

# ***Actinobacillus suis* septicemia in mature swine: Two outbreaks resembling erysipelas**

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## **Abstract**

In the winter of 1987/88 a previously unrecognized septicemic disease syndrome — actinobacillosis in mature sows and gilts — was diagnosed in two minimal-disease swine herds in southwestern Ontario. In herd 1, 34 sows, 2 boars, 13 feeder pigs, and 30 suckling pigs were affected; 11 sows, 2 feeders, and 18 suckling pigs died. In herd 2, 13 sows and 1 feeder pig were affected; 1 sow and 1 feeder pig died. The disease was manifested by moderate fever (39–40.5°C), round or rhomboid erythematous skin lesions, and inappetence. Sudden deaths without previous clinical signs were frequent. Histologically, coccobacillary thromboemboli in superficial and deep dermal vessels were associated with infarcted dermal and epidermal tissues. The causative organism, *Actinobacillus suis*, was isolated from the affected pigs. Treatment with commonly used antibacterial drugs was effective.

In many respects, the disease resembled acute swine erysipelas and presented diagnostic problems for this reason.

## **Résumé**

### **Septicémie à *Actinobacillus suis* chez le porc mature: deux épidémies ressemblant à l'érysipèle**

Durant l'hiver 1987–1988, un syndrome septicémique d'actinobacillose qui n'avait pas été reconnu auparavant chez des truies matures fut diagnostiqué dans deux élevages de porcs dans le sud-ouest ontarien. Dans l'élevage 1, 34 truies, 2 verrats, 13 porcs à l'engrais et 30 porcelets non-sevrés furent affectés; 11 truies, 2 porcs à l'engrais et 18 porcelets non-sevrés sont morts. Dans l'élevage 2, 13 truies et 1 porc à l'engrais furent atteints; 1 truie et 1 porc à l'engrais sont morts. La maladie se caractérisait par une fièvre modérée (39–40,5°C), des lésions cutanées érythémateuses circulaires ou rhomboïdes et de l'inappétence. On observa fréquemment de la mortalité subite sans l'apparition de signes cliniques. À l'histologie, des thrombo-embolies coccobacillaires furent observées dans les vaisseaux cutanés superficiels et profondément dans le derme et associées à des infarcti dermiques et épidermiques. L'agent isolé des porcs affectés fut *Actinobacillus suis*. Un traitement à l'aide d'antibiotiques usuels fut efficace.

Le syndrome comportait beaucoup d'éléments similaires à l'érysipèle aigu du porc et, par le fait même,

présentait certaines difficultés dans l'établissement d'un diagnostic.

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## **Introduction**

Two species belonging to the genus *Actinobacillus* — *Actinobacillus suis* and *A. equuli* — are considered commensal opportunistic pathogens of both swine and horses (1–7). Occasionally they may assume the role of primary disease-causing agents (1–9). Only *A. suis* is known to have been isolated from diseased pigs in Canada (10–12).

Sporadic outbreaks of porcine actinobacillosis have been reported in several countries, including those of northern Europe (8,13,14), the United Kingdom (4,7,15), the USA (9), and the provinces of Alberta (10) and Ontario (11,12) in Canada. Until recently, the disease has been of minor importance in Canada. However, in the last few years, it has been diagnosed with an increasing frequency (11,12).

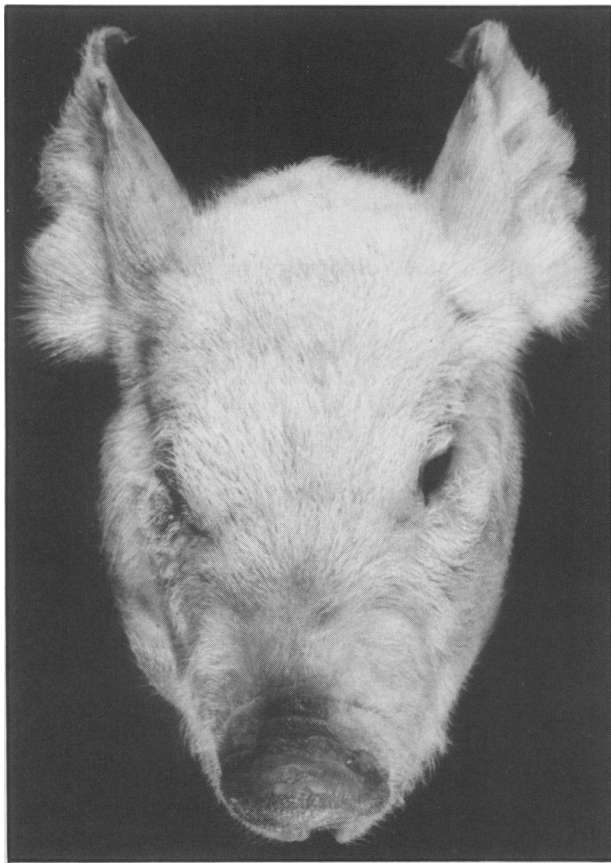
Actinobacillosis in swine has been either a fatal septicemia, or a localized infection causing endocarditis, polyarthritis, or subcutaneous abscesses (8,9,12,13). The disease has been sporadic and self-limiting, affecting mostly young pigs up to three months of age (5,6,8,9,11,14). *Actinobacillus suis* as the cause of disease involving older animals has seldom been reported (5,8,13). The organism has also been found as a secondary invader in systemic viral diseases, such as hog cholera (8,9).

We previously published a brief account of a severe *A. suis* septicemia affecting mature animals and resembling erysipelas in two isolated minimal disease (MD) swine herds in southwestern Ontario during the winter of 1987/88 (10). This is a more detailed description of the outbreaks.

## **Herd backgrounds**

**Herd 1:** This 350-sow herd was established in 1980/81 by the use of cesarean and hysterectomy-derived foundation stock. Since then, it has been maintained in strict isolation, with additions introduced solely by cesarean section. Entry of personnel and visitors has been through a shower. The herd is classified as "excellent" according to the Ontario Swine Herd Health Plan, and is free of demonstrable ectoparasites, atrophic rhinitis, mycoplasmal pneumonia, swine dysentery, *Actinobacillus (Haemophilus) pleuropneumoniae*, and *Haemophilus parasuis*. *Actinobacillus suis* had never been isolated from pigs in this herd. Except for routine vaccination of sows against enteropathogenic *Escherichia coli*, no other vaccine has been used.

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**Figure 1.** The head of a one-week-old piglet which died with *Actinobacillus suis* septicemia. Note the discoloration of the nose and the ears and small erythematous spots elsewhere on the skin.

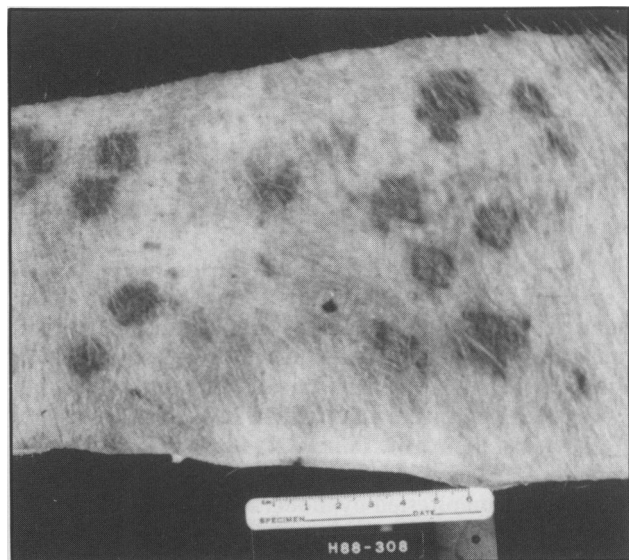
The pigs were housed in three isolated wings of a single building. The west wing housed dry sows and boars, the east wing housed farrowing sows, their litters and weaners, and the north wing housed feeder pigs. The prime purpose of this herd was to provide pigs for research.

**Herd 2:** This MD herd located some 200 km from herd 1 comprised 140 Canborough sows and was established in 1985. In contrast with herd 1, the breeding herd was vaccinated twice yearly between farrowings with a combination vaccine of porcine parvovirus, *Erysipelothrix rhusiopathiae*, *Leptospira canicola*, *L. grippityphosa*, *L. hardjo*, *L. icterohaemorrhagiae*, and *L. pomona* (Farrowsure, Norden Laboratories, Lincoln, Nebraska). As in herd 1, *A. suis* had never been identified on this farm.

### Clinical outbreaks

**Herd 1:** On November 8, 1987, five dry sows exhibited anorexia, listlessness, unwillingness to rise, and mild fevers (39°C–40.5°C). Three sows had round or oval, raised erythematous skin lesions, 1–3 cm in diameter, on their backs and thighs. Two others had small red pimples and diffuse areas, 5–12 cm in length, with white centers and erythematous margins on the udders. One sow aborted at about 30 days of gestation.

As an atypical case of swine erysipelas was suspected, one sow was sent for necropsy and microbiological diagnosis and the others were treated with



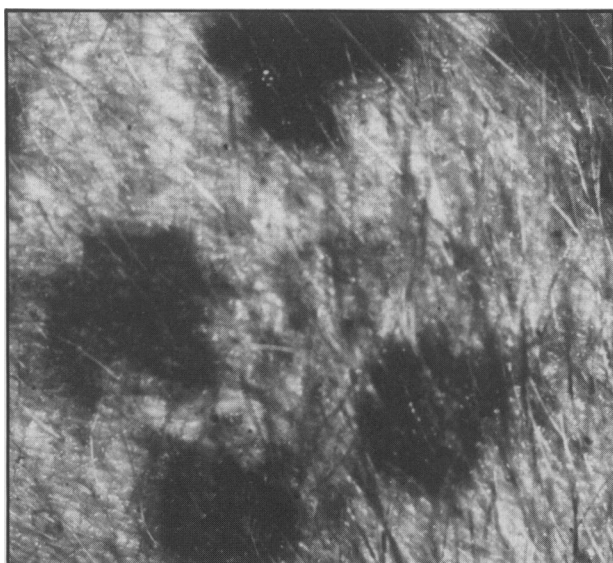
**Figure 2.** Skin lesions of a sow affected with *Actinobacillus suis* septicemia in herd 2. Round, oval, and rhomboid erysipelas-like lesions were present on the same animal.

injectable procaine penicillin,  $1.8\text{--}2.4 \times 10^6$  IU IM. (Ethacillin, rogar/STB Inc., Montreal, Quebec).

During the next 10 days, another 29 sows and 2 young boars became sick. A total of 36 adult animals was affected. Eleven sows died, three aborted, four had polyarthritis, one had metritis and two had mastitis. Further cases were not seen among the mature animals after November 17, 1987. Nevertheless the entire sow herd was medicated with oxytetracycline hydrochloride 550 g/tonne (Terramycin premix 50, Pfizer Canada Inc., Montreal, Quebec) administered in the feed for one week beginning November 17.

On November 17 and 18, three nursing litters in the farrowing wing became sick. During the next two weeks, 18 piglets died; most died suddenly, others died after one or two days of sickness. Most of these piglets had red spots 1 cm or smaller in diameter all over the body, and/or purple discoloration of the skin on the lower parts of the body, the ears, the nose and the extremities; findings that were typical of a septicemia (Figure 1). Finally, between December 9 and 26, 13 three-to-four-month old pigs in the feeder wing had clinical signs similar to those seen in the sows; two died. Major outbreaks have not recurred in the pigery since December 26, 1987, but sporadic deaths due to *A. suis* septicemia have occurred. Furthermore, *A. suis* has been isolated mixed with other bacteria from pigs which died from other causes.

**Herd 2:** On January 7, 1988, one sow and one gilt in the farrowing room became anorectic and developed raised red, square, rhomboidal and irregularly shaped skin lesions along the back, sides, neck and upper portions of all four legs (Figures 2 and 3). Temperatures were normal or slightly elevated (39–40°C). During the next three weeks, 10 sows located in both the farrowing and dry sow rooms exhibited similar clinical signs. Each animal was treated with and responded rapidly to intramuscular injection of benzathine-procaine penicillin G 15–20 mL ( $2.25\text{--}3.0 \text{ IU} \times 10^6$  IM (Penlong XL, rogar/STB, Montreal, Quebec). On January 29,



**Figure 3.** A close-up of a diamond-shaped skin lesion in a sow affected with *Actinobacillus suis* septicemia in herd 2. It is clinically indistinguishable from a lesion of erysipelas.

1988, one sow and one weaned pig died suddenly without being treated.

As this herd had been routinely vaccinated against swine erysipelas, a break in the vaccinal protection was first suspected. Further cases have not been reported from this herd.

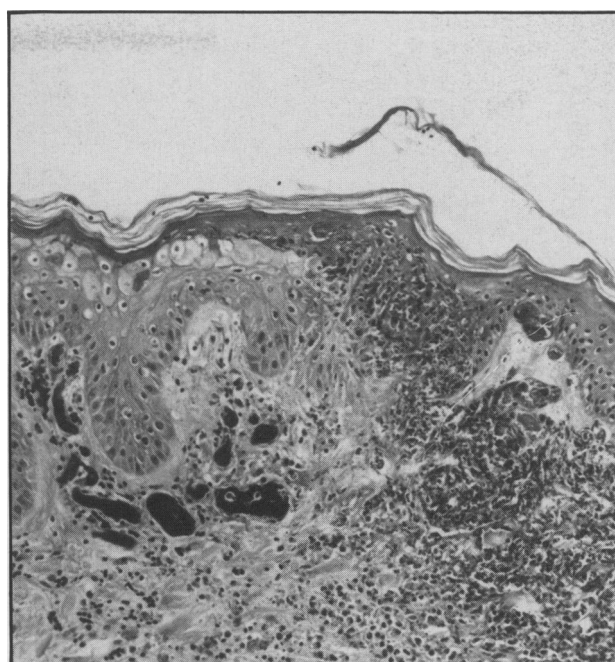
### Pathology

From herd 1, 5 mature sows, 2 feeder pigs, 6 piglets from birth to two weeks of age, and 18 fetuses aborted at 90–110 days of gestation were necropsied. From herd 2, both animals that died — one sow and one weaned pig — were necropsied. Gross and microscopic lesions were similar in pigs from both herds and are described together.

The most severe lesions were generally noted in the sows. Three sows had prominent skin lesions distributed over the back, abdomen, and extremities. These ranged from blotchy purple discoloration of the skin to multiple 0.5–2.0 cm raised plaques, typified by a white central region encircled by an erythematous margin. The underlying dermis and subcutis were thickened, edematous and focally hemorrhagic. Mammary glands in two sows were firm, congested, and edematous. There was generalized mesenteric, mediastinal, and occasionally, peripheral lymphadenopathy.

Pulmonary congestion and edema were present consistently, along with occasional subpleural petechiae. Lung tissue was firm. A unilateral fibrinous pleuritis was observed in one sow. Epicardial and endocardial ecchymoses were common.

The abdominal cavity usually contained a small quantity of clear yellow fluid and occasional fibrin strands. Splenic involvement varied from no visible change to mild enlargement. One sow had multiple, generalized, random foci of pinpoint hepatic necrosis. The fundic mucosa of the stomach was congested frequently.



**Figure 4.** Section of skin with focal epidermal necrosis in a mature sow. A zone of karyorrhectic neutrophils demarcates infarcted skin from less affected regions. H & E.

Gross lesions in feeder pigs and piglets were characteristic of septicemia. These included generalized congestion and edema of viscera, with foci of petechial serosal hemorrhage and lymphadenopathy. Two piglets also had focal hepatic necrosis, and another had a subcutaneous abscess. Significant gross lesions were not seen in the aborted fetuses.

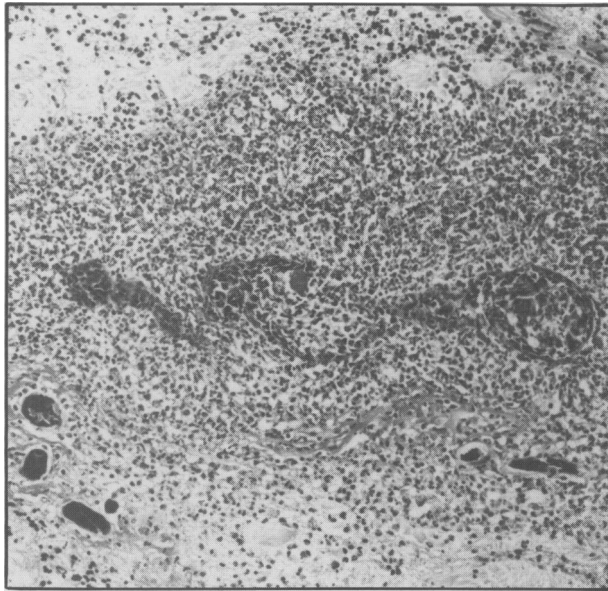
Histologically, affected skin had partial to full-thickness epidermal, as well as dermal, necrosis which was demarcated from less-affected skin by a broad band of degenerate neutrophils (Figure 4). Fibrinoid necrosis and thrombosis of superficial and deep dermal vessels were common (Figure 5). Colonies of coccobacilli were present within vessels and infarcted dermal tissue.

Random foci of necrosis were observed, with little inflammatory cell response, in the liver (Figure 6). Bacterial colonies were evident centrally in necrotic regions and in congested sinusoids. Similar necrotic foci were observed in the spleen of one sow. Bacterial colonies and fibrin thrombi were occasionally visible within vessels. One sow had a fibrinous pleural exudate, and focal parenchymal necrosis was noted in the lung of another.

Spleen had prominent lymphoid necrosis and depletion with perifollicular hemorrhage. Scattered throughout the red pulp were numerous neutrophils and occasional clusters of coccobacilli. Lymphocytolysis was also seen in lymph nodes and Peyer's patches.

Mammary glands had widespread interstitial edema and congestion, with a patchy neutrophilic infiltrate. Focal hemorrhage and bacterial colonies were located primarily within the interstitium.

Microscopic lesions in tissues from feeder pigs and piglets consisted of widespread visceral congestion and edema, rare focal hepatic necrosis, generalized lymphoid depletion, and numerous bacterial colonies located within sinusoids and vessels. Visible abnormalities were not evident in tissues from fetuses.



**Figure 5.** Section of deep dermis from same sow as in Figure 4. Vessel undergoing leukocytoclastic vasculitis, surrounded by intense inflammatory reaction. H & E.

### Microbiological findings

In herd 1, from all of the dead pigs except the euthanized sow and the aborted fetuses, *A. suis* was isolated in large numbers and pure cultures from all the organs and tissues sampled. From the skin lesions of the euthanized sow were recovered alpha-hemolytic streptococci and *A. suis*, and from the aborted fetuses — streptococci and *E. coli*, but no *A. suis*. From the two pigs that died in herd 2, *A. suis* was isolated from the lungs, tonsils, spleen, skin and the placenta. Mycoplasma or viruses were not found in any of the affected pigs from herd 1. Pigs of herd 2 were not examined for the presence of these agents.

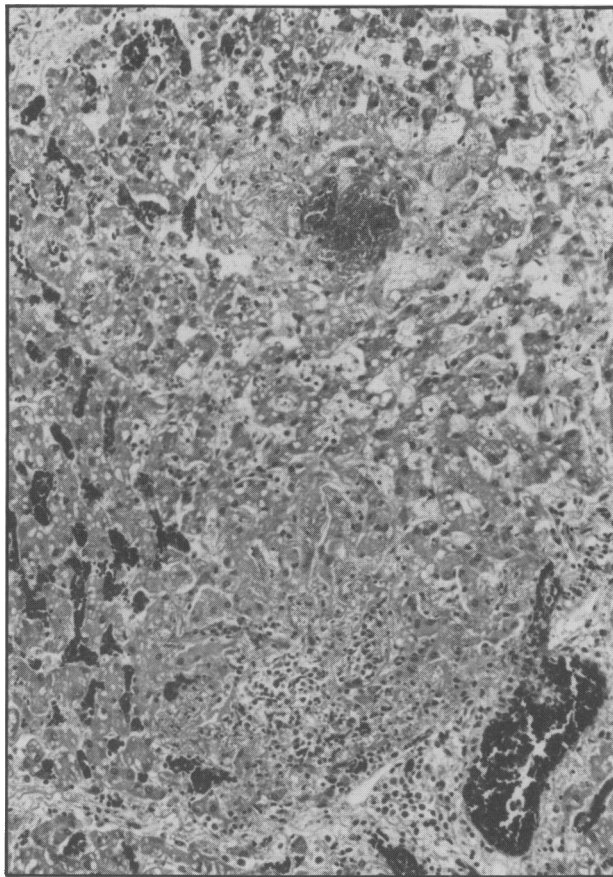
The *A. suis* isolated was sensitive *in vitro* to ampicillin, penicillin, tetracycline, chloramphenicol, nitrofurazone, cephalothin, gentamicin, trimethoprim-sulfamethoxazole, and cefoxitin.

Serum from animals in herd 1 had been consistently negative for antibodies to *Actinobacillus pleuropneumoniae* hemolysin antigen prior to the outbreak. After the outbreak, serum samples from 15 pigs all had antibodies to this antigen (15). Because there is considerable antigenic similarity between the *A. pleuropneumoniae* hemolysin and the *A. suis* hemolysin and herd 1 is free of *A. pleuropneumoniae* infection, the seroconversion indicates wide dissemination of *A. suis*. This has been confirmed by isolation of *A. suis* from tonsils of carrier animals on several occasions after the outbreak.

### Treatment

In herd 1 the affected animals were treated with several injectable antibacterial drugs. The regimen and results are presented in Table 1.

In herd 2, 12 sick sows were treated with high doses of benzathine procaine penicillin G, as described in the previous section of this paper and responded rapidly. Two animals died suddenly without treatment. Most



**Figure 6.** Section of liver from same sow as in Figure 4. Focus of hepatic necrosis is typified by absence of a marked cellular reaction. A colony of coccobacilli is visible in the center of the necrotic region. H & E.

of the suckling pigs in herd 1 died suddenly without treatment.

Beginning on day 10 of the initial outbreak, the sows in herd 1 were medicated for one week with feed containing oxytetracycline hydrochloride as already described. It was impossible to evaluate the effectiveness of this treatment as the disease appeared to have abated in this group of animals at the time treatment began.

### Discussion

In contrast with previous reports of sporadic outbreaks of actinobacillosis affecting mainly young pigs, in these two herds a severe septicemia occurred initially and principally in the adult population. Clinically, in many respects, the disease resembled acute swine erysipelas, but the lesions did not exclude other severe septicemic diseases of swine, including *Salmonella choleraesuis* infection. Moreover, as *A. suis* has been isolated from pigs affected with viral diseases, e.g. hog cholera (8,9), a thorough microbiological examination of cases of this nature is mandatory in order to arrive at a correct diagnosis.

Presumably this was the first exposure of the MD pigs in both herds to *A. suis*. This fact may account for the high susceptibility of pigs of all ages to the infection. Treatment with common antibacterial drugs was reasonably effective, but the high incidence of sudden deaths resulted in serious loss. Furthermore, for

**TABLE 1**  
**Response of *Actinobacillus suis* infected pigs to treatment (herd 1)**

Drug	Dose IM/24 h	Age pig	Number treated	Number recov.	Recov. time (days)
Procaine penicillin G	(1.8-2.4 IU) × 10 <sup>6</sup>	Sows and boars	25	22	4-5
Trimethoprim sulfadoxine	1 mL/15 kg	Sows	3	3	2-4
Trimethoprim sulfadoxine	1 mL/15 kg	Feeders	10	10	2-4
Oxytetracycline HCl <sup>a</sup>	1 mg/kg	Feeders	2	2	3-4
Total treated			40	37	

Of 10 nontreated pigs, 9 died, most died suddenly, and one recovered

<sup>a</sup>Liquamycin — L.P., rogar/STB Inc., Montreal, Quebec

researchers working on problems related to *A. pleuropneumoniae* at the Ontario Veterinary College, the presence of *A. suis* in herd 1 meant losing valuable research material, as the two organisms cross-react serologically (16,17). Sale of pigs from herd 2 to other breeders with closed MD herds which may not carry *A. suis* presents a hazard, as evidence obtained so far indicates that the organism can persist in an infected herd for at least 18 months.

The rapid spread of the disease in herd 1 from one isolated area to others, indicates that *A. suis* can be readily transmitted by humans and/or by carrier pigs. As pigs had never been introduced directly into the herds in question and no contact with horses (a suggested possible source (7)), could be established, the mode of introduction of the organism into these herds remains unexplained.

The appearance of porcine actinobacillosis as an acute septicemic disease on the Ontario scene creates two principal problems. First, by closely resembling acute erysipelas, it presents diagnostic problems and may lead to disputes regarding the efficacy of the available vaccines against *E. rhusiopathiae*. Secondly, vigilance has to be exercised to prevent spreading of *A. suis* from infected to noninfected herds where the previously nonexposed pigs may be highly susceptible to this organism. Thus porcine actinobacillosis may have to be added to that category of diseases, e.g. Glasser's disease, which previously were considered to be of minor importance but which, with the introduction of modern management practices where large numbers of animals are kept isolated from each other, present a health hazard to previously nonexposed susceptible swine populations. Closed MD swine herds, such as those described, fall into the latter category and should be considered at risk. CVJ

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