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Steven Claas

Michael Klatt

Layout and CD-ROM

David Brown

Logo Design

Ruth Cronje, and Jan Swanson;

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Personal experience with *Actinobacillus suis*

Luc Dufresne, D.V.M., Dipl of ABVP
Seaboard Foods

Introduction

Actinobacillus suis (*A. suis*) is a gram-negative, facultative, anaerobic bacteria traditionally associated with septicemia and death in suckling and recently weaned piglets.¹ In the 1990's cases of septicemia resembling Erysipelas in mature animals, and pneumonia resembling pleuropneumonia in growing pigs, were described in North America.² Although initially described as a disease of high health, start-up herds, the disease is now seen in several herds with standard health and normal parity distribution.

Epidemiology

Actinobacillus suis can be carried in the tonsils and nostrils of healthy pigs and in the vagina of healthy sows. The most likely source of infection is through the birth canal or nose-to-nose contact between pigs. Aerosol or other area spread may happen but appear to be rare considering the following epidemiological observations:

- Observation of the disease in breeding herds is common from herds sharing similar gilt suppliers or GDU's
- Finisher disease strongly correlated with sow source
- Absence of transmission between finisher barns in close proximity to each other

Clinical signs

Disease in suckling pigs is characterized by sudden death in piglets of all ages. Few litters are affected, but several piglets within the same litter are affected. Animals affected show pyrexia, anorexia, cyanosis, and panting and usually die quickly if not treated. The lesions observed are cyanotic, petechial hemorrhages in several organs. The impact on mortality is variable but it usually will not increase preweaning mortality by more than 2%.

Grow-finish disease is also characterized by cyanosis and sudden death, but thumping may also be present. Lesions of septicemia may also be seen in these animals, but the most striking lesion is a necro-hemorrhagic pneumonia similar to the lesions created by *Actinobacillus pleuropneumonia*. The clinical picture is usually enzootic with a few cases appearing sporadically throughout the finisher.

The total impact is a 1 to 2% increase in finisher mortality. A small number of clinical outbreaks may be quite severe causing mortality of 0.5 to 1.0% per day for a week.

Adult septicemia lesions are usually seen in high health herds. In these cases clinical expression is very similar to Erysipelas with animals displaying fever, anorexia, and red, rhomboid skin lesions. The lesions in these animals are of a septicemic nature with multifocal, petechial hemorrhages and serofibrinous exudates in the thoracic and abdominal cavities.

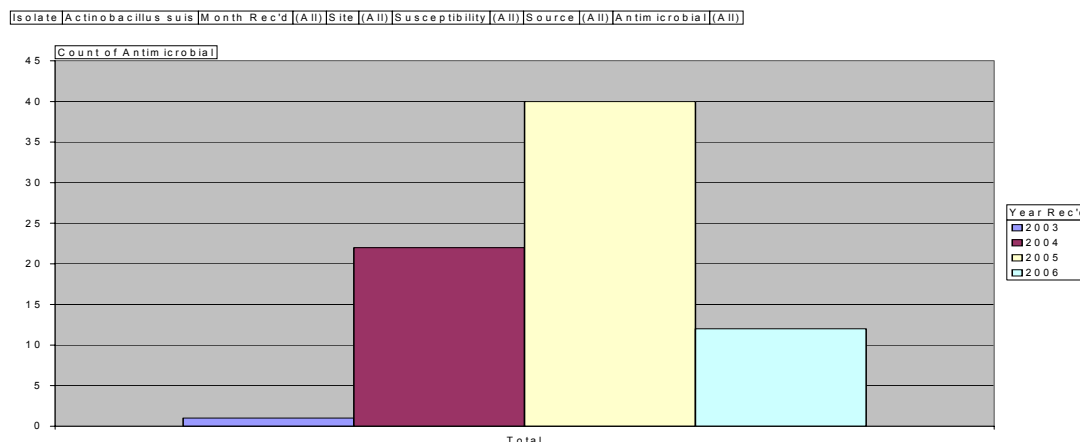
In the Seaboard Foods system we have seen the majority of the cases in suckling piglets and pneumonia/septicemia in growing pigs. We isolated the bacteria from clinical cases 22 times in the last half of 2004, 40 times in 2005, and 12 times in the first half of 2006. All cases in 2004 and 2006 were isolated in the finisher, where over 50% of the cases in 2005 were isolated from sucking piglets. During the same period, we have seen an increase in septicemia issues in the affected herds.

In my experience *A. suis* behaves like other early colonizers (*Haemophilus parasuis*, *Streptococcus suis*, and *Actinobacillus pleuropneumonia*) which suggests that the vertical infection of a piglet born from an infected sow is very high. The risk of horizontal transmission decreases as pigs grow older and is significantly reduced if animals are kept in separate rooms or barns. Most of the isolates have been from the lung, but the bacteria has also been isolated from the liver, kidney, spleen, lymph node, and brain. It has also been isolated in a few cases from the intestine with questionable clinical relevance. (Figure 1)

Prevention

Introduction of healthy carriers remains the main route for introduction of *A. suis* into a naive farm. For most early colonizing bacteria, *Mycoplasma hyopneumoniae* excluded, the introduction of infected animals is the common source of introduction into a population. This is one reason why a change in gilt sourcing or the mixing of different gilt sources should be avoided. If such introductions are necessary, the health status of both populations should always be evaluated with extreme care. *A. suis* behaves epidemiologically like APP. If you do not have a history of isolating the bacteria within your system, you

Figure 1: Number of *A. suis* isolates per year



probably do not have it.

Control

Antibiotic therapy is usually effective in controlling mortality if animals are detected quickly. Chlortetracycline, Ceftiofur, Ampicillin, and Florfenicol are the drugs of choice in our system according to the sensitivity patterns of 75 isolates from the last 3 years. The main problem with effective treatment is the fact that employees are often not able to recognize diseased animals until it is too late.

Preventive injections in the farrowing house using one of the drugs can be clinically effective in reducing mortality. The sporadic clinical picture of *A. suis* in grow-finish ani-

imals makes it very difficult to use prophylactic antibiotic therapy in controlling the problem.

In my experience autogenous bacterins have been effective in controlling the problem, particularly in the farrowing house. Clinical septicemia in the farrowing house disappears following the initiation of an *A. suis* vaccination given to sows pre-farrowing. Vaccination of growing pigs has been effective in reducing clinical losses associated with *A. suis* in problem flows, although sporadic pleuropneumonia lesions are still detected. Economical relevance of the clinical expression should be evaluated and compared to the cost of a vaccination program to determine if this route is a cost effective way of controlling the disease.

Table 1: Antibiotic susceptibility of 75 *A. suis* isolates

| Antimicrobial | Susceptibility | | | Total |
|----------------------------|----------------|-----------|-------------|-------|
| | Intermediate | Resistant | Susceptible | |
| Ampicillin | 10 | 1 | 64 | 75 |
| Apramycin | 23 | | 11 | 34 |
| Ceftiofur | | 1 | 73 | 74 |
| Chlortetracycline | 1 | 2 | 72 | 75 |
| Clindamycin | 2 | 72 | 1 | 75 |
| Erythromycin | 72 | 2 | 1 | 75 |
| Florfenicol | | 1 | 73 | 74 |
| Oxytetracycline | 1 | 11 | 63 | 75 |
| Penicillin | | 74 | | 74 |
| Spectinomycin | 8 | 67 | | 75 |
| Sulphachloropyridazine | | 13 | 52 | 66 |
| Sulphadimethoxine | | 40 | 26 | 66 |
| Sulphathiazole | 48 | 27 | | 75 |
| Tiamulin | 4 | 34 | 37 | 75 |
| Tilmicosin | 1 | 4 | 70 | 75 |
| Trimethoprim/Sulphamethole | | 1 | 74 | 75 |
| Tylosin (Tartrate) | 2 | 72 | | 74 |
| Total | 174 | 429 | 814 | 1418 |

Conclusion

A. suis was previously described as bacteria expressing itself mostly in high-health and newly populated/repopulated herds. My personal experience has been that the organism can create problems in conventional and mature herds. Relevant clinical expression is septicemia in suckling piglets and septicemia/pneumonia in growing pigs. Prevention can be achieved by applying basic biosecurity rules and avoiding the introduction of infected animals. Short term control can be achieved by using effective antibiotics strategically. Long term control can be achieved by using autogenous bacterins on sows pre-farrowing or on growing pigs.

References

1. Sanford SE, et al. *Actinobacillus suis* infection in pigs in southwestern Ontario. *Can vet J.* 1990;31:443-447
2. Sanford SE. *Actinobacillus suis*: an overview of an emerging disease. *Proc Am Assoc Swine Prac.* 1995;26:425-428

