# Salmonellosis in Feed Yards: Epidemiology, Clinical Management, and Public Health Risk

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## Introduction

Since the advent of the germ theory in the late 1800's and George Soper's[10] discovery of the link the between Mary Mallon's employment history and 7 cases of typhoid fever in early 1907, salmonellosis has presented a diagnostic and clinical challenge to medical practitioners. Salmonellosis is caused by gram-negative, rod-shaped, facultative anaerobic, intracellular bacteria of the family Enterobacteriaceae, genus *Salmonella.*[12] There are at least 2300 serotypes (serovars) of *Salmonella* based on their somatic or O antigens (lipopolysaccharides or endotoxins), flagellar or H antigens, and in some cases virulence or Vi antigens. There is debate as to whether these serovars are distinct species or are a subspecies of one species, *S. enterica*, but most often serovars are considered species.

Most of the serovars of Salmonella are potential pathogens, but approximately 15 are commonly identified in diseased animals. Some Salmonellae are host specific, such as *S. choleraesuis* in swine, *S. typhi* in people[8] and *S. abortus-ovis* in sheep. In contrast others, such as *S. typhimurium*, infect cattle, sheep, goats, swine, horses, poultry, rodents, people, and occasionally dogs and cats.[22] Salmonellosis is a serious zoonotic disease[1] (CDC reported 41,901 cases in people in 1997)[11] and is estimated to cost $0.6 to 3.5 billion[23] in medical expenditures and lost production in the U.S. annually. Consequently, educating clients and their employees about the zoonotic hazards of working with animals during a Salmonella outbreak is as important as treating the animals. Salmonellosis is extremely important to all of the food animal industries because their products are common sources of Salmonella for people.[1]

## Clinical Signs

In feed yards, salmonellosis occurs as three clinical syndromes: septicemic, acute enteritis, or chronic enteritis.[7, 15] The septicemic form is characterized by depression, anorexia, fever, CNS signs, respiratory disease, and sudden death with or without diarrhea. In the acute form, diarrhea is the most common sign. Fever, anorexia, depression, abdominal pain, dehydration, and occassionally mucopurulent nasal discharge and cough occur as well. The diarrhea varies from clear watery to feces that contain blood, mucus, fibrin, and casts of the intestinal lining. Pregnant animals may abort and many other tissues may become infected in the acute enteritis form of the disease. Chronic enteritis is a continuation of the acute form, wherein animals become unthrifty, lose weight, and develop lesions in other tissues. A few animals will have avascular necrosis[17] of the pinnae of the ears, the tail, and the legs distal to the fetlock. The diarrhea of both enteric forms has the putrid odor of decaying tissue.

## Pathogenesis

Infection occurs after the ingestion of feed or water contaminated by fecal material containing Salmonella bacteria; generally a minimum infective oral dose of $10^7$ to $10^9$ is required to infect cattle.[8] The bacteria adhere to the enterocytes and colonize in the distal small intestine or colon. The normal intestinal flora usually inhibit the growth of the Salmonella[5] and prevent their attachment to the entrocyte, but when the normal flora is disrupted, such as occurs with water and feed deprivation during transit,[2] overcrowding, weather distress, or even antibiotic therapy that changes the intestinal flora, host defenses are impaired and colonization occurs.[18] If colonization and multiplication occur, eventually bacteria penetrate the enterocyte. Further multiplication occurs and then the bacteria penetrate the lamina propria. Once the mucosa is penetrated, the bacteria move through the lymphatics to regional lymph nodes either in macrophages or as free bacteria. If additional multiplication occurs in the lymph nodes, septicemia results, with either localized lesions in the tissues or organs such as the liver, spleen, lungs, meninges, mammary glands, and joints or death may occur.[8,22] If the immune system controls

the disease at any step, the animal recovers clinically, but there is potential for chronic infection or the carrier-state to develop. This is especially true if septicemia occurs with infection of intestinal lymphatics or the liver. With the host-specific serovars, a long-term, possibly lifetime, carrier-state may develop. With serovars that are not host-specific, the carrier state is usually much shorter.[12] Finding a host-specific serovar indicates a carrier animal is the probable source, but other serovars can come from any source, such as birds, rodents, or feed.[12] Salmonella survive intracellularly in carrier animals even in the face of high extracellular concentrations of immunoglobulins or antimicrobial drugs.[19] Consequently, elimination of the carrier-state is difficult if not impossible. While it remains to be proven if the carrier-state can be maintained with intestinal infection alone, re-seeding of bacteria from the intestinal lymphatics or the liver/gall bladder can maintain the intestinal infection.

There are 5 common sources of Salmonella infections: 1) animals with the disease, 2) carrier animals, 3) the environment contaminated by diseased or carrier animals, 4) rodents[19,21] and 5) feed. It is important to consider all of these sources when investigating outbreaks of Salmonellosis.

The carrier animal is very important to the epidemiology of Salmonellosis and is probably the source of infection in most feed yard outbreaks. A study of 91 dairies determined that large herd size was an important risk factor for fecal shedding of Salmonella.[9] The authors of that study speculated that herd size was related to the need to bring new animals into the herd and thus increased the risk of introducing animals subclinically infected with *Salmonella*. Feed yards receive new animals almost daily, therefore it islikely that carrier animals reside in feed yards. An asymptomatic carrier can shed $10^9$ *S. dublin* per day ($10^6$ per gram of feces),[19] or several infective doses. Once other animals become acutely infected, numbers of bacteria in the environment increase dramatically because animals with clinical signs may shed $10^8$ to $10^{10}$ of Salmonella bacteria per gram of feces,[3,19] resulting in exposure of more animals. When this occurs in feed yard hospital pens, exposed animals may carry the organism back to their home pen.

It is clear that feed yards should be prime candidates for salmonellosis because of the epidemiology of the disease. Cattle that are exposed, commingled, newly weaned, deprived of water and feed during transit, and placed in a new environment are certainly under stress. Under such conditions, it is expected that carrier animals would shed Salmonella, and other animals would be susceptible to infection because of changes in their immune system and intestinal flora and exposure to other diseases. Dual infections of Salmonella and bovine virus diarrhea virus (BVDV) occur frequently[12] and BVDV may exacerbate the effects of Salmonella during outbreaks.[24]

Cray[4] *et al* surveyed 100 feed yards by collecting fecal samples from floors of 25 pens of newly arrived animals (approximately 8 days on feed) and compared them to 25 pens of animals that been on feed for a longer period of time (approximately 180 days on feed). They reported that 38 % of the feed yards were culture positive for Salmonella. Interestingly, Salmonella was isolated from 3.5% of newly arrived cattle and 7.4% of samples from cattle that had been on feed longer. Those authors suggested that more animals become infected, or shed the organism as the animals are penned together. They also concluded that infection in 3.5% of the population results in spread the organism throughout the pen.

In another study, Galland[6] *et al* collected fecal samples from animals when they arrived at the feed yard. They isolated Salmonella from 40% of a subset of 120 animals selected from 2731 steers known to be involved in an outbreak of Salmonella approximately 45 days prior to arrival. By day 60 of the feeding period fewer than 1% were culture positive and the cecal-colon content was not culture positive at slaughter. In these same animals, 38% were ELISA positive for Salmonella serogroup B on arrival, 53% were positive at day 60 and 38% were positive at slaughter. Although there were no clinical signs at arrival, a significant percentage were poositive, however signifcant recovery occurred by day 60. Both the culture and ELISA data indicate that recovery from the disease does occur, at least in some animals, and that feed yard cattle will have fewer infections at slaughter than on arrival in the feed yard.

There could have been many differences in these two studies. The first to consider is the prevalence of infection at arrival and the time between the acute outbreak and arrival at the feed yard in Galland's study. Since cattle arrive in feed yards from different sources, differences in prior exposure and prevalence of disease would be expected. It would not be surprising that there would be differences between groups in prevalence of disease 60 or 180 days later. Thus, if prevalence on arrival was very high, prevalence would be expected to be higher in the same animals 60 or 180 days later compared to animals having a low prevalence at arrival.

## Clinical Management

Controlling salmonellosis in feed yards is difficult, feed yards cannot operate as all-in-all-out systems, closed herds, or prevent birds or rodents from having access to feed bunks. Therefore, prevention must focus on isolation of acutely infected animals and minimizing contamination of feed, water, hospital equipment, and

the environment, especially hospital buildings, sick pens and receiving pens.

Clinically recovered animals continue to shed Salmonella bacteria for several weeks. During outbreaks, establishing recovery pens where animals can recuperate for an extended period of time minimizes contamination of the feed yard environment and reduces horizontal transmission of the disease. Salmonella can survive for several months in moist conditions out of direct sunlight[19] so these recovery pens should not be used by other animals until they are thoroughly cleaned. In addition, rations can be adapted to the needs of animals recovering from enteric infections.

Cleaning, disinfecting, and drying of hospital facilities and equipment prevents explosive outbreaks. Chlorine bleach, or phenol disinfectants will control Salmonella in the environment. Culturing is an effective way to monitor the environmental sanitation program. Instruments contaminated with nasal discharge, blood or feces should be washed and disinfected with quaternary ammonium or iodine-based compounds between treatment of each animal.

Treatment is directed at dehydration, endotoxic shock and septicemia. Fluid therapy to correct dehydration, metabloic acidosis, and shock are often needed. In feed yards fluids are often given orally with good response if done early in the course of the disease. Antimicrobial therapy is controversial and may not be necessary with enteric infections, but because septicemia frequently occurs with salmonellosis, treatments usually include antimicrobials based on antimicrobial sensitivity patterns. Kansas State University reported that more than 90% of bovine isolates are sensitive to ceftiofur[12]. Neomycin is approved for oral use in cattle, but only 50% of the isolates are sensitive. Most are sensitive to fluroquinolones, but fluroquinolones are not approved for treatment of salmonella and it is illegal to use fluorquinolones extra-label in food producing animals in the United States.

The use of salmonella vaccines has been disappointing in feed yard cattle.[7] In addition, adverse reactions have been reported with some whole-cell bacterins caused by the gram-negative endotoxin.[19] Since Salmonella are often facultative intracellular bacteria[17],[19] vaccines must produce both humoral and cellular immunity to provide protection. Genetically altered, modified-live vaccines that stimulate both humoral and cellular immunity have shown positive results.[20]

Competitive exclusion cultures are commercially approved for use in the poultry industry.[13] There is evidence that these cultures decrease mortality,[13] cecal colonization of Salmonella in treated birds,[25] and contamination in the processing plant.[14] Similar research studies are currently being conducted in the swine and cattle industries. Results from a recent challenge study in swine identified a decrease in numbers of Salmonella recovered from multiple tissues when quantitive cultures were performed.[16]

## Public Health Risks

It is impossible to eliminate the carrier-state of Salmonella infected cattle and therefore to obtain Salmonellae-free foods of animal origin.[1] Education of food handlers in homes and restaurants on correct procedures for cooking, refrigeration, re-heating, environmental sanitation and personal hygiene[1] is currently the best prevention. The risk of Salmonella is minimal when foods are properly cooked, handled or pasteurized.

## Summary

Salmonellosis is a severe enteric and septicemic disease affecting animals and people. Infection occurs by the fecal-oral route that often involves a carrier animal and distress, such as marketing or shipping, prior to infection. Clinical management involves isolation of sick animals, sanitation of facilities, equipment and instruments, and supportive and antimicrobial therapy of affected animals. Vaccines currently are of limited value but with advances in technology could provide future help. Competitive exclusion products may provide a future avenue to reduce infections. Finally, there is a potential public health risk to people working with animals, especially when outbreaks occur and when food is improperly prepared or handled.

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