

SALMONELLOSIS IN CATTLE: A REVIEW

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Salmonella spp. infection occurs when a susceptible animal ingests the bacteria. Dairy cattle ingest feed or water that has been contaminated with feces from animals shedding the organism. Salmonellosis has a wide spectrum of manifestations in cattle. Asymptomatic, mild clinical or fulminant bacteremia/septicemia and endotoxemic infections can occur. The manifestations vary with virulence of the strain, infectious dose, and immunity of the host. On many dairies, salmonellosis is an opportunistic infection. Recent large, multi-state studies within the US dairy industry suggest that approximately half of all herds will have at least one culture positive cow on any one day, and that approximately 1 in 20 apparently healthy dairy cows will be shedding *Salmonella* at any one time in the US. These numbers increase when researchers refine their sample populations to sick cows, cull cows and cows at slaughterhouses. There appears to be geographic variation in the level of feedstuff contamination whereby temperate, higher rainfall dairy areas have higher levels of *Salmonella* contamination of forages and other ration components than do the harsher Northern climates in the mid-Western and Eastern US. It is apparent from recent research in the Mid-Western US that organic dairies do not differ significantly from larger free stall farms in the prevalence of environmental and individual cow and calf *Salmonella* infection. Environmental isolation of *Salmonella* organisms is frequently possible (about 40-50% of farms) even on farms with no known history of clinical Salmonellosis.

WHERE DOES IT COME FROM?

On a dairy, the source of the infection is usually feces from infected cows. It may be difficult to tell which cows are shedding bacteria because asymptomatic and subclinically affected cows can shed as many organisms in their manure as the cows that are sick with salmonellosis. Other sources of infection may be rodents, birds (including waterfowl), flies, feral cats, dogs, raccoons and, rarely, people.

- **Fecal – oral transmission**
- **Aerosol transmission – in confinement facilities**
- **Saliva and nasal secretions – especially in shared waterers**
- **Milk and colostrum**

WHY DOESN'T IT RUN ITS COURSE AND END?

Salmonella outbreaks commonly last several months. Protracted problems can be the result of a number of factors – persistence in the environment, persistence of risk factors, carrier state or

prolonged shedding, or reinfection of susceptible animals. On some dairies, particularly those with large numbers of cattle, the disease may become **endemic**.

◆ **Reservoirs**

a) Feces

b) Feed:

- Under appropriate moisture, temperature and pH conditions, the organism can replicate about every 30 minutes. *Salmonella spp.* can also be introduced in contaminated feeds. The ban on feeding ruminant protein has reduced this risk but *Salmonella* can contaminate animal and vegetable fats fed to cattle

c) Water

d) Sick cows and asymptomatic shedders:

- The most recent studies show that approximately 5% of apparently healthy dairy cows may be shedding the organism in their feces and that approximately 20% of all sick cows on the cull list shed *Salmonella spp.* This makes the on-farm location of these cattle as well as cattle with obvious diarrhea very important because clinically affected animals shed more than 10^{14} organisms per day (infectious dose 10^9 - 10^{11} organisms).

◆ **Survival Outside Animals:**

In the environment, it survives for 4 to 5 years in water, soil, dust, moist areas out of direct sunlight and on or within foods. *S. dublin* can survive in dry feces for over a year, however freezing at -4 F kills 85% of *Salmonella spp.* in 2 days.

Crops irrigated with salmonellae-contaminated wastewater will lead to contamination of forages and water sources.

Rendering kills salmonellae; however post processing contamination accounts for 50% of contamination of rendered feed products.

◆ **Carrier Animals:**

Infection with a host-adapted *Salmonella* strain (*S. dublin* in cattle) can result in a cyclic, endemic disease that is maintained on a farm by carrier animals shedding in the feces and/or milk. The carriers can shed constantly or intermittently.

Cattle – chronically affected carriers may shed 10^8 to 10^9 salmonellae per day in feces and 10^2 to 10^5 organisms per ml of milk.

Since *Salmonella spp.* can cross from one species to another, other potential animal sources include dogs, birds, cats, people and pigs. Flies also pose a risk for spread.

Fomites – feces and oropharyngeal secretions on the following can be significant sources of cross contamination between cattle:

- Medication equipment – esophageal feeder, stomach tubes, buckets, stomach pumps
- Calf nipples
- Nose tongs
- Exam gloves
- Coveralls
- Boots – walking through feed areas
- Calves suckling
- Equipment - scoops and loaders that handle manure, wheels track manure from pen to pen, buckets

◆ **Risk Factors:**

- More frequent in dairy herds than beef herds, mixed dairy and beef herds and calf herds
- Outbreaks more common in calving season, and also appear to be more common in the summer months
- Outbreaks more common in large herds
- Purchasing cattle from “dealers” rather than source herds
- Expansion
- Confinement
- Sick and calving cows comingled
- High density of feral cats
- Wild birds having access to feed storage facilities
- Antimicrobial use prior to or at the time of exposure
- Use of flush water systems
- Feeding brewers’ products, animal by-pass protein sources, vegetable or other fat sources to lactating cows
- Allowing commodity storage areas, particularly those that drain poorly or can retain moisture, to become wet

◆ **Clinical Signs**

- Pyrexia
- Lethargy, depression
- Decreased milk production
- Anorexia
- Dehydration
- Increased salivation
- Diarrhea progressing to dysentery
- Clinically inapparent carriers can shed for up to 18 months (non *S. dublin* carriers)

◆ **Treatment (or fighting your way out of an outbreak!)**

- **Supportive care applied early is the most effective in limiting the course and severity of disease. A rise in rectal temperature often precedes the diarrheic episode by 24 to 36 hours.**

Fluids – oral or IV, with electrolyte supplementation

NSAIDs

Probiotics

- **Prudent (maximize the therapeutic effect while minimizing the development of resistance) antimicrobial use**

Susceptibility patterns – ampicillin, ceftiofur, trimethoprim-sulfonamide combinations, fluoroquinolones, and florfenicol (some of the isolates highly resistant to chloramphenicol are florfenicol susceptible)

Pharmacokinetic properties - parent compound vs. metabolites

Pharmacodynamic properties – tissue penetration, intracellular effect

Will antibiotics make a difference? In adult cattle, antibiotics may improve recovery and lessen the severity of the disease but will likely extend the duration of shedding. Calves and young stock are especially prone to becoming bacteremic, but subtherapeutic use of antibiotics in the milk replacer may actually make the disease worse.

Disseminated, invasive or systemic disease (how do you tell?)

- Severe clinical signs of endotoxemia : fever, tachycardia, tachypnea, scleral injection, weakness, ruminal stasis, leukopenia (early), and leukocytosis (later), frequently with a left shift
- Death in some animals
- Pneumonia, polyarthritis
- Post mortem findings are not restricted to dilation, edema and hemorrhage of the bowel and mesenteric lymph node enlargement. There may be enlargement of the spleen, renal infarcts and gallbladder inflammation. Ecchymotic hemorrhages throughout the carcass and pneumonia may also be present

Antibiotics are not likely to affect the carrier state.

Economics

◆ **Testing - Strategy, Monitoring Device or Costly Data?**

a) Cultures

- **Fecal samples:**
Pool samples from several cows
Culture at least 20% of any group
Sequential samples on same animal
Culture from semi-formed feces (better sample than very liquid feces)
To define the true infection status of apparently healthy animals, it is necessary to perform multiple cultures for 3 to 6 months to distinguish convalescent animals from carriers.
PCR based testing is expensive but can be applied to feces and tissues.
- **Environmental samples:**
Locate laboratories with experience, interest or expertise in environmental samples and make prior arrangements for transport and delivery of your samples. Drag swab samples can be very useful when placed in appropriate transport media and delivered to the lab within 6 hours of collection. Some labs are capable of using media that can neutralize the affect of disinfectants.
- **Feed samples:**
Contact a lab with expertise in this area. Most samples can be delivered without special transport media but they may require refrigeration.
- **Use cultures to find the source of infection!**

b) Serovars of Salmonellae – characterizations based on phenotypic expression of O and H antigens

2,200 serotypes; 2% responsible for 80% of disease

95% associated with serogroups B, C, D and E

Type B – Typhimurium, Agona

Of Recent Concern: *S. typhimurium* DT 104 really is *S. enterica* var Typhimurium DT104. It is a phage type DT (distinguished type) 104, with an antibiotic resistance gene that is chromosomally coded and involves integrons. It has a single 60-megadalton plasmid that gives it a unique plasmid profile. Resistance to ampicillin, chloramphenicol, streptomycin, sulfonamides and tetracycline gives it its name - R-type ACSSuT. The R-type ACSSuT pattern is considered a good marker for phage type DT-104 in the US and is the test most commonly used in this country. R-type ACSSuT pattern is not always DT-104. Also not all DT-104's have the same resistance pattern. The resistance to chloramphenicol includes florfenicol. It was first detected in cattle in the UK and NW US at about the same time in the late 1980's. DT-104 is not a superbug per se but the case fatality rate among cattle and calves in a case-control study in the UK was 40-60% (higher figure in calves). Infected animals may shed in higher numbers and there are clinically normal carriers. There is some debate whether the prevalence of this serotype is increasing (appears so in the UK) or whether it peaked in 1995 and has declined since. In 4 of 5 outbreaks in the US, association was made between cattle or consumption of dairy products and the people infected. This phage type has been isolated from cattle in Wisconsin.

Type C – Newport, Montevideo, Kentucky, Infantis

Type D – Dublin

Epidemiologically, this isolate was not common outside of CA until the late 80's, emerging for the first time in NY, PN and OH in 1988. Unlike other serotypes, *S. enterica* Dublin affect older calves (8 weeks of age or older) which is atypical for salmonellosis, presents as pneumonia and septicemia rather than the primarily diarrheal syndrome that is more commonly recognized.

Type E – *anatum*

The common salmonella serotypes of concern to bovine practitioners – *S. Anatum*, *Dublin*, *Montivideo*, *Typhimurium*) are now classified into a single species, *Salmonella enterica*. For example, what was *S. typhimurium* is now *Salmonella enterica* serovar *Typhimurium* or *S. Typhimurium*.

OPPORTUNIST OR PRIMARY PATHOGEN?

In dairy cattle, salmonellosis commonly occurs close to parturition. In this circumstance, it is frequently opportunistic, riding on the coat tails of concurrent disease, dietary stress and the natural depression of immunity at this time of the cow's cycle. Decreased dietary intake influences the growth of ingested salmonellae in the rumen. High concentrations of VFA's and the resultant acidic pH inhibit growth. Feeding after starvation causes salmonellae to multiply. Certain serotypes are associated with primary infections. These are *S. Typhimurium*, *S. Dublin* and *S. Montivideo*. This temporal association underscores the importance of not housing sick, recently fresh cattle close to late pregnant or early lactation, apparently healthy cattle.

Control Strategies:

The strategies implemented should be prioritized and focused on minimizing the source of infection and maximizing host immunity.

- **Adopt an all in – all out system in calf and heifer raising facilities.**
- **Maintain a closed herd or make purchases from low risk herds.**
- **Manage new additions to minimize stress and infection of residents.**
- **Minimize stress by feeding good rations, providing adequate time and space for transitions, and maintain clean, uncrowded maternity pens.**
- **Use different facilities for calving cows and sick cows.**
- **Avoid adult to calf contact. Isolate heifers from the lactating herd.**
- **Disinfect waterers in high risk areas (dilute bleach twice daily).**
- **Scrape manure, remove organic debris, disinfect clean, non-porous surfaces and expose to sun or UV light.**
- **Minimize fecal contamination of feedstuffs, feeding surfaces, water troughs and equipment.**
- **Drain and level areas that collect water.**
- **Allow no access to pond water or feeding areas cohabited by birds and waterfowl.**
- **Isolate the entire group in which affected cows commingle.**
- **There should be no shared bunk spaces, water source, feeding or manure handling equipment. Left-over TMR from the cows should not be fed to the heifers.**
- **Segregate *Salmonella* test-positive cows at calving.**
- **Do not use colostrum or milk from test-positive cattle.**
- **Manure-handling equipment is not used to handle feed and it is kept out of feed lanes or food storage areas.**
- **Make certain that feed delivery vehicles do not travel through manure or across manure-scraping lanes.**
- **Control rodents, birds and feral cat populations.**
- **You must be vigilant about waste management, control of effluent, and the distribution of recycled flush water.**
- **Vaccination – Vaccination will not stop infection but selected vaccines may reduce the severity of infection and curtail the mortality rate. Vaccines are no substitute for management to reduce contamination and decrease stress. Most salmonella vaccines licensed for commercial uses are formalin-inactivated products adjuvanted with aluminum hydroxide.**

- × Bacterins – efficacy ranges from good to ineffective; occasional anaphylactic reactions occur. Very few products contain *Salmonella* only. Autogenous bacterins are made and may have some benefit. Adverse reactions are frequently a complication of the latter.
 - × Modified live – naturally occurring and genetically manipulated, attenuated strains will provide better protection than bacterins, presumably from their ability to stimulate humoral and cellular immunity. The most widely tested is the genetically altered aromatic amino acid (aro) and purine (pur) auxotrophic mutants. To our knowledge, these are not commercially available in the US. Some modified live vaccines are given orally and these can be shed or found in tissues up to 3 weeks after administration. Modified live vaccines depend on persistency in the host for efficacy. A *Salmonella* Dublin vaccine (Entervene D, Fort Dodge) is currently available in the US and has been used in young calves in efforts to reduce clinical problems with this particular serotype. Some severe allergic type reactions have been reported with it, especially on endemic ranches and farms, and so clients and practitioners are advised to try this product on a small number of calves prior to widespread use, and to have corticosteroids and/or epinephrine on hand when using it. Currently in the US, a vaccine based upon SRP technology (Siderophore receptor and porin proteins) has become available that is enjoying widespread use in the dairy industry. The vaccine contains modified extracts of siderophore and porin proteins from *Salmonella* Newport. Immunologically these proteins invoke an antibody response against critical, iron scavenging and processing proteins that the bacteria need for logarithmic growth and replication. These antibodies block bacterial growth within the host by depriving them of iron – they are not specific to *Salmonella* Newport and cross protection against other serotypes and gram negative species is anticipated. Some herds have experienced increases in production and reduction in cell counts, independent of the effect on clinical Salmonellosis. Currently the product is given as a 2 shot series to dry cows according to label.
 - × Gram negative core antigens – these do not prevent infection or multiplication of the organism but provide protection against endotoxemia. With certain outbreaks, these can significantly lower mortality rates.
 - × Passive immunity with colostrum – There undoubtedly is some benefit when calves receive colostrum from vaccinated dams. After 3 weeks of age, colostrum immunity has little effect.
- **Pasteurization of waste milk and colostrum; even refrigeration will contain growth of salmonellae in contaminated colostrum and waste milk.**

From the AABP Food and Water Symposium, **11 action steps** were suggested to tackle herd Salmonellosis:

1. **Break the fecal-oral transmission link** by minimizing fecal contamination of feedstuffs, feeding surfaces, water troughs and equipment.
2. **Maximize host resistance** of susceptible animals (transition animals and newborns) and **minimize exposure dose**.
3. Control anything in the livestock environment that can perpetuate the organism – rodents, flies, nuisance birds, feral dogs and cats.
4. Because many of the infected animals are subclinical, **in an outbreak, handle all animals as if they were shedding**.
5. Implement a sound **sanitation program based on cleaning all organic matter** – feces, saliva, milk and blood – prior to use of disinfectants (orthophenylphenol on surfaces and boots and chlorhexidine for equipment).
6. Look for development of **newer vaccines** that target signaling pathways and other unique strategies rather than relying on conventional bacterins for prevention and control.
7. A **healthy intestinal environment** gives cattle a competitive resistance. Survival of competitive lactobacilli offer resistance to calves. Maintain the normal gram negative flora (< 1% of the gi mass of bacteria and these are primarily anaerobes) by minimizing oral antibiotic therapy.
8. **Maximize rumen function** by consistent DMI in transition and parturient cows. VFA's are toxic to salmonella.
9. Recognize extended survival time of salmonellae in the environment and deal with potential for spread 4 to 5 years after outbreak.
10. Minimize the chance for salmonellae to replicate by minimizing time in moist, warm environmental conditions. Mix feeds in smaller batches and feed soon after mixing. [Don't feed the waste TMR to young stock].
11. Warn farm families about **zoonotic potential** and assist them in implementing the steps below to minimize the risk.

Avoiding Zoonoses:

- **Outer layer of protective clothing is left at farm or at the site of contamination**
- **Gloves and protective wear**
- **Hand washing**
- **No eating or drinking in work areas**
- **Don't drink raw milk**