Salmonella Control in Feeder Barns

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Introduction

Salmonella infections in humans and livestock are not a new phenomenon. However recently much more attention has been directed to this disease for two reasons. Firstly, in the 1990's, surveillance of food-borne pathogens has increased which gives the impression that levels of salmonella in meat products are increasing dramatically. This rise in surveillance has coincided with more attention to the emergence of virulent strains and more attention to antibiotic resistance. Secondly, from the swine medicine perspective, there have been more reports in the late 90's of *Salmonella typhimurium* clinical outbreaks in "high-health" growing pigs raised in well-run, sanitary barns. This report presents such a case.

History

In July 1996, a new 4 room -2000 head - feeder barn had just filled with 25 kilogram feeder pigs from 6 sow farms each with on-site nurseries. The feeder barn was a fan ventilated barn with about 30% of the pen floor space slatted. Water and feed were provided in a wet/dry feeder. Water was obtained from a man-made pond on the farm, and feed was a pelleted barley/soybean formulation purchased from a local feed supplier.

The batch of pigs started with no problems. On the seventh day after the fill the manager found 4 dead pigs in one room during his morning pen checks. They had a slightly reddish skin coloration especially on the belly area. By that afternoon about 25 pigs in 4 pens were noticed to have a watery amber-coloured diarrhea. Four more pigs in the same room died later that day. The next morning 6 more pigs were dead, 5 from the same room and 1 from another room. A clear yellow-tinged diarrhea was seen in 9 of the 24 pens in the room that first had the problem and in 2 pens in the other room. Problems seem to "cluster" around certain pens in the barn. Diarrheic pigs were lethargic.

The pigs did not appear to be dehydrated. Post mortems were performed and intestine, liver, lymph nodes, lung, spleen, kidney were submitted to the local diagnostic laboratory for culture and sensitivity. Feed samples were submitted for salmonella isolation. The pigs were treated with Neomycin (Neomix Soluble Powder®, Pharmacia Animal Health, 125 gm / 678 litres drinking water) in the water and all sick pigs were injected with sulfa-trimethoprim (Borgal®, Intervet Canada Ltd., Dose – 3 ml per 45 Kg BW intramuscularly). By day 6, mortality rose to 52 pigs in the first room and 25 pigs in the other infected room. Interestingly the problem stayed out of the other two rooms. Once the "hot wave" of infection subsided after about 10 days there was no recurrence of problems and the batch went on to finish out normally. Overall mortality from the outbreak was 3.9%.

The laboratory isolated salmonella from intestines and, in some pigs, they also found it in other tissues such as lung and kidney (Table 1). It was later typed as *Salmonella typhimurium* Phage types 104 and 108. No salmonella was found in the feed samples.

Table 1. Positive *Salmonella typhimurium* isolations from necropsied pigs

Pig #	Intestinal Content	Mesenteric Lymph Nodes	Other Lymph Nodes [1]	Lung	Kidney
1	+[2]	+[2]	-	-	-
2	+[2]	+[2]	+[2]	+[2]	+[2]
3	+[2]	-	-	-	-
4	+[2]	+[2]	+[2]		+[2]
5	+[3]	+[3]	-	-	-
6	+[3]	+[3]	-	-	-
7	+[2]	+[2]	+[2]	+[2]	+[2]
8	+[3]	+[3]	-	+[3]	-

[1] Inguinal and Popliteal Lymph Nodes
[2] Phage type DT 104
[3] Phage type DT 108

But the story is not finished. The barn was completely emptied, and it, and all equipment, was cold water washed – no detergent- and disinfected thoroughly. The next group was brought in (same sourcing as the first batch) and the same outbreak of the characteristic clear yellow diarrhea occurred. It affected about 20/96 pens in the barn. The manager started the group on neomycin in the water immediately and mortality was contained to 26 animals or 1.3% of the

batch. At the end of that batch the barns were washed and disinfected more thoroughly, the pits were cleaned and disinfected, and water lines were pulse disinfected with chlorine. Source herds were examined for any evidence of salmonellosis and laboratory cultures of random fecal and pen sampling did not pick up any salmonella. The feeder pig trailer was swabbed after the routine washing, and before transporting the feeder pigs to the barn, and no salmonella was cultured. The barn was filled and within two weeks the same thing happened but again less severe than in the previous batch.

This problem continued for two more batches, each time less prevalent and severe than the previous time. The barn manager was able to detect the presence of pigs with salmonella enteritis very quickly and would inject the entire pen with a sulfa-trimethoprim product. This approach would contain the problem to a small number of pens in the barn. An internal biosecurity program was started where boots were changed between rooms and one pair of boots was designated to be worn only in pens with pigs being treated for salmonella enteritis. Between uses they were stored in a disinfectant bath. The barn was cleaned and disinfected in a normal manner and then various areas were swabbed to detect any salmonella present. Table 2 indicates where salmonella was found. A more thorough cleaning was done with hot water and with special attention to feeders and areas of dust accumulation. Routine chlorination of drinking water was started. A more thorough rodent control program was initiated. From that batch to the present no salmonella problems have been seen clinically.

Table 2. *Salmonella typhimurium* Isolations from the barn environment.

Area Tested	Number Positive/Number Tested
Dust on feed lines	3/6
Dust in re-circulation box	1/6
Feeder tray	2/8
Solid floors	0/8
Slat tops	0/8
Slat sides	2/8
Pit slurry	0/6
Concrete penning	0/6
Mice	2/6

■ Discussion

A few questions remain about this disease outbreak.

Where did the Salmonella come from?

Did it arrive in the purchased feeder pigs?
There were about a dozen other feeder barns that were taking pigs from the same sow sources that did not experience any clinical problems from salmonella. A random sampling of pigs in the source herd nurseries did not pick up salmonella.

Did they pick it up from contaminated transportation?
The delivery trailers were used only for feeder pig delivery within this network of pig producers. Other barns filled from pigs transported on these trailers did not break with salmonella.

Did the salmonella arrive on that farm through other sources?
Did people or wild animals track it in? Was it brought to the farm in the intestinal tract of birds, rodents or other animals? During the construction phase there was free access to the building by all.

Was contaminated feed the source?
Possibly, but no salmonella was isolated in this case. No clinical salmonella was seen in other herds that I service that purchased feed from that mill. North American and Danish surveys indicate that *Salmonella typhimurium,* unlike certain other serotypes, is not commonly a contaminant in swine feed.

The problem with verifying a likely source of infection is the ability of all salmonella to live in low numbers in the intestines of healthy animals. Manure sampling could turn up negative results even on infected carrier animals. This is due to both the sensitivity of the sampling and the testing procedure, which might be too low to detect a low number of organisms present. Also chronic carrier animals will shed salmonella in their feces only sporadically. When trying to find the bacteria in the environment or feed, low grade levels probably would not be detected because the number of samples needed for detection would be higher than the number that are often taken in barn checks.

Once on the farm, where does salmonella survive between all-in-all-out batches of pigs?

The salmonella remained on farm in spite of more stringent washing and disinfection. It was not until our investigation showed that this manure-borne organism was found in areas other than floors, slats and pits, that a more thorough reduction of the salmonella in the barn environment could be accomplished. And what was the role of rodents as a reservoir for the disease

organism? My colleagues in the poultry industry have told me that in some barns, rodent control was the turning point in salmonella reduction programs. A study showed that mice can shed up to 230,000 salmonella per fecal pellet (Fedorka-Cray, 1999). It takes as few as 10,000 salmonella of some serotypes to clinically infect a healthy pig.

What allows the salmonella to become a clinical problem?

Were the animals challenged with a high level of salmonella? Usually we would associate these problems with unsanitary conditions where the level of salmonella in the environment is high. This barn was very clean. The pigs dunging pattern was normal and any manure accumulation was scraped away daily. It did not appear likely that a large salmonella build up caused the problem. One study in chickens indicated that salmonella could be transmitted through the air (Fedorka-Cray, 1999). It also indicated that the dose of infective bacteria required to cause disease was less when infection came via the air than via oral exposure. This was dependent on the virulence of the organism. Our barn check showed salmonella in dust in recirculation ducts and on feed lines (Table 2). This is most likely dust from dried feces and may present another route of salmonella spread in the barn.

Was it a factor of virulence?

Some serotypes of salmonella require a very small dose of bacteria to create disease in healthy non-stressed pigs (Wilcock and Schwartz, 1999). This may be as few a 10,000 organisms compared to a normal infective dose of 100,000,000 bacteria.

Were the pigs in a period where they were more susceptible to infection and the clinical disease? Stressors that suppress the animals' immune system such as transportation, and mixing, are known to allow healthy salmonella carriers to start shedding the bacteria in large numbers in their manure. The same stressors increase the pig's susceptibility to a disease. Changes in feed, water, or antimicrobial use may alter the normal balance of bacteria in the gut and allow salmonella numbers to reach a clinical disease level.

■ Conclusion

This paper has dealt mainly with the clinical disease as it occurs in pigs, but the control of salmonellosis is only one part of picture. A much bigger issue facing the livestock industry is the potential for salmonella and other food-borne pathogens to move through the food chain and cause problems in humans.

■ References:

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