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Bovine Herd Salmonellosis

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Purpose and Note on Changing Salmonella Nomenclature

The purpose of this webpage is to provide students and practitioners an on-line review on *Salmonella enteritica* var Typhimurium DT104, some pointers on dealing with herd outbreaks of salmonellosis, and links to other resources. These materials are updated from presentations at veterinary practitioner conferences and the associated proceedings. These conferences include the University of Minnesota Dairy Health Conference, May 22, 1997, and the 31st Annual Conference of the American Association of Bovine Practitioners, September 26, 1998. This webpage is not intended to provide herd-specific advice and may not reflect the current state of the veterinary literature.

Changing Salmonella Nomenclature

For those practitioners searching and reading the clinical literature, the following may reduce some confusion about the changing nomenclature of salmonella. 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 is 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 bovine practitioners (e.g. *S. anatum*, *dublin*, *montevideo*, *typhimurium*) are now classified into a single species, *Salmonella enterica*, and are subclassified by their traditional serotype 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* serotype Typhimurium or *S. Typhimurium* rather than *S. typhimurium*. Recognizing this renaming will become more important as more papers are published using this new terminology.

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Tackling Herd Salmonellosis Problems

Much recent information on bovine salmonellosis is in practitioner literature, including thorough reviews of the pathogenesis (e.g., Lax et al. 1995) and treatment (e.g., House and Smith, 1998, McDonough, 1995) of salmonellosis. Two reviews specific to DT104 are on websites (Dargatz et al. 1998, Hogue et al. 1997). With these in mind, the following is based on those factors we have observed in investigations of salmonella outbreaks on livestock farms.

To slow a clinical outbreak:

Several practitioners have found that in the midst of a clinical outbreak in adult cows, once or twice daily monitoring of rectal temperatures in those animals most at risk of clinical disease, such as periparturient cows, and initiating systemic antibiotic treatment upon temperature rise may help. They have observed that body temperatures rise to 105-107°F a day or so before the diarrhea starts.

Cetiofur administered by normal dose and route appears to be the antibiotic of choice (Dr. Roger Ellis, AABP-L 3/16/98, 3/22/98). However, keep in mind that some strains are resistant to cetiofur. **Note:** Producers should institute this program only under the guidance of their veterinarian within a proper veterinary-client-patient relationship, only after a complete on-farm outbreak workup and only after complete identification of the organism including antibiotic sensitivities.

Eleven characteristics of Salmonella and salmonellosis to keep in mind

1. **Salmonella infection of a farm is maintained primarily by transmission of the agent from the feces of infected animals to the mouths of susceptible animals.**
 - The primary transmission route is fecal-oral; the epidemiology of salmonellosis is primarily the epidemiology of fecal pollution.
 - Transmission by inhaled aerosols occurs as well as by other routes (e.g., ocular, via teat streak canal, rectal) occurs, but less frequently.

Action: Enhance within herd biosecurity. Break the links in this chain by minimizing the opportunity for fecal contamination of feedstuffs, feeding surfaces, water troughs and equipment.

2. **Salmonella infection and subsequent clinical disease (the two are not synonymous) is a result of:**

1) **The innate resistance of the host animal.**

2) **The infectious dose received by the animal.**

3) **The infectivity and virulence of the particular strain of the organism.**

The livestock producer has the most impact on the salmonella cycle through maximizing 1) and minimizing 2) (and probably in that order) but cannot change 3) once the strain has been introduced to the farm. The only option for 3) is minimizing the chance of introduction by improving herd biosecurity.

- **Actions:**
- **Maximize the resistance of susceptible animals.**
- Pay careful attention to the transition of the most susceptible animals (periparturient cows, newborn calves).
- Concentrate on those things known to decrease host resistance and remember that above all else salmonella is an opportunist.
- For newborn calves this means calving them in a clean maternity pen, getting 4 quarts of clean, high antibody concentration colostrum into them within the first 2 hours of life before they are exposed to enteric pathogens and moving them into a clean calf hutch immediately.
- For closeup and fresh cows, this means such things as
- Getting them to appropriate body condition before dry off, maintaining that body condition score through the dry period,
- avoiding overcrowding of the closeup and fresh cows, avoiding dominance problems between heifers and mature cows and maximizing dry matter intake during the transition.
- The occurrence of any other periparturient problems, such as dystocia, displaced abomasum, ketosis and hypocalcemia, are clear indicators that cows are being stressed and thus are more susceptible to this disease. Concurrent diseases such as BVD should be minimized by implementation of a sound general herd vaccination program.
- **2) Minimize the exposure dose.** Considering the other points in this document, eliminate or control all means by which this infectious agent can get from infected animals to the mouths of susceptible animals either directly or indirectly, particularly by the contamination of feedstuffs,

feeding surfaces, water sources, or oral treatment equipment such as esophageal feeders, oral speculums, stomach tubes, balling guns and hands.

- One basic way to minimize exposure dose is to isolate susceptible animals from healthy appearing but potentially infected animals. This is a major weakness of many farm systems.
- For example, one of the strongest risk factors for a clinical salmonellosis problem in adult dairy cows is the practice of placing fresh cows in the hospital pen yet according to the NAHMS data this is a common practice. Another dangerous practice is holding back poor doing youngstock, potentially exposing susceptible younger animals in the group.
- For general considerations of infectious disease transmission, see Epidemiology Concepts for Disease in Animal Groups at <http://www.vetmed.wsu.edu/courses-jmgay/EpiMod2.htm>

3. Salmonella infects anything in the livestock environment that has an intestinal tract.

Besides cows and calves, salmonella infections occur in feral cats, dogs, 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.

Action: Initiate control programs for rodents, flies, nuisance birds, and feral dogs and cats. Rodent proof and bird proof feed storage and cattle housing facilities by removing nesting and roosting opportunities and by removing protective cover.

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.

- *Misunderstanding this "iceberg" effect by both practitioners and producers leads to inappropriate management of individuals in infected herds, meaning that often attention is paid only to the animals that are or were clinically ill.*
- *Although clinically affected animals shed much higher numbers of organism in the feces than do subclinically infected animals, the latter clearly shed enough to provide an infectious dose for many normal animals and are usually far more numerous in a herd.*

Action: *In an outbreak, handle all animals as if they were shedding, not just the sick ones. Institute procedures to protect all animals from all other animals, such as reducing contamination of water sources by installing guards and reducing contamination of feed.*

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 the biggest weakness of many farm treatment programs.

Action: Implement a sound sanitation program for potentially contaminated equipment, including training of personnel. All organic matter (e.g., saliva, manure, milk, milk fat film, blood) must be removed prior to the application of a disinfectant. The disinfectant must be in sufficient concentration and remain in contact for a sufficient period to kill the organism. Contact time is dependent on temperature. We recommend chlorhexidine for treatment equipment and orthophenylphenol for items such as boots and floor mats. After contact with infected or potentially infected animals and their discharges, wash hands well, scrubbing for 20 seconds.

6. Salmonella has a complex relationship with its animal host, which is only beginning to be understood. (Background for VACCINATION discussion)

Why many animals are subclinically infected while some others are clinically affected is unknown but research is advancing in this area.

- 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 cytoskeleton. 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.
 - However, this protein signal is only produced when the salmonella is near an enteric cell and is not produced during laboratory fermentation. *Thus autogenous bacterins will not decrease colonization [CU- sms].*
 - Because of this complex relationship, bacterins simply targeted at producing antibodies against antigens produced during standard laboratory fermentation are not likely to be highly efficacious. Other than anecdotal experience, little empirical evidence suggests that current commercial *bacterins are or are not beneficial and good clinical trials are needed in this area* (House and Smith, 1997).

- Bacterins, or deletion mutants, targeted at specific parts of this relationship, such as blocking the Type III signaling, are more likely to provide protection.
- For more information on vaccination as an option for control - see also - Evaluation of Bovine Salmonella Vaccines (JK House, BP Smith) <http://www.usaha.org/speeches/bosava97.html>.

7. Salmonella are a small part of an extremely competitive, complex, dynamic microbial environment in 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 (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 in part 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. Less clear in the ruminant is the effect of antibiotic use on the prolongation of the carrier state.

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 usually killed by exposure to the volatile fatty acids of fully functioning normal rumens.

- The level of VFAs in rumens of most cattle on continual full feed are toxic to most salmonella (Chambers and Lysons, 1979, Mattila et al. 1988).
- 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 may be more acid resistant and appears to be able to survive in the rumen of some chronic carrier cows.

Action: 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 livestock farm.

Salmonella have several different survival mechanisms (reviewed by Foster and Spector, 1995) that 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 and other control measures, such as heat or disinfectants, 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. Once dehydrated, some strains have been shown to survive exposure to 100° C for one hour (Kirby and Davies, 1990).

Salmonella survives in lagoons and can be recycled back to the herd in the flush water (Gay and Hunsaker, 1993).

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

Salmonella replicates rapidly in mixed feeds and on surfaces (wood!) 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, is contaminated and is at a sufficient temperature, which it will often be when laid upon by resting cows.

Salmonella is often present at low levels in many purchased feedstuffs, such as vegetable protein sources and both vegetable and animal fat sources. Under warm environmental conditions, mixing these contaminated feed ingredients with wet feeds, such as silage or haylage, may allow the organism to replicate until infectious doses for normal animals are reached if sufficient time between mixing and consumption passes. Placing wet mixed feeds on contaminated surfaces such as feed alleys may allow the same consequence.

Action: Minimize replication time, such as by not mixing large batches that are stored or not mixing well in advance of consumption.

11. Salmonella Typhimurium DT104 in livestock is a significant zoonotic disease risk for in-contact people, particularly young children.

Human disease due to this strain is a particular risk to farm families, particularly young children, and to employees in contact with infected cattle.

Actions:

- 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 strongly discouraged.
- Outer garments and footwear exposed to infected animals and their discharges should not be brought into the household.
- Hands should be washed well, using soap and warm water and scrubbing for 15 seconds, before returning to the household.
- In the household, food preparers should be particularly cautious about proper food handling to minimize opportunities for salmonella contamination and for the replication of this contamination. This includes refrigerating cool enough (<39°F), heating hot enough (>160°F) and minimizing the time that foods requiring cooking or refrigeration are exposed to temperatures between 40°F and 140°F. Prolonged exposures in this temperature range, even when thawing or cooling, allow minimal salmonella contamination to replicate to above infectious doses.
- Ten Steps to a Safe Kitchen at <http://www.exnet.iastate.edu/Pages/families/fs/steps/steps.html>
- The Consumer Control Point Kitchen at <http://www.exnet.iastate.edu/Pages/families/fs/ccp/ccpkitchen.html>

- Four Simple Steps to Fight BAC(teria)! at <http://www.fightbac.org/steps/index.html>
- Iowa State Extension Food Safety Publications (pdf) at <http://www.exnet.iastate.edu/Pages/families/fs/fspubs.html>
- Use sodium hypochlorite (bleach) to sanitize (3 tablespoons per gallon of water) or to disinfect (3/4 cup per gallon of water) surfaces and items, allowing at least two minutes of contact time.
- See <http://www.clorox.com/health/foodsafety.html> for further information.
- Minimize the opportunities for replication in moist, contaminated items such as dish cloths and sponges by disinfecting or changing and washing these frequently rather than reusing these between meals.
- As we have isolated this agent from clinically normal domestic pets associated with infected herds, in the presence of young children these animals should either be restricted to the household or from the household.

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 the facilities of that farm and to determine the best places to begin breaking the salmonella cycle. In herds that don't have a salmonella problem, proactively increasing herd biosecurity will reduce the likelihood of having an outbreak. Begin implementing bovine food and water safety. Otherwise, it may be only a matter of time. 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. Salmonella is a very worthy adversary for the veterinary practitioner.

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Review of *S. enterica* var Typhimurium DT104 (*S. typhimurium* DT104)

- In the early 1990's a new strain of salmonella, *Salmonella enterica* serotype Typhimurium variant DT104 (*S. Typhimurium* DT104), with broad antibiotic resistance emerged as a pathogen for humans and farmed animal species in the U.S. (Besser et al. 1997).
- This epidemic strain is identified by the following characteristics: it is phage type DT (distinguished type) 104, it had initially an antibiotic resistance pattern characterized by resistance to ampicillin, chloramphenicol, streptomycin (spectinomycin), 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 not widespread among strains of serogroup B salmonella and thus can be used as an initial method for screening and preliminary classification of group B isolates as the epidemic strain.

- This resistance to chloramphenicol includes florfenicol, which can also be used as a preliminary marker. In humans, the CDC reported that R-type ACSSuT increased from 9% of *S. Typhimurium* isolates in the U.S. in 1990 to 32% in 1996, when the first group outbreaks were recognized (Anon., 1997).
- WSU Results: 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, increased to 64% subsequently (Besser et al. 1997) and now appears to be declining. We have obtained *S. Typhimurium* with this R-type from a broad range of species in both farm and non-farm environments in the Northwest, having isolated it from the cow, horse, goat, emu, cat, dog, deer, elk, mouse, coyote, ground squirrel, raccoon, chipmunk and birds (pigeon, starling, pine siskin).
- The epidemic strain was first definitively recognized in England in 1984 in a human isolate (Threlfall et al. 1994). Human isolates of this phage and R-type had been detected between 1973 and 1982 in Hong Kong (Ling et al. 1987) but the resistance was plasmid-mediated (Ling and Chau 1987).
- In 1990 the epidemic strain began rising rapidly as a proportion of salmonella isolated from man, becoming second only to *S. Enteritidis*. The number of isolates peaked in 1996 and now appear to be declining, having decreased by 25% in 1997 (Threlfall et al. 1998).
- Of note is that during this period the total number of human *Typhimurium* isolates in England did not increase significantly, suggesting that this epidemic strain was displacing other *Typhimurium* strains in humans rather than occupying new niches.
- Compared with other *Typhimurium*s, this variant created great concern among the public health community (Akkina et al. 1999, Glynn et al. 1998, Poppe et al. 1998) for the following reasons.
 - First, the genes encoding this broad antibacterial resistance are integrated into the chromosome, suggesting that this resistance is likely retained in the absence of the selective pressure of drug use (Threlfall et al. 1994). Further, these genes are part of a "cassette" or integron, a mechanism that is easily exchangeable between different bacterial species (Mecenas and Strauss, 1996).
 - Of further concern is that *S. Typhimurium* DT104 emerged in Britain that are also resistant to fluoroquinolones (Threlfall et al. 1996), which are important drugs for the treatment of invasive salmonellosis in humans. This has very serious implications for antibiotic use in food animals, particularly of the fluoroquinolones. Initial British reports also suggested that this strain had a significantly higher human morbidity and case mortality than other *Typhimurium* strains (Threlfall et al. 1996, Wall et al. 1994, Wall et al. 1995), although this has yet to be confirmed by subsequent publications. The risk of bacteremia does not appear to be higher than other *S. Typhimurium*s (Threlfall et al. 1998).
 - Finally, human cases are significantly associated with contact with cattle in Britain, Scotland and the U.S. (Besser et al. 1997, Calvert et al. 1998, Wall et

al. 1994, Wall et al. 1995). In-contact children appear to be at significantly greater risk of clinical infection than in-contact adults (Calvert et al. 1998). Also of concern is that we have isolated the agent from clinical normal pet dogs and cats associated with infected herds, presenting another potential route of significant exposure for young children.

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 or only cases in calves are observed even though the environment is heavily contaminated (Anon., 1996, unpublished FDIU observations).
- 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.
- WSU: In our studies, we have observed a clinically normal carrier cow shedding over a million salmonella per gram of feces for over six months. At necropsy, all intestinal lumens including her rumen were salmonella positive, suggesting that her saliva was likely intermittently positive, potentially contaminating items the environment such as water troughs. The reasons for the variability have not been delineated, but the existence of such a wide variation of clinical syndrome suggests that factors in a farm's environment and management modulate the herd's experience with this agent. The frequency of these clinically normal, long-term chronic carriers has not been established.

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).
- We have observed a similar occurrence in an outbreak affecting a group of 20 in-contact cows, most of which shed on at least one occasion but none of which ever exhibited clinical signs.
- In another outbreak, 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 and Rawson, 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 our studies, we have isolated the agent from normal members of virtually all species associated with farms except for humans, which we have not sampled.

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).

- WSU Data: A preliminary analysis of our case-control study suggests that the strongest risk factors are purchasing animals and using a common area for both calving cows and housing sick cows. We have not detected any association between prior antibiotic usage and outbreak occurrence.

Neither the future course of this strain or its impact on the bovine industries are yet clear, although emerging evidence suggests that compared to other *Typhimurium*s it is not a super bug. Besides the broad antibiotic resistance, what makes DT104 different from other *S. Typhimurium* strains, if indeed 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? Our preliminary observations suggest that it can be shed in very high numbers for long periods by chronic carrier animals. How common these clinically normal chronic carrier animals are is unknown but evidence is mounting that they exist.

- Preliminary work in our lab suggests that it may survive in the environment only marginally if at all longer than other Typhimuriums. How long and where does it persist on infected livestock operations?
- What are the best ways to determine a herd's infection status? Testing cows or calves? If carrier animals are involved, what are the best means of detecting them? Performing conventional bacterial culture, using molecular based methods, running serological or milk-based ELISAs or a combination of these methods? How often and on what samples?

To prevent this infectious agent's spread and to eradicate it from infected premises, much remains to be learned.

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Evidence from Outbreaks of Other Salmonellas

Investigations of outbreaks due to other strains and serotypes may be useful for practitioners dealing with outbreaks of this agent.

- ❖ Investigators have reported that animals other than cattle are associated with the salmonella cycle of this and other serotypes.
 - 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).
 - 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. Infected droppings of wild birds have been found in feedmill environments (Davies and Wray, 1997) and the presence of

salmonella-contaminated feeds of swine farms has been shown to be associated with the lack of bird-proofing (Harris et al. 1997).

- 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).
 - A single rodent fecal pellet from infected mice typically contains up to 10^4 salmonella (Davis and Wray, 1995). Large rodent populations can be present before their signs (e.g., rodent droppings and runways) 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 work best if left baited and open for several days in areas frequented by the rodents before they are set. In our ongoing studies of affected herds, we have found several in which all rodent fecal collections were DT104 positive.
- Live-trapped raccoons have been reported to harbor *S. Typhimurium* (Morse et al. 1983). In one outbreak, we found that the feces of raccoons living in the bale stacks were DT104 positive. 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). In our ongoing studies of affected herds, we have found several in which all fly collections (primarily the house fly) were DT104 positive.
- That feedstuffs are often contaminated with salmonella and that outbreaks in livestock can be caused by this contamination is established. In fact, given the frequency of feedstuff contamination, the unanswered question is why outbreaks due to this contamination aren't recognized more frequently.
 - Although the recent ruminant protein feeding ban has potentially eliminated one likely source of exposure, recent evidence suggests that both animal and vegetable fat sources may be involved in salmonella infection (Jones et al. 1982, Losinger et al. 1997) and in outbreaks of clinical disease (Anderson et al. 1997). The epidemic DT104 strain has been reported both in off-farm (Davies and Wray, 1997) and on-farm (Kyrtenberug et al. 1998) feed samples. In the later study, *S. Typhimurium* DT104 replicated in 9 of 10 mixed rations from dairy farms when inoculated in the laboratory.
- 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 and Hunsaker, 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).

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On-line Resources:

Current bovine salmonellosis references -

Cornell Consultant at

http://www.vet.cornell.edu/consultant/consult.asp?Fun=Cause_134&spc=All&dxkw=salmonellosis&sxkw=&signs=
(current Cornell Consultant salmonella references)

National Library of Medicine PubMed

<http://www.ncbi.nlm.nih.gov/PubMed/>

[PubMed](#) with search terms "salmonella cattle"

Emergence of Multidrug-Resistant *Salmonella enterica* Serotype Typhimurium DT104 Infections in the United States (abstract, NEJM 338, 1998)

<http://www.nejm.org/content/1998/0338/0019/1333.asp>

Emerging Quinolone-Resistant Salmonella in the United States (CDC Emerging Infectious Diseases 3(3))

<http://www.cdc.gov/ncidod/EID/vol3no3/hayes.htm>

Evaluation of Bovine Salmonella Vaccines (JK House, BP Smith)

<http://www.usaha.org/speeches/bosava97.html>

Human Health Aspects of Salmonella Serotype Typhimurium Definitive Type 104 (DT104) (FJ Angulo, CDC)

<http://www.usaha.org/speeches/hhasal97.html>

Molecular Mechanisms of Bacterial Virulence: Type III Secretion and Pathogenicity Islands (J Meccas, EJ Strauss)

<http://www.cdc.gov/ncidod/EID/vol2no4/meccas.htm>

O is for Outbreak (US News and World Report article on DT104)

<http://www.usnews.com/usnews/issue/971124/24food.htm>

Salmonella typhimurium DT 104 (Institute of Food Science and Technology (UK))

<http://www.easynet.co.uk/ifst/hottop20.htm>

The Veterinarian's Role in Diagnosis, Treatment, and Prevention of Multidrug Resistant *Salmonella typhimurium* DT104

<http://www.aphis.usda.gov/vs/ceah/cahm/cahm-act.htm>

Travel and the Emergence of Infectious Diseases (ME Wilson)

<http://www.cdc.gov/ncidod/EID/vol1no2/wilson.htm>

USDA FSIS Situation Assessment: *Salmonella* Typhimurium DT104 (review of information as of December, 1997)

<http://www.fsis.usda.gov/ophs/stdt104.htm>

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