The premises should then be cleaned and disinfected before they are re-stocked with healthy swine.

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Atrophic Rhinitis in Swine

RICHARD D. SHUMAN, JOHN S. ANDREWS, AND F. L. EARL

ATROPHIC rhinitis affects mainly the upper respiratory tract of the pig. It causes atrophy or shrinkage of the turbinates and distortion of the nasal septum. The disease process also can involve the bones of the face.

Atrophic rhinitis was first reported in the United States in 1944 by L. P. Doyle, C. R. Donham, and L. M. Hutchings, of Purdue University. Records indicate that it may have been present before then.

It has been encountered throughout the swine-raising areas of the United States and Canada. Rhinitis has been observed in the Scandinavian countries, England, Germany, and France.

It may be identical to a condition known as "sneezing sickness," which has been recognized in Europe for more than 100 years.

This disease caused alarm among swine producers and veterinarians in the United States in the late 1940's and early 1950's and has come to be considered a major disease of swine.

Atrophic rhinitis alone does not seem to kill pigs. But litters farrowed by affected dams tend to have greater losses than litters farrowed by dams that do not have the disease.

F. K. Kristjansson and R. Gwatkins, in Canada, reported that pigs affected with rhinitis were significantly lighter at 56, 84, 112, 140, and 168 days of age than their normal litter mates. Work at the Agricultural Research Center at Beltsville has shown that under Record-of-Performance conditions, the average weight of normal pigs at 56 and 140 days of age exceeded that of affected pigs by 3.9 and 6.4 percent, respectively. The normal pigs also exceeded affected ones in daily gain by 5.2 percent.

R. S. Smiley, of the Division of Animal Industry in Columbus, Ohio, summed up the effect of this disease on farm herds as follows: In some farm herds there is a 30-percent mortality rate after weaning. Some of the affected pigs stop growing after they attain a weight of 40 to 50 pounds. Some of the hogs take 3 to 5 months longer to reach market weight than others. Other animals belonging to the same herd show no apparent ill effects from the disease.

It is possible, however, that the damage attributed to the disease may be caused mainly by secondary conditions, such as pneumonia: Severe upper respiratory disturbance that causes a complete or partial loss of the turbinates when accompanied by a suppurative process could easily result in a type of pneumonia caused by mechanical inhalation of bacteria and debris.

On the other hand, while pneumonia and atrophic rhinitis can be seen in
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In the same pig, the two conditions can also exist separately. In addition, malformation of the jaw, irritation of the nose when eating dry feed, and the lack of aggressiveness on the part of weakened pigs would have a cumulative effect and lead to an inadequate consumption of food. That in turn would be reflected by a deficiency in the normal rate of development.

How atrophic rhinitis is transmitted from animal to animal is not known exactly. The disease has been traced from one specific source of infection to 50 farms.

The disease may possibly be spread through contact with droplets of infective material blown from the noses of affected animals. W. P. Switzer, of Iowa State College, has observed that some of the baby pigs in a pen next to an infected litter became infected although separated from it by a solid tile partition more than 3 feet high.

Baby pigs can be infected experimentally through the intranasal instillation of fresh material from the noses of affected pigs. Experiments at Beltsville, Md., have shown that this type of material will produce the disease in baby pigs after being stored at the temperature of dry ice (—94° F.) for about 7 months. Some evidence also was found that the atrophy-producing factor may survive in the nose of the albino rat for about 3 weeks—an indication that wild rodents may be able to transmit the disease to pigs.

F. W. Schofield, of the Ontario Veterinary College in Canada, reported in 1955 that the domestic cat can be a carrier of the disease.

Not all pigs in constant contact with affected pigs develop the characteristic lesions of the disease. This occurred in experiments at the Agricultural Research Center at Beltsville, when littermate pigs were inoculated with material from the noses of pigs affected with atrophic rhinitis. It also occurred naturally in litter-mate weanlings, on Record-of-Performance tests, which were in direct contact with pen littermates and pigs in adjacent pens that did have atrophic rhinitis. The findings indicate that pigs differ in their susceptibility to the disease or that affected pigs lose their infectiveness. In the same herd, contact with the soil seemed to have been important in the high incidence of atrophic rhinitis in the pigs on pasture (67 percent), compared with the incidence in the pigs kept on concrete floors (27 percent). The following year, however, the disease was distributed almost equally in both pasture-raised pigs and the pigs raised in concrete pens.

Atrophic rhinitis is not transmitted to the offspring at birth or through the milk of the dam. Hand-raised baby pigs have not become affected with the disease after having been fed or injected with pooled bacteriologically sterile swine sera obtained from two affected herds.

The causative agent or agents of atrophic rhinitis also must be considered as unknown. Investigators have presented experimental evidence pointing to the identification of the etiological agents of the disease, but as of 1956 their findings await conformation.

“Sneezing sickness” has been attributed in Europe to a bacterium, Pseudomonas aeruginosa, as well as a particular localization of the cause of lung inflammation or pneumonia in influenza.

Dr. Switzer first found that species of Trichomonas, a microscopic parasitic organism, were present in a greater number of affected animals than in those not affected.

Observations at the Agricultural Research Center indicated that trichomonads may not be essential to turbinate atrophy. Attempts by other research workers to produce the condition experimentally, using pure cultures of trichomonads, were unsuccessful.

R. Gwatkins, L. Dzenis, and J. L. Byrne, of the Animal Disease Research Institute, Canada, and J. L. Flatla and M. Braend, of the Norwegian Veterinary College, Norway, reported that the disease could be transmitted exper-
imentally by a bacterium, Pasteurella multocida.

K. A. McKay and G. R. Carter, of the Ontario Veterinary College, reported that they repeatedly produced rhinitis in young pigs by the nasal instillation of Pasteurella multocida and Spherophorus necrophorus.

Dr. Switzer found that filter-passing agent or agents could produce atrophy of the turbinates.

The first symptoms of atrophic rhinitis may be limited to persistent sneezing, which usually appears about 3 weeks after the farrowing. A clear mucous discharge may come from the nose. As the disease progresses, the discharge sometimes may become mucopurulent. The mucus from the nose is tinged at times with blood. Some mucopurulent debris may be passed from the nose following violent sneezing. Persistent bleeding also may occur following sneezing, vigorous exercise, or injury to the nose.

The irritation in the nose may become so severe that the pig will rub its snout on the ground or against some solid structure. The atrophy of the structures within the snout in some animals leads to visible distortion of the facial bones, and the snout may be turned to either side or upward and have an accordionlike appearance. Affected pigs with crooked noses have been found to be more the exception than the rule, however.

In light-colored hogs, a blackish streak that extends from the inner corner of the eye and bulging of the facial bones also may be circumstantial evidence of atrophic rhinitis. Associated conditions, such as pneumonia, diarrhea, and unthriftiness, may occur in affected herds.

A postmortem examination of the normal animal reveals the turbinates as delicate, scroll-like structures of bone and tissue, which fill up the right and left nasal passages. They are separated by a relatively thick cartilaginous structure, called the nasal septum. The function of the turbinates is to warm the air before it passes into the lungs and assist in filtering out such foreign material as dust and bacteria. The mucous membrane that covers the structures inside the nose normally is pink and not excessively moist.

Considerable gross variation in the appearance of the structures within the nose occurs in animals affected with atrophic rhinitis. The variation can range from a decrease in size or loss of whorls of one or both turbinates, leaving fleshy residual stumps, to the absence of all the turbinates. One side may appear completely normal; the other side may show considerable variation in the extent of shrinkage. The nasal septum may be perpendicular and normal in appearance or somewhat thin. It may also show varying degrees of bowing. Marked bowing of the septum in many instances apparently causes flattening of the turbinates on one side and leads to distortion or compensatory enlargement of the turbinates on the other side. The resulting decrease in size and loss of turbinates with or without a change in the nasal septum brings about a varying increase in size of the nasal passages.

The surface of the membrane lining the structures within the nose may show an excessive amount of mucus. There may be a collection of mucopurulent exudate on the floor of the nasal passages. Occasionally one passage will be completely plugged with a mass of dirty, cheeselike material.

The postmortem findings usually are quite characteristic. Some individuals, however, will present changes that may be normal defects or are not differentiated enough to permit reaching a definite conclusion as to whether or not the animal is affected.

The diagnosis of atrophic rhinitis may not appear to be difficult. When it is not suspected, however, the disease can be present in a herd for a year or two before it is recognized, because the various symptoms can easily be attributed to other causes. The sneezing may be thought to be due to in-
halation of dust or particles of dry feed. The coughing could be considered as caused by the migration of larvae of intestinal worms. The bleeding could be considered insignificant in the mistaken belief that it may have been due to a ruptured blood vessel following injury or extreme exercise. A crooked nose could be thought of as having been due to injury, "bull nose" (or necrotic rhinitis), or the influence of a breed characteristic.

A postmortem examination, however, conducted on a few individuals that showed symptoms will confirm a herd diagnosis.

In most instances, to see within the nose, it is sufficient to make a cross section with a saw midway between the eyes and the end of the snout. For more detailed observations, the snout and face can be divided lengthwise down the midline and the nasal septum removed. This will reveal the nasal cavities in their entirety.

Although a herd diagnosis of atrophic rhinitis can be reached, the number of individual animals affected can only be approximated on the basis of clinical findings. In order to arrive at an individual diagnosis, a rhinoscopic examination may be conducted. That is done with the aid of an otoscope, a lighted instrument for examining the external ear. The instrument can be used with speculums of various sizes, depending on the size of the pig to be examined. When the attached speculum is inserted in the nostril, the light will reveal the anterior portion of the turbinates and nasal septum. Unfortunately the nasal structures cannot be seen completely or in the most desirable perspective; therefore there may be inaccuracies of diagnosis, particularly when the changes are not far advanced. This method is about 75 percent accurate.

A rhinoscopic examination cannot be conducted unless the animal is restrained properly. Several methods can be used: An anesthetic agent, a castreting trough with a crosspiece at one end, or a chute equipped with an adjustable stanchion or yoke to accommodate pigs of any size. The stanchion or yoke must be designed so that it is somewhat wider at the bottom than at the top to conform to the shape of the neck.

The X-ray can be employed in making a diagnosis on the individual live animal.

No satisfactory treatment has been devised for atrophic rhinitis. Adequate treatment cannot be evaluated until the causes are established. Because it appears doubtful that regeneration of atrophied parts occurs, any treatment must necessarily be directed towards its prevention or arrest in the early stages.

R. Gwatkins, P. J. G. Plummer, J. L. Byrne, and R. V. L. Walker, of the Animal Disease Research Institute, Canada, found experimentally that penicillin and streptomycin inhibited the infective agent or agents when added to the inoculum before being instilled into noses of susceptible pigs.

Gwatkins and Dzenis also found that the early use of streptomycin intranasally will reduce the number of pigs that develop atrophic rhinitis. T. L. Jones, also of the Ontario Veterinary College, reported that intramuscular injections of streptomycin administered three times during the first month after birth reduce the incidence of rhinitis but will not eliminate the disease.

Control measures must proceed along generally recognized principles of disease control.

The most satisfactory method of control is to try to keep the disease out of a herd. Much can be accomplished by learning everything possible about conditions in the herd from which replacements are to be obtained.

A more drastic method is to dispose of the affected herd. Thorough cleaning and disinfection of the buildings and equipment should follow. It would be well to renovate the lots by filling in wallows and by providing
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Hog Cholera

J. P. TORREY

HOG CHOLERA is a 40-million-dollar annual expense to the 4-billion-dollar swine industry in the United States.

It occurs in every State and in parts of every country where hogs are raised, except those—Northern Ireland, Denmark, and Australia, among them—that have undertaken vigorous and continuing eradication measures. Reports are that hog cholera broke out in countries where meat from the United States was shipped for use by American Armed Forces.

We have the means to stamp out hog cholera, would we but use them.

The first authentic report of hog cholera in the United States came from Ohio in 1833. The disease spread rapidly. By 1887 it existed in 35 States. It became unusually prevalent at intervals of about 10 years between 1887 and 1926—1887, 1896, 1913, 1926.