

***Streptococcus suis* meningitis in finishing pigs of a repopulated herd**

Lisa M. Tokach, DVM

Repopulation of swine herds is an increasingly common strategy used to simultaneously enhance the genetic potential for lean gain, improve health status, and advance reproductive potential. This is a relatively new management tool, but one that can result in unexpected diseases in the herd.

Veterinarians and herd managers must be prepared to manage these unexpected health problems when they emerge in a repopulated herd. The source animals for repopulation are ideally from closed herds with high-health status. A repopulated herd of these gilts typically has less immunity than an established herd with a normal parity distribution. Dramatic illnesses in newly repopulated herds can include:

- vaginitis/cervicitis;
- staphylococcal dermatitis;
- viral mummification;
- *Haemophilus parasuis*;
- neonatal colibacillosis; and
- streptococcosis (Henry, 1992, Proc AASP Depop/Repop Symp, p. 112).

The agents considered to be the primary causes of these conditions are indigenous to most swine herds. Outbreaks in repopulated herds can be severe, but they are usually transient.

Streptococcus suis meningitis is reported to be most prevalent in intensive, total confinement systems with high population densities.¹ Prevalence and severity may be greater in herds where other major pig diseases are absent (Clifton-Hadley, 1986, Proc. AASP, p. 474). The disease can occur in pigs of any age in susceptible herds, but most cases occur in 3- to 12-week-old pigs.² The highest incidence is reported when weaned pigs are mixed together (Sanford, 1989, Proc AASP, p. 192). Although *S. suis* has been isolated from finishing pigs,^{3,4} we found no documentation of an outbreak of *S. suis* meningitis in finishing pigs in the United States prior to this case.

Herd History

A 600-sow, farrow-to-finish operation in Kansas was depopulated in July 1991. The herd was repopulated from a high-health status herd also in Kansas. Gilts were acclimated and bred in a new, off-site facility, then moved to the depopulated farm, which had been thoroughly cleaned, dis-

infected and was free of pigs for 1 month. Farrowing began in August 1991.

Production records revealed 9.5 pigs weaned per litter and an average adjusted 21-day litter weight of 131 lb (59.4 kg) for 683 litters born from August 1991 to March 1992. Neonatal colibacillosis outbreaks were recorded in October and November even though gilts were vaccinated prior to farrowing. At the peak of the colibacillosis outbreak, 50%–60% of litters were affected, resulting in a 2.6% increase in preweaning mortality (9.9% to 12.5%). In November and December, staphylococcal dermatitis affected pigs in both farrowing and nursery stages. Approximately 20% of pigs in the single-stage nursery were affected. Minimal death loss was incurred as a result of this disease. Both problems ceased by January 1992.

Meningitis Outbreak

In January 1992, seven gilts died suddenly in the off-site selection facility. *S. suis*, hydrogen sulfide toxicity, salmonellosis and *H. parasuis* were all considered as possible causes. New replacement stock, still from the original source, had been introduced into the facility 1 week prior to these deaths, but none of them were affected. The affected gilts were distributed throughout the facility.

Extensive diagnostic effort, including repeated bacterial culture, and histopathology, confirmed that the meningitis was caused by *S. suis*. All animals in the facility were treated with 10 mL (3,000,000 IU) procaine penicillin G intramuscularly for 3 consecutive days. Clinical signs ceased immediately and have not returned.

During a 2-week period in April 1992, streptococcal meningitis was diagnosed in finishing pigs ranging from 150–250 lb (68–113 kg). No signs were observed in farrowing, nursery or growing pigs. A total of 39 pigs died, with a morbidity rate of approximately 5% and mortality rate of 1.6% of the finishing pigs. The diagnosis was confirmed by histopathology and repeated isolation of *S. suis* from the brain. Clinical signs included conjunctivitis, ataxia, tremors, paddling, recumbency and death.

Gross necropsy revealed severe conjunctivitis and hyperemia of meningeal vessels. Fibrinopurulent exudate was observed on the dorsal surface of the brain and on the floor of the cranial vault. One pig had polyarthritis from which a pure culture of *S. suis* was obtained. No other significant gross lesions were noted in 21 pigs necropsied.

Abilene Animal Hospital P.A., Abilene, Kansas, 67410

Clinical signs and deaths ceased immediately after all pigs in the barn were injected with 3,000,000 IU of procaine penicillin G.

No clinical signs have been observed in the grower or nursery pigs. Third-parity sows have just begun to farrow in the herd. Two groups of pigs have been moved from grower to finisher areas and no new cases of meningitis have been reported. However, it is significant to note that a new gilt finishing barn was completed at the same time, alleviating some overcrowding in the existing finishing barns.

Conclusion

As average herd parity increases, it is thought that the *S. suis* meningitis will subside. Other repopulated herds have reported short-lived *S. suis* outbreaks in the nurseries. It appears that herd immunity develops rapidly. It is uncertain at this point why these animals were not affected until the pigs reached the finishing barns. The question remains whether this was a highly virulent strain of *S. suis*, or a low-virulence strain in a highly susceptible population. A future trial in which both conventional pigs and immunologically susceptible animals are exposed to the *Streptococcus* strain isolated in this case may help answer that question.

References

1. Sanford SE, Higgins R. Streptococcal Diseases. In: Leman AD, Straw BE, Mengeling WL, D'Allaire S and Taylor DJ, eds. *Diseases of Swine*. 7th ed. Ames, IA: Iowa State University Press: 1992:588-590.
2. Taylor DJ. *Pig Diseases*. 4th ed. Foxton, Cambridge: The Burlington Press: 1979:156.
3. Davies PR, Ossowicz CJ. Evaluation of methods used for detecting *Streptococcus suis* type 2 in tonsils, and investigation of the carrier state in pigs. 1991 *Res Vet Sci*; 50:190-194.
4. Sanford SE. Gross and histopathological findings in unusual lesions caused by *Streptococcus suis* in pigs II. Central nervous system lesions. 1987 *Can J Vet Res*; 51:486-489.

Editors' Note— We received the following from Dr. Tokach as we went to press:

Addendum

Since the original report, an additional *S. suis* outbreak has been documented in a similar high health herd.

The second unit is a new 1200-sow herd that began farrowing in April, 1992. Acute death loss of 72 head was observed in the oldest finishing animals in October, 1992. It then spread to younger growing and finishing animals. There was a positive clinical response to procaine penicillin G. All clinical signs had ceased within 4 weeks of the first signs. A total of 106 pigs died, with a morbidity rate of 3% and a mortality rate of 1.4% of the growing and finishing pigs.

As with the other herd in this report, it appears that naturally acquired resistance to clinical streptococcal meningitis may represent expected homeostasis in intensely managed pig populations.