An outbreak of salmonellosis in a swine finishing barn

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Summary

This case report describes an outbreak of disease caused by *Salmonella* serovar Typhimurium, phage type 108, in a batch finishing barn in Ontario, Canada. The outbreak was treated with both water-soluble and injectable antibiotics. Normal cleaning and disinfecting protocols were modified to decontaminate the facilities. Two members of the producer's family required medical treatment for salmonellosis.

Keywords: swine, salmonellosis, *Salmonella* serovar Typhimurium

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Resumen – Un brote de salmonelosis en un sitio de finalización de cerdos

Este reporte describe un brote de enfermedad causado por la *Salmonella* serovar Typhimurium fago tipo 108 en un grupo de finalización en Ontario, Canadá. El brote se trató con antibióticos tanto solubles en agua como inyectables. Se modificaron los protocolos de limpieza normal y de desinfección para descontaminar las instalaciones. Dos miembros de la familia del productor requirieron de tratamiento médico contra la salmonelosis.

Résumé – Une première manifestation de salmonellosis dans un troupeau de finition porcin

Ce rapport décrit une première manifestation de maladie causée par la *Salmonella* serovar Typhimurium phage type 108 dans un troupeau de finition dans une ferme à Ontario, Canada. La première manifestation a été traitée avec les antibiotiques solubles dans l'eau et les injectables. Les protocoles de nettoyage normal et de désinfection ont été modifiés pour décontaminer les installations. Deux membres de la famille du producteur ont exigé le traitement médical contre la salmonellosis.

Salmonellae are hardy organisms that can survive freezing and desiccation, persisting for months or even years in suitable organic substrates.[1] They are readily inactivated by heat, sunlight, and some classes of disinfectants.[1] The reservoir for *Salmonella* serovars that are either pathogenic or nonpathogenic in swine is the intestinal tracts of animals and humans.[1,2] Salmonellae are present in a significant number of carcasses and retail meat products.[1] Salmonellosis is the most common foodborne illness in humans,[1] with *Salmonella* serovar Typhimurium the most frequently reported serovar.[3,4]

In swine, *Salmonella* infections cause two separate problems: clinical salmonellosis and a clinically silent carrier state that may cause foodborne disease if fecal contamination of pork occurs. The carrier state is much more common than clinical disease. Salmonellae are facultatively intracellular organisms, and the carrier state is believed to be a function of this characteristic.[5] Macrophages are the most likely disseminators of infection inside the body. Inapparent long-term carrier animals may shed salmonellae in feces either intermittently or continuously.[1] Those shedding when transported to market contaminate the truck and abattoir.[6] Prevalence of infection increases with the length of pre-slaughter lairage.[7]

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Sources of *Salmonella* Typhimurium infection for swine include other pigs, feed, water, bedding, rodents, birds, insects, and people, with transmission primarily by ingestion. Aerosol transmission for short distances is possible via aerosolized secretions, feces, or dust[1,8] and results in rapid proliferation in the digestive tract.[3]

When pigs are fed coarse, nonpelleted rations, stomach contents have a lower pH and the concentration of lactic acid and in vitro death rate of *Salmonella* Typhimurium are higher, resulting in a lower rate of fecal shedding than when pigs are fed fine-pelleted rations.[6] It is likely that during periods of stress (eg, commingling, transportation, chilling, and concurrent disease or starvation), release of catecholamine reduces gastric acid production, thus permitting increased fecal shedding of salmonellae.[1]

Salmonella prevalence varies widely among farms and at different growth stages within the same farm, indicating the dynamic and variable pattern of fecal shedding.[9] Risk factors associated with *Salmonella* prevalence on swine farms include personnel hygiene, flooring type, housing contamination, pig flow management, insects, rodents, wild birds, cats, feed form, season and temperature, stocking density, and herd health status.[2]

Most outbreaks of salmonellosis occur after weaning, with the low frequency in suckling piglets presumably the result of lactogenic immunity.[1] The initial sign of clinical disease is watery yellow diarrhea, with blood sporadically seen in the feces. Affected animals are febrile, anorexic, and dehydrated. Severity of disease depends on many factors, including strain virulence, host resistance, route of infection, and infecting dose.[1] Mortality is variable. Most pigs make a complete recovery and eliminate the organism, but others may remain carriers and intermittent shedders for several months.[1,3,10]

Clinical diagnosis is supported by isolation of salmonellae. Preferred tissues for sampling include ileum, ileocecal lymph nodes, tonsil, and cecum. In live animals, tonsil scrapings are preferable to rectal swabs for isolation

because of the unpredictability of fecal shedding in asymptomatic carriers. Isolation alone is not sufficient for a definitive diagnosis due to the ubiquitous nature of the organism in clinically normal animals. Bacteriology results should be supported by histology and other tests, such as phage typing or polymerase chain reaction. Current serological tests are neither sensitive nor specific enough to be used for individual animal diagnosis, but are essential in determining the prevalence of asymptomatic carrier animals and are thus essential to herd control measures.

In an outbreak of salmonellosis, antimicrobials are usually administered in the drinking water to all animals in the affected group.[1] Because of the need for immediate therapy, antibiotic choices are usually based on previous experience rather than on the results of laboratory testing. Visibly affected animals respond to parenteral antimicrobials, and anti-inflammatory agents are useful in treatment of critically ill animals to combat the effects of endotoxin. Isolation of sick animals and good nursing care are essential in reducing mortality.

Control of salmonellosis depends on minimizing exposure dose and maximizing immunity. Methods of reducing exposure include all in-all out (AIAO) management, single-source groups, and sanitation protocols. Ensuring proper pig density, adequate ventilation, and minimal temperature variation may maximize pig resistance. Killed *Salmonella* Typhimurium bacterins do not stimulate cell-mediated immunity, which is important in eliminating the organism.[4] Under research conditions, live attenuated *Salmonella* Typhimurium vaccines reduce tissue colonization,[11,12] and, in at least one study,[11] reduced fecal shedding and prevented development of clinical signs. However, the zoonotic potential of *Salmonella* Typhimurium raises concerns regarding the use of live vaccines in the context of increasing the prevalence of this foodborne pathogen at the farm level.[13] Commercially available live attenuated vaccines for *Salmonella* serovar Choleraesuis can provide cross-protection against *Salmonella* Typhimurium.[4,13–15]

Case description

In March 2000, the owner of a 2000-head contract finishing barn in southwestern Ontario contacted his veterinarian to report diarrhea in 10% to 15% of the animals. The animals were farrowed in two 1250-sow herds serologically negative for *Mycoplasma hyopneumoniae* and serologically positive for porcine reproductive and respiratory syndrome virus (PRRSV). Sows were vaccinated against parvovirus, leptospirosis, erysipelas, and enterotoxigenic *Escherichia coli.* Piglets were weaned at 15 to 17 days of age into one of four 2100-head, mechanically ventilated contract nurseries, and received no vaccinations. Each nursery was filled from the two sow herds over the course of a 10-day period. When the pigs were approximately 10 weeks of age, each nursery was emptied on a single day and the feeder pigs were transferred to one of eight 2000-head, AIAO, curtain-sided, dual-ventilated contract finishing barns. Each barn was divided into two rooms separated by a door. In each room, 34 pens each housed approximately 30 pigs (space allowance, 0.72 m^2 per pig). The pigs were housed on fully slatted concrete floors, with feed and water supplied ad libitum. Pelleted corn-soybean rations were provided by the feed mill that owned the pigs. The rations were medicated with tylosin phosphate (22 g per tonne) during the entire growing-finishing period and contained added poultry fat. Barns were refilled every 16 weeks after cleaning and disinfection. Manure was removed from the pits under the slats twice annually.

Both feeder pigs and market-weight animals were transported on vehicles that belonged to a single company. This company also used the same trucks to transport pigs owned by other production systems. The trucks were washed and disinfected between loads unless they were hauling several loads of market hogs from a single barn. If time permitted, the trucks were dried overnight in a heated bay, and otherwise were dried outside. In the winter, this meant that some of the trucks did not dry completely after washing.

Feeder pigs had been delivered to the barn during the third week of December 1999, and, until March, there had been no significant health problems. The producer began to weigh and sort pigs the week before the outbreak occurred. Market-weight animals were mixed for several days prior to shipping. Diarrhea was first noticed in pens from which market hogs had been selected on March 12. The producer interpreted diarrhea in these pigs as a sign of ileitis, and affected animals were treated intramuscularly (IM) with injectable tylosin phosphate (20 mg per kg) once daily for 3 days. Treatment response was poor, and six of the treated animals died. During the visit by the veterinarian on March 14, approximately 85% of the pigs appeared normal. The remaining animals were lethargic and gaunt. Watery yellow to pale brown diarrhea was evident in many pens. Two affected animals were euthanized for necropsy. Both animals had empty stomachs, pale livers, enlarged mesenteric and ileocolic lymph nodes, and fluid-filled small and large intestines. Fresh and formalinized samples of lung, spleen, liver, kidney, ileum, and spiral colon from each animal were submitted to the Animal Health Laboratory (AHL; University of Guelph, Guelph, Ontario, Canada) for culture and histopathological examination.

Diagnostic differentials included salmonellosis, ileitis, transmissible gastroenteritis, spirochaetal colitis, rotavirus, swine dysentery, and trichuriasis. On the basis of clinical signs and the previous health status of the animals, salmonellosis was the primary differential, and immediate antibiotic therapy was initiated in order to prevent further deaths. Drinking water for the entire barn was medicated with neomycin sulfate (9.3 mg per kg) for 5 days, beginning on March 14. Individual affected animals were injected IM with ceftiofur sodium (3 mg per kg) once daily for 3 days. The most severely affected animals also received isoflupredone acetate IM (0.036 mg per kg) once daily for 2 days. Market shipments were delayed for 3 weeks because of the antibiotic withdrawal period.

Laboratory results confirmed the clinical diagnosis. *Salmonella* Typhimurium phage type 108 was isolated from the ileum and mesenteric lymph nodes of both sampled pigs. The organism was sensitive to ampicillin, ceftiofur, neomycin, and trimethoprim-sulfa. Histologically, there was congestion of the liver and kidneys, congestion and edema of the mesenteric lymph nodes, and marked necrosis and erosion of the colon, with a marked mononuclear cell reaction in the lamina propria.

Response to treatment was good. Within 2 days of starting antimicrobial treatment, the pigs showed considerable improvement, although a few were still clinically ill. Death losses stopped within 7 days, and there was no recurrence of the problem before the animals were shipped to market. The total group mortality was 1.8%, with 0.85% dying during the outbreak of salmonellosis.

When market shipments resumed in the first week of April, 2 weeks after the last

case of diarrhea in the pigs, the slaughter plant was notified so that they could segregate the animals from this barn for slaughter at the end of each day in order to allow for a thorough cleaning and disinfecting of the facilities.

The protocol for decontamination of the barn included washing the ceiling, feed lines, barn tools, coveralls, and footwear, in addition to the routine high-pressure cold-water washing of walls, floors, and feeders. A quaternary ammonium product was used to disinfect the barn.

The first empty room was cleaned and disinfected according to the protocol, and refilled on May 3. However, due to trucking problems, it was not possible to completely empty the second room until May 4; after cleaning and disinfection, it was refilled on May 6. Therefore, for a 24-hour period, both batches cohabited the barn. Despite biosecurity precautions (eg, changing clothing and footwear between rooms), the new batch of pigs developed clinical signs of salmonellosis within several days of arrival. Antibiotic treatment was initiated within 12 hours of the first clinical signs; as a result, there were fewer severe cases than in the previous outbreak, and mortality due to salmonellosis was only 0.3%. After the barn was emptied again in September 2000, in addition to the protocols described above, the water lines were chlorinated, the barn and pig transport trailers were washed with hot water and disinfected with a formaldehyde-based disinfectant, and an intensive rodent-baiting program was implemented. The barn was allowed to dry for several days before refilling. In subsequent batches of pigs, there have been occasional, milder signs of salmonellosis that have been controlled with injection of antibiotic in individual animals.

The producer's family was diagnosed with salmonellosis prior to the outbreak in the pigs. His 10-year-old son, who had assisted with unloading the pigs when they arrived in December, developed a diarrheal disease a few days later. While caring for him, his mother also contracted the disease. Both recovered fully after medical treatment. Diagnosis was salmonellosis caused by *Salmonella* Typhimurium. Phage-typing results were not available for the human cases.

Discussion

Infection of swine with more than one serovar of salmonellae is common, but clinical disease caused by serovars other than *Salmonella* serovars Choleraesuis or Typhimurium is uncommon. In Ontario, most outbreaks of salmonellosis have been attributed to infection with *Salmonella* Typhimurium, and this serovar accounted for 69% of *Salmonella* isolations in porcine submissions received in the years 2000-2003 at the AHL (AHL database).

Several risk factors may have contributed to the disease outbreak in this case. The pigs were probably infected before they arrived at the finishing barn, either prior to transport (although *Salmonella* Typhimurium has never been isolated in the sow barn or nursery) or after exposure to a contaminated truck. Selecting market hogs for shipment to slaughter might have induced a stress-related outbreak of clinical disease. In addition, these pigs were on a pelleted diet, even though pelleted rations are associated with a higher incidence of salmonellosis,[6] because coarse, nonpelleted feed has a detrimental effect on growth performance, increasing the cost of production. Third, the use of antibiotic growth promoters may select for antibiotic-resistant bacteria.[16] The pigs in this case received tylosin phosphate during the entire growing-finishing period. However, feeding tylosin as a growth promoter has not been associated with an increase in the risk of infection with *Salmonella* Typhimurium,[16] nor does it select for *Salmonella* with new resistance phenotypes.[17]

Côté et al[18] found that salmonellae persisted for at least 7 days in extraintestinal tissues after oral infection and recommended that affected animals not be slaughtered for at least 1 week after a clinical episode in order to reduce carcass contamination. In this case, all animals were kept on the farm for 14 days after resolution of clinical signs.

It was necessary to make changes in the cleaning and disinfection protocol in order to reduce exposure in future batches of pigs. Not all disinfectants are effective in killing salmonellae: products with a residual effect are necessary.[19] In this case, it was necessary to change from a quaternary ammonium product to a formaldehyde-based disinfectant. In addition, the increased "down time" allowed pig contact surfaces to dry properly, which is also important in killing salmonellae.[19]

Humans may become infected with salmonellae from food, water, the environment, and directly from infected people or livestock.[20,21] The timing of clinical signs in the producer's son suggested exposure from contact with the pigs' feces, although there was no confirmation that the same phage type was involved in the porcine and human infections. Children are more likely to put their fingers in their mouths, thus increasing their risk of developing a zoonotic enteric disease. In this case, the herd veterinarian recommended that the producer not allow children under the age of 16 to enter the barn. An adult was hired to assist with barn chores. Neither the owner nor this hired man has ever developed signs of salmonellosis.

Implications

- In a group of swine exposed to infection with *Salmonella* Typhimurium, clinical signs of infection may develop in only a small percentage of the animals and group mortality may not be affected.
- Stressful events, eg, transportation and commingling, may contribute to outbreaks of salmonellosis in swine.
- After an outbreak of salmonellosis, more stringent AIAO management and changes in cleaning and disinfection procedures, including more rigorous cleaning, change in type of disinfectant, and increased drying time, may be necessary to reduce exposure of future batches of pigs.

References

1. Schwartz KJ. Salmonellosis. In: Straw BE, D'Allaire S, Mengeling WL, Taylor, DJ, eds. *Diseases of Swine.* 8th ed. Ames, Iowa: Iowa State University Press; 1999:535–551.

2. Funk J, Gebreyes WA. Risk factors associated with *Salmonella* prevalence on swine farms. *J Swine Health Prod.* 2004;12:246–251.

3. Fedorka-Cray PJ, Kelley LC, Stabel TJ, Gray JT, Laufer JA. Alternate routes of invasion may affect pathogenesis of *Salmonella* Typhimurium in swine. *Infect Immun.* 1995;63:2658–2664.

4. Letellier A, Messier S, Lessard L, Chénier S, Quessy S. Host response to various treatments to reduce *Salmonella* infections in swine. *Can J Vet Res.* 2001;65:168–172.

5. Ebner PD, Mathew AG. Effects of antibiotic regimens on the fecal shedding patterns of pigs infected with *Salmonella* Typhimurium. *J Food Prot.* 2000;63:709–714.

6. Mikkelsen LL, Naughton PJ, Hedemann MS, Jensen BB. Effects of physical properties of feed on microbial ecology and survival of *Salmonella enterica* serotype Typhimurium in the pig gastrointestinal tract. *Appl Environ Microbiol.* 2004;70:3485–3492.

*7. Hurd HS, McKean JD, Griffith R, Rostagno MH. Lairage is a hazard for *Salmonella* infection in market swine. *Proc AD Leman Swine Conf.* St Paul, Minnesota. September 2002;223–225.

8. Proux K, Cariolet R, Fravalo P, Houdayer C, Keranflech A, Madec F. Contamination of pigs by nose-to-nose contact or airborne transmission of *Salmonella* Typhimurium. *Vet Res.* 2001;32:591–600.

*9. Rostagno M, Hurd S, McKean J. Are there high and low *Salmonella* prevalence farms? *Proc AASV.* Des Moines, Iowa. March 2004;445–446.

10. Barker IK, Van Dreumel AA, Palmer N. The alimentary system. In: Jubb KVF, Kennedy PC, Palmer N, eds. *Pathology of Domestic Animals.* Vol 2. 4th ed. New York: Academic Press; 1993:2–237.

11. Roesler U, Marg H, Schroder I, Mauer S, Arnold T, Lehmann J, Truyen U, Hensel A. Oral vaccination of pigs with an invasive gyrA-cpxA-rpoB *Salmonella* Typhimurium mutant. *Vaccine.* 2004;23:595–603.

12. Springer S, Lindner T, Steinbach G, Selbitz, HJ. Investigation of the efficacy of a genetically-stabile live *Salmonella* typhimurium vaccine for use in swine. *Berliner Munchener Tierarztliche Wochenschrift.* 2001;114:342–345.

13. Letellier A, Messier S, Lessard L, Quessy S. Assessment of various treatments to reduce carriage of *Salmonella* in swine. *Can J Vet Res.* 2000;64:27–31.

14. Charles SD, Abraham AS, Trigo ET, Jones GF, Settje TL. Reduced shedding and clinical signs of *Salmonella* Typhimurium in nursery pigs vaccinated with a *Salmonella* Choleraesuis vaccine. *Swine Health Prod.* 2000;8:107–112.

*15. Maes D, Gibson K, Trigo E, Saszak A, Grass J, Carlson A, Blaha Th. Evaluation of cross-protection afforded by a *Salmonella* Choleraesuis vaccine against Salmonella infections in pigs under field conditions. *Proc AASV.* Kansas City, Missouri. March 2002;191–194.

16. Baggesen DL, Wingstrand A, Carstensen B, Nielsen B, Aarestrup FM. Effects of the antimicrobial growth promoter tylosin on subliminal infection of pigs with *Salmonella enterica* serotype Typhimurium. *Am J Vet Res.* 1999;60:1201–1206.

17. Shryock TR, Elliott RA, Bennett TH, Basson RP, Bowen RE. Effect of tylosin on an experimental *Salmonella* infection in pigs. *Swine Health Prod.* 1998;6:211–216.

18. Côté S, Letellier A, Lessard L, Quessy S. Distribution of *Salmonella* in tissues following natural and experimental infection in pigs. *Can J Vet Res.* 2004;68:241–248.

*19. Menard J. Strategic control of *Salmonella*. *Proc Pfizer Can Swine Symp*. Vancouver, British Columbia. January 2001.

20. Smith-Palmer A, Stewart WC, Mather H, Greig A, Cowden JM, Reilly WJ. Epidemiology of *Salmonella enterica* serovars Enteritidis and Typhimurium in animals and people in Scotland between 1990 and 2001. *Vet Rec.* 2003;153:517–520.

21. Hendriksen SWM, Orsel K, Wagenaar JA, Miko A, van Duijkeren E. Animal-to-human transmission of *Salmonella* Typhimurium DT104A variant. *Emerg Infect Dis.* 2004;10:2225–2227.

* Non-refereed references.