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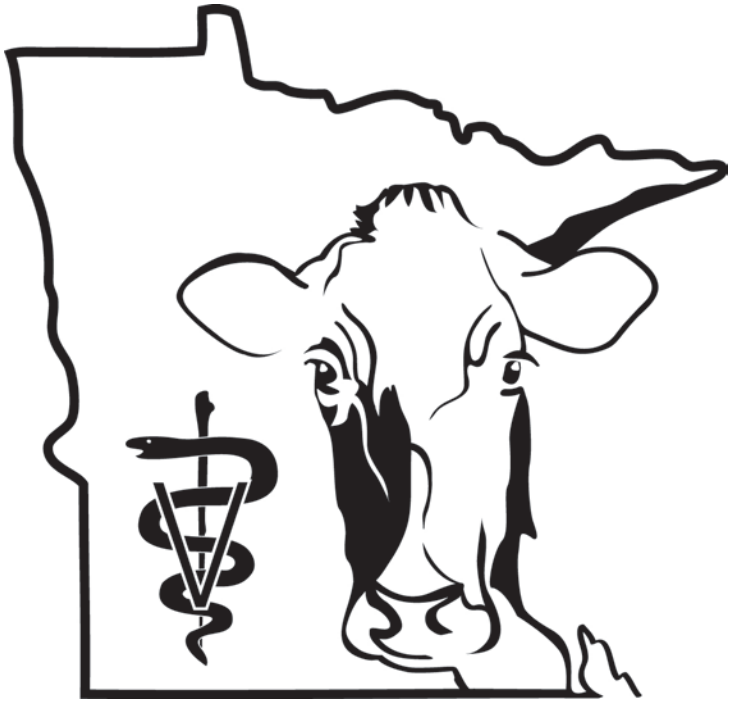

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Tackling Herd Salmonellosis Problems

Introduction:

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Much information on tackling bovine salmonellosis is in practitioner literature (e.g., McDonough, 1995). Some of this material is from Minnesota, where much salmonellosis work has been done, and has been presented previously at this conference (e.g., Bender, 1994, Robinson and Bender, 1994). With this in mind, the following emphasizes those factors not discussed by these previous authors and is oriented more toward the non-host adapted salmonella (e.g. *S. typhimurium*). The host-adapted salmonella (*S. dublin*) adds the additional complications associated with persistently infected carrier cattle, particularly those of congenital transmission and abortion.

Far from being a simple organism with simple functions, salmonella interact with the environment and their animal hosts in very complex ways that are only beginning to be understood. Because of the following characteristics, salmonella is a very worthy adversary for the veterinary practitioner.

Due to research findings, the nomenclature of salmonella is changing. Molecular biology research is providing a better understanding of the organism, one result being a better understanding of how the various serotypes are genetically related. While the serogrouping was based on the phenotypic expression of particular O and H antigens, molecular methods based on the organism's genotype are under development. These molecular-based classification systems will likely be more rapid and repeatable than methods based on phenotype. In the meantime, the same organism may have two different names in the scientific literature. The common salmonella serotypes of concern to dairy practitioners (e.g. *S. anatum, dublin, montevideo, typhimurium*) are now classified into a single species *Salmonella enterica* and are subclassified by their traditional serovar name. Confusion will rein for some time because other salmonellas, such as *S. enteritidis*, are also classified under *S. enterica*. In the research literature what was *S. typhimurium* before the renaming is now *Salmonella enterica* serovar Typhimurium or *S.* Typhimurium rather than *S. typhimurium*. Recognizing this renaming by those searching the clinical literature will become more important as more papers are published using this new terminology.

Ten characteristics of Salmonella and salmonellosis to keep in mind:

1. Salmonella infection of a farm is maintained primarily by a transmission of the agent from the feces of infected animals to the mouth of susceptible animals. Transmission by inhaled aerosols as well as by other routes (e.g., ocular, via teat streak canal) occurs, but less frequently.
2. Salmonella infection and subsequent clinical disease (the two are not synonymous) is a result of:
   a) The innate resistance of the host animal.
   b) The infectious dose received by the animal.
   c) The infectivity and virulence of the particular strain of the organism.

   The dairy producer has the most impact on the salmonella cycle through maximizing a) and minimizing b) (and probably in that order) but cannot change c) once the strain is on the farm.
3. Salmonella infects virtually anything in the dairy environment that has an intestinal tract.

   Besides cows and calves, salmonella infections occur in feral cats, rodents, birds, waterfowl, flies, humans, fish, and indigenous wild mammals (raccoons, porcupines, deer). Under the right conditions, any of these species (even flies) can serve as biological multipliers of this organism.

   Initiate control programs for rodents, flies, nuisance birds, and feral dogs and cats. Rodent proof and bird proof feed storage and cattle housing facilities.

4. The majority of salmonella infections in a herd over time are subclinical; the clinical infections are only the tip of the iceberg, even during outbreaks of clinical disease.

   In an outbreak, handle all animals as if they were shedding, not just the sick ones.

5. Septicemic animals shed the agent in oral and nasal secretions and urine as well as feces. These animals don't necessarily have clinical signs associated with enteric salmonellosis at the time.

   Such animals are very dangerous because they contaminate water bowls, nipples, oral treatment equipment, and human hands. Often this equipment (e.g. balling guns, esophageal feeders) is used without proper sanitizing between animals and transmits the infection to other animals that are in a most susceptible state.

   This sanitation failure is likely to be the biggest weakness of many farm treatment programs.

6. Salmonella has a complex relationship with its animal host, which is only beginning to be understood.

   For example, researchers have found that *S.* Typhimurium sends a protein signal through the wall of a nearby intestinal cell of the host. In response to this signal, the host cell flattens its brush border and builds a large, projecting pedicle with an interior cytoskelton. Salmonella then attaches securely to this pedicle. Salmonella and other pathogens are able "trick" the intestinal mucosal M cells into ingesting them, which gives the bacteria direct access to the reticuloendothelial system but are protected from it.

   Because of this complex relationship, bacterins simply targeted at producing antibodies against antigens are not likely to work. Bacterins targeted at specific parts of this relationship, such as blocking the signalling, may provide protection.

7. Salmonella are a small part of an extremely competitive, complex, dynamic environment in most intestinal tracts and this competition is a very important part in resistance to infection.

   An understanding the aspects of intestinal microbiology is helpful in developing strategies to prevent salmonellosis are useful For a primer on mammalian intestinal microbiology, see Drasar and Barrow (1985). Calves are born with a sterile intestinal tract that is at a neutral pH and provides an excellent environment for bacterial growth. Succeeding waves of microflora are established that change as the calf develops and its diet changes. The lactobacilli are normally the first to populate the tract. The strains of lactobacilli change with time and only those with specific characteristics that enable them to bind to the mucosal surface persist. The presence of these lactobacilli increases the resistance of the calf to salmonella infection. Over time, other flora populate the gut until over 400 species of bacteria are present, the numbers of each that are present varying along the length of the gut. The gram negative fecal coliforms represent less than 1% of the mass of bacteria present in the normal animal's gut with most being strict anaerobes. Many of these other species are very important in resisting salmonella infection. Normal mice require 10,000-fold the infective dose to establish salmonellosis as gnotobiotic mice or mice treated with streptomycin, which have no or altered competing flora. Because most of these other species are more sensitive to antibiotics than are salmonella, the use of antibiotics precipitates clinical salmonellosis in sub-clinically infected humans and animals by allowing overgrowth of the salmonella. Poorly absorbed oral antibiotics and antibiotics that are secreted into the gut will particularly predispose the gut to an overgrowth of salmonella by killing the more sensitive competitive microflora. Some research shows that even those antibiotics to which the salmonella are sensitive will cause a cessation of salmonella shedding and that shedding resumes when the antibiotic is withdrawn.

   This suggests that antibiotics should be used with considerable prudence in salmonella-infected herds and then only in those cases with systemic involvement.

8. Salmonella are killed by exposure to the volatile fatty acids of fully functioning normal rumens.

   The level of VFAs in rumens of cattle on continual full feed are toxic to salmonella. However, if dry matter intake drops for any reason, the VFA levels decline rapidly. A drop in DMI may be precipitated when animals don't have regular access to feed, such as during transport through the marketing system, for physiologic reasons such as impending parturition, subclinical ketosis and hypocalcemia, and sudden ration changes or ration maladaptation, and for husbandry failures, such as inadequate bunk and pen space and mixing submissive heifers with dominate cows at parturition.

   Ration fats may also encapsulate the bacteria, protecting them from the rumen VFAs. *S.* Typhimurium DT104 (see below) is rumored to be more acid resistant.

   Maximize rumen function by maximizing a consistent dry matter intake in periparturient and early fresh cows.

9. Salmonella survives for long periods under environmental conditions common on the dairy farm.

   Salmonella have several different survival mechanisms (reviewed by Foster and Spector, 1995). These systems enable the organism to survive sudden environmental changes and to survive for long periods in different environments. Moreover, the pathogen can quickly turn these different systems on and off in response to changing environments. Once some of these systems are turned on in the dehydrated organism, the organism becomes much more resistant to environmental factors such as heat that would otherwise kill it. As a result, it survives very well on surfaces, in dust, and in dried manure that are protected from sunlight.

   These survival times are very long if the organism isn't exposed to sunlight. In an experiment that simulated a barn floor under defecating cows, salmonella survived for 5 ½ years (Forshell and Ekesbo, 1996). These researchers found *S.* Typhimurium in an empty slurry pit that had not been used for 4 years.

   Salmonella survives in lagoons and can be recycled to the herd in the flush water.

10. Salmonella replicates in moist environments (< 85% dry matter) even with scarce nutrients.

    Salmonella replicates rapidly in mixed feeds and on surfaces that have been washed but have not been adequately sanitized. Other workers found that the bacteria survived for at least 119 days in contaminated pond water.

    Salmonella replicates very well in composted manure solids used for bedding once it becomes wet and contaminated.

Keeping the above factors in mind when working with a farm facing a salmonellosis problem will enable you to detect the weak points in the management and facilities of that farm and to determine the best places to begin breaking the salmonella cycle.

S. Typhimurium DT104: An emerging problem for the bovine industries

The following is specific information about an emerging strain of salmonella that has serious animal and human health as well as potential foreign trade implications. The first human outbreaks of this agent in the U.S. were reported within the past year. The Field Disease Investigation Unit at Washington State University has been investigating farm outbreaks of this agent for several years. You and your clients will likely be hearing a lot more about this agent in the near future. Some have declared that it will become the bovine equivalent of the *S. enteriditis* problem in the poultry industry.

An antibiotic resistant strain of salmonella, *Salmonella enterica* serovar Typhimurium variant DT104 (*S.* Typhimurium DT104), commonly R-type ACSSuT, is an emerging pathogen for humans and farmed animal species in the U.S. (Besser et al. 1997). Because people in contact with livestock are at markedly higher risk for infection by this agent (Besser et al. 1997, Wall et al. 1994, 1995), it is an occupational disease for farm workers and the farm family. This strain is of particular concern in humans because cases are reported to have a hospitalization frequency that is double, and a mortality frequency that is ten times that experienced with other salmonella (Wall et al. 1994). Veterinarians dealing with farms infected with this strain are remiss if they do not warn people associated with the farm about the hazard presented by the exposure of the very young, the elderly, people who are immunocompromised and those taking antibiotics to animals shedding this organism. Likewise, consumption of raw milk from the farm should be discouraged.

The epidemic strain of *Salmonella* Typhimurium DT104 is identified by the following characteristics: it is phage type 104, has an antibiotic resistance pattern characterized by resistance to ampicillin, chloramphenicol, streptomycin, sulfonamides and tetracycline (R-type ACSSuT) and a plasmid profile characterized by the presence of a single 60 megadalton plasmid. The R-type ACSSuT is unique in isolates of *S. typhimurium* and can be used as a method for screening and preliminary classification of isolates as the epidemic strain. In humans, the CDC reports that R-type ACSSuT has increased from 9% of *S.* Typhimurium isolates in the U.S. in 1990 to 32% in 1996 (Anon., 1997). The first group outbreak in the U.S. was detected in late 1996. In a bank of *S.* Typhimurium isolates collected from Northwest animals, we found that R-type ACSSuT was absent in cattle isolates obtained prior to 1986, comprised 13% of the isolates prior to 1991 and 64% subsequently (Besser et al. 1997). We have evidence that *S.* Typhimurium with this R-type infects a broad range of species in the Northwest, having isolated it from the cow, horse, goat,emu, cat, dog, elk, mouse, coyote, ground squirrel, raccoon, chipmunk and birds (pigeon, starling, pine siskin).

This variant was first recognized in England in 1984 (Threlfall et al. 1994) and its proportion of salmonella isolated from man began rising rapidly in the 1990's, becoming second only to *S.* Enteriditis. This variant is unusual in several respects. The genes encoding the unique antibiotic resistance are integrated into the chromosome, meaning that this resistance is likely to be retained even in the absence of the selective pressure of drug use (Threlfall et al. 1994). In Britain, this strain appears to have a significantly higher human morbidity and case mortality than other strains (Threlfall et al. 1996, Wall et al. 1994, Wall et al. 1995). Of further concern is that *S.* Typhimurium DT104 are emerging in Britain that are resistant to fluroquinolones (Threlfall et al. 1996), which are important drugs for the treatment of invasive salmonellosis in humans. In both Britain and the U.S., human cases are significantly associated with contact with cattle (Besser et al. 1997, Wall et al. 1994,Wall et al. 1995) and thus human disease is a particular risk in farm and rural families. This has very serious implications for antibiotic use in food animals, particularly of fluroquinolones, by the veterinary profession.

After *S.* Typhimurium DT104 has been introduced into a herd, the clinical course is highly variable. In some herds a significant proportion of the cows, including mid-lactation cows, and an even larger proportion of the calves are affected. In other herds only a few postpartum cases are observed or only cases in calves even though the environment is heavily contaminated (Anon., 1996). An *S.* Typhimurium DT104 enteric infection persisting for four months (Anon., 1995d) and udder infection persisting for eight months through a non-lactating period (Sharp and Rawson, 1992) have been

documented. The reasons for the variability have not been delineated, but the existence of such a wide variation of clinical syndrome suggests that certain factors of a farm's environment and management modulate the herd's experience with this agent.

Active surveillance and investigation of farm outbreaks of this agent in British herds by government personnel has been occurring since the agent was first recognized and reports of their findings indicate the infectivity of the agent and its risk to livestock, non-farmed animals, farm families and the rural environment. In one outbreak, 15 isolations were made from 22 normal in-contact calves, indicating that subclinical infection of the bovine may not be uncommon (Anon., 1995a). In another, index bovine cases shed the agent for four months (Anon., 1995d), showing the persistence of infection in individual cattle. Persistent infection in a herd has been documented over 8 months (Sharp andRawson, 1992). The introduction of a large number of feral cats to control a rat infestation was associated with one livestock outbreak (Anon., 1995c). The agent was isolated from the cat feces, suggesting that they were an established part of the salmonella cycle on the premises. The agent was isolated from another cat with intermittent bloody diarrhea but the cat was not associated with cattle (Anon., 1994a), from pigeons (Anon., 1994b), and from rabbits, one of which was associated with a secondary human case (Anon., 1994c). Cats also have been implicated as a common source of infection for humans (Anon., 1997, Low et al. 1996, Threlfall et al. 1996). Another human outbreak of approximately 20 cases was traced to milk from a producer whose bulk tank milk was positive for the agent (Anon., 1995e). In other cattle outbreaks, the agent was isolated from a healthy dog (Anon., 1994d), healthy ducks (Anon., 1995b), and from pond water accessible to cattle (Anon., 1995d). Two cattle outbreaks were associated with exposure to human sewage, suggesting that waterborne routes may permit transfer from humans to cattle (Anon., 1995d, 1995e). A large number of starlings were noted in one outbreak, leading investigators to suggest their role in heavily contaminating the environment and point to their potential danger to surrounding farms (Anon., 1996). The agent was found in the bird feces from various places on the premises, within the silage and throughout the buildings even though only two clinical cases had been noted in the cattle.

In a case-control study of *S.* Typhimurium DT104 in British cattle herds, significant associations were found between clinical case occurrence and seven risk factors (Evans, 1996, Evans and Davies, 1996). These were being a cattle dealer (OR = 14.25) as well as a farmer, introducing newly purchased animals (OR = 2.51), being in the calving period for seasonally calving herds (OR = 2.48), birds accessing stored feeds (OR = 1.67), cattle being housed (OR = 1.51), lack of isolation facilities for sick animals (OR = 1.51) and cats accessing stored feeds (OR = 1.35). In a pilot study, we found that*S.* Typhimurium DT104 replicated in 9 of 10 mixed rations from dairy farms.

Information from investigations of outbreaks due to other strains andserotypes may be useful for practitioners. Investigators have reported that animals other than cattle are associated with the salmonella cycle of this and other serovars. These include an association between various bird species contaminating feeds and the transmission of salmonella to cattle (Coulson et al. 1983, Glickman et al. 1981, Johnson et al. 1979, Tizzard et al. 1979). Rodents have been implicated in outbreaks in a dairy herd (Tablante and Lane, 1989), in a beef herd (Hunter et al. 1976) and in poultry flocks (Davis and Wray, 1995, Henzler and Opitz, 1992). Live-trapped raccoons have been reported to harbor *S.* Typhimurium (Morse et al. 1983). Insects may also be vectors (Devi and Murray, 1991, Kopanic et al. 1994). Flies may function as a biologic vector as well as a mechanical vector, with*S.* Typhimurium replicating in flies under the right conditions (Greenberg et al. 1970).

Other factors in the farm environment may also perpetuate the salmonella cycle. In a review of salmonellae in the environment, Murray (1991) states "the predominate feature of Salmonella spread is human influence on the natural environment, including animal management practices, waste management and effluent control, all which contribute significantly to the spread of salmonellae." The use of recycled flush water may have maintained it on one dairy farm (Gay andHunsaker, 1993). For other strains of salmonella, the agent has been shown to persist in cattle or the farm environment for many months or even years, sometimes persisting after the clinical syndrome has ceased (Gay and

Hunsaker, 1993, Giles et al. 1989, Jones et al. 1983, Richardson, 1975, Taylor, 1979). In one study, a strain of *S.* Typhimurium (not DT104) persisted in a herd for 3.5 years (Giles et al. 1989).

The avian species are among the many animal species that have been identified as susceptible to infection and could be involved in the spread and survival of *S.* Typhimurium DT104. The mobility of avian species makes them of particular concern if they are carriers and shedders of *S.* Typhimurium DT104 for extended periods. As birds follow their seasonal migration patterns and mating instincts, they could potentially disperse *S.* Typhimurium DT104 over wide geographic regions. Of particular interest are species that are commonly found on or near farms and are known to feed on dead animal tissue (e.g. dead carcasses, placentas or mucous shreds), animal feeds or on material in livestock droppings (e.g., undigested feed, invertebrates living off of or in the dung). The Corvidae (crows, ravens, magpies), known carrion feeders and prevalent in the farm environment, could readily ingest such animal tissues and become infected. Starlings, blackbirds and pigeons are common pests on many farms and feed either directly from feed bunks or search for food in livestock droppings. Feeding on the later is likely a means of ingesting large numbers of fecal microorganisms. Infected starlings, blackbirds or pigeons could readily contaminate cattle feed as well as the environment.

Sampling rodents and birds on a farm to determine if they are part of the salmonella cycle on that farm is not as simple as it first appears. Fairly large rodent populations can be present before their signs are obvious. Rodents resident in farm buildings and feed storage areas can be captured using Sherman live traps baited with peanut butter and rolled oats (Schemnitz 1994). These traps often work best if left baited and open for several days in areas frequented by the rodents before they are set. Rodents commonly defecate in the trap, providing fresh fecal samples for analysis. Birds can be captured using simple funnel traps baited with grain (Schemnitz 1994). Place traps at sites where sparrows, pigeons, and starlings are observed feeding on the farm. Similar to rodents, trapped birds will commonly defecate in the trap, providing fresh fecal samples. Trapped animals can be rapidly and humanely euthanized by placing the entire trap in a plastic bag and filling it with pure CO_2 from a CO_2 fire extinguisher. In Washington, trapping introduced species (e.g. old world mice and rats, starlings) does not require a permit but trapping indigenous species does.

We do not know what the future course of this strain will be and its impact on the bovine industries will be. What makes DT104 different from other *S.* Typhimurium strains, if indeed it is different? It sure appears to be spreading like it is different. Is a lower infectious dose required, is it shed in higher numbers by infected animals or does it survive better in the environment? How long and where does it persist on infected dairies? What are the best ways to determine a herd's infection status? Testing cows or calves? Performing conventional bacterial culture, using molecular based methods, running serological ELISAs or a combination of these methods? How often and on what samples? There is much we need to learn about this infectious agent to prevent its spread and to eradicate it from infected premises.

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