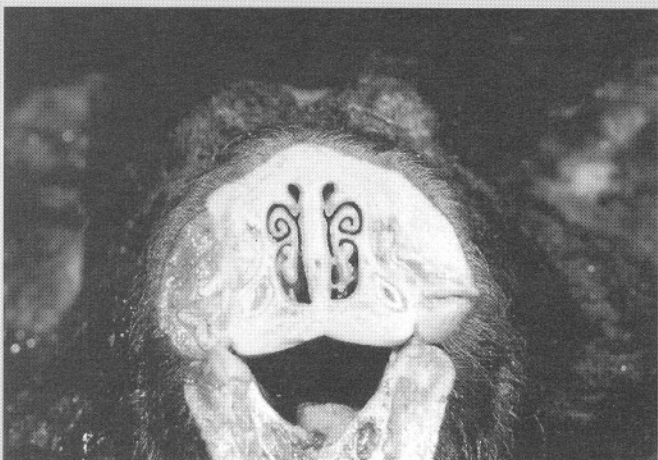
- Restrain the pig and clean the external nares.
- Insert a long, slender, sterile cotton-tipped swab into the nasal cavity and rotate along the mucosa.

**Figure 1**



Cross section of a normal snout.

**Figure 2**



Cross section of snout exhibiting moderate atrophy of the ventral scrolls. Minimal change is evident in the dorsal scrolls.

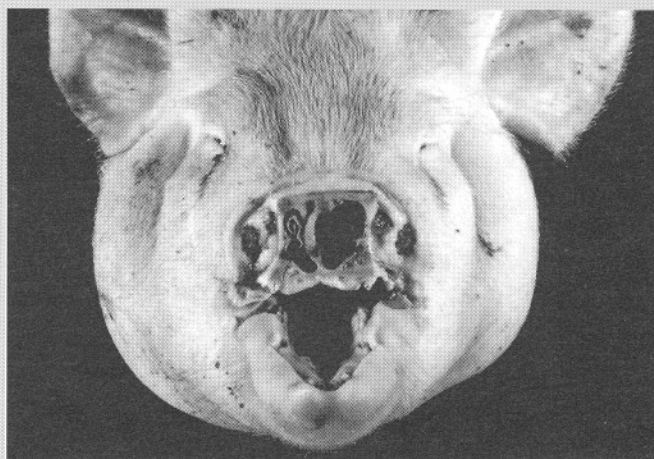
Without visualizing the turbinates, there is no certainty that the swabbed pig is clinically affected or has the typical gross lesions. Radiographic methods have been developed to diagnose AR in live pigs; however, this method is cost prohibitive.

Alternatively, live or dead pigs may be submitted to a diagnostic laboratory for diagnostic evaluation. Some diagnostic laboratories with high porcine caseloads examine snouts on all pigs over 3 weeks of age as a matter of routine; however, they may not routinely perform bacterial culture.

## Submitting samples and interpreting results

Swabs should be placed into a nonnutrient transport medium such as BACTI-SWAB™ Modified Stuart's Transport Medium (REMEL, Lenexa, Kansas), cooled (8°C), and submitted to the diagnostic laboratory as soon as possible. Nutrient media allow

**Figure 3**



Cross section of snout from the pig in Figure 4. Note the deviated septum and complete absence of ventral and dorsal scrolls on the left side and the ventral scroll on the right side.

**Figure 4**



Pig with facial deformity as a result of atrophic rhinitis. Note the lateral deviation of the snout.

overgrowth of commensal organisms and should be avoided if possible. The ability to recover a significant pathogen declines remarkably after 24–48 hours in transit.

*Bordetella bronchiseptica* and *P. multocida* are normal inhabitants of the porcine upper respiratory tract, and isolating these agents does not necessarily indicate a problem. Isolating the toxigenic forms is significant; however, most laboratories are unable to distinguish toxigenic from nontoxigenic strains. ELISA assays and DNA probes have been developed to identify the toxigenic strains of *P. multocida*; however, these tests are not yet routinely performed at most diagnostic laboratories in the United States. Bacterial culture results must be carefully correlated with clinical signs and turbinate lesions. Severe lesions have been observed in some pigs in which it has been impossible to isolate *P. multocida*, indicating that there may be as-yet-unidentified agents capable of causing progressive atrophic rhinitis.

## References

1. Backstrom L. Atrophic rhinitis in swine. *Agri-Practice*. 1992; 13:21-24.

2. Bowerstock TL, Hooper T, Pottenger R. Use of ELISA to detect toxigenic *Pasteurella multocida* in atrophic rhinitis in swine. *J Vet Diagn Invest*. 1992; 4:419-422.
3. Chanter N. Advances in atrophic rhinitis and toxigenic *Pasteurella multocida* research. *Pig News and Info*. 1990; 11:503-506.
4. Chanter N, Goodwin RFW, Rutter JM. Comparison of methods for the sampling and isolation of toxigenic *Pasteurella multocida* from the nasal cavity of pigs. *Res Vet Sci*. 1989; 47:355-358.
5. Chanter N, Magyar T, Rutter JM. Interactions between *Bordetella bronchiseptica* and toxigenic *Pasteurella multocida* in atrophic rhinitis of pigs. *Res Vet Sci*. 1989; 48-53.
6. Cowart R, Boessen CR, Kleibenstein JB. Patterns associated with season and facilities for atrophic rhinitis and pneumonia in slaughter swine. *JAVMA* 1992; 200:190-193.
7. De Jong ME. (Progressive) Atrophic rhinitis. In: Leman A, Straw B, Mengeling W, D'Allaire S, Taylor D, eds. *Diseases of Swine*. 7th ed. 1992:414-435.
8. Scheidt AB, Mayrose VB, Hill MA, Clark LK, Einstein MW, Frantz SF, Runnels LJ, Knox KE. Relationship to growth performance of pneumonia and atrophic rhinitis lesions detected in pigs at slaughter among four seasons. *JAVMA*. 1992; 200:1492-1496.

