Case Report – Management of an Outbreak of Salmonellosis on a Commercial Calf Raising Unit

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Abstract

In the fall of 2001, a large commercial dairy calf and heifer raising operation suffered an outbreak of acute neonatal diarrhea. Prior to the outbreak, mortality before weaning was 2.5%, and during the outbreak preweaning death loss reached 10%. Multidrug-resistant *Salmonella* serogroup C2 (serotype Newport) was recovered from clinically affected calves as well as many environmental sites, including footbaths. This paper identifies some risk factors, such as low serum total protein, and reviews management protocols that were helpful, as well as those that were not (e.g., footbaths), for controlling transmission of *Salmonella*.

Résumé

À l'automne 2001, une flambée de diarrhée néonatale aiguë a sévi dans un gros élevage commercial de veaux et de taures laitières. Avant la flambée, le taux de mortalité pré sevrage était de 2.5% alors que durant la flambée le taux a atteint 10%. Des salmonelles du sérogroupe C2 (Sérotype Newport) multirésistantes aux médicaments ont été isolées à partir veaux affectés cliniquement de même que de plusieurs sites dans l'environnement incluant les bains de pieds. Cet article identifie certains facteurs de risque, tel que la faible quantité de protéines totales sériques, et fait un survol des pratiques de régie qui se sont avérées utiles de même que celles qui ne l'ont pas été (e.g. les bains de pieds) dans le contrôle de la transmission de la salmonellose.

Introduction

Assembling susceptible cattle in close contact increases risk of pathogen transmission and clinical disease. Large calf and heifer raising operations are becoming common, and exemplify this situation. In 2002, 10.5% of dairy cattle in the United States (US) were raised on sites other than their current milking operation.¹⁹ Cattle can be very productive in these intensive management schemes, but prevention and intervention strategies devoted to a single or few control points often fail in the face of serious disease challenges. Risky management practices may only become evident when a sufficient dose of a virulent pathogen is introduced.

Several types of Salmonella enterica subspecies enterica can infect dairy cows and calves, and cause clinical disease.²⁵ A national study identified the most common serotypes of Salmonella shed in the feces of dairy cattle as Montevideo, Cerro and Kentucky,24 while National Veterinary Service Laboratory testing in 1990 identified Typhimurium, Dublin and Typhimurium (var. Copenhagen) as the most common serotypes from clinically ill cattle.⁵ Some reports comment on the prevalence of Salmonella fecal shedding, but there is limited data regarding the prevalence of clinical salmonellosis in dairy cattle. In the northeast and midwestern US, fecal shedding was reported from approximately 6 to 9.3% of samples.^{8,22} The incidence of clinical S. Newport appears to be increasing in the US, and the number of animal origin S. Newport isolates tested by the National Antimicrobial Resistance Monitoring System at slaughter has increased since 1997.²¹ This serotype can show multi-resistance to antimicrobials.¹⁵

Salmonella is most often transmitted via the fecal-oral route and typically colonizes the gastrointestinal tract. It can also be spread by inhalation of aerosolized bacteria,⁶ across the placenta, or by excretion in milk.¹⁰ Salmonella can enter a herd through feed materials, new animals, fomites, water, or wildlife.¹⁰ Once the organism is on a farm, carrier animals may serve as a source of ongoing infection. Salmonella has been shown to persist in cattle herds for months to years after clinical cases have occurred.^{3,4,9,22} Identifying carriers can be difficult, as they may shed the bacteria only intermittently, making false negatives common when fecal cultures are performed.²⁵

Clinical signs of salmonellosis can include fever, diarrhea with or without blood and mucus, weight loss, dehydration, septicemia, recumbency and death. ^{16,17} Differential diagnoses for etiologic agents associated with diarrhea in calves include E. coli, rotavirus, coronavirus, Cryptosporidium parvum, bovine viral diarrhea virus, Clostridium perfringens and coccidia.¹⁴ Fever and watery diarrhea containing blood or mucus are highly suggestive of Salmonella, but a fecal culture, preferably from an untreated case, is confirmatory. Treatment may consist of oral and/or intravenous fluid therapy, anti-inflammatory agents and/or appropriate antimicrobials. A recent study showed that ceftiofur at 2.27 mg/lb (5 mg/kg) for five days reduced diarrhea, fever and fecal shedding of S. Typhimurium in experimentally infected calves.²

Humans, particularly if immunocompromised, in contact with cattle infected with *Salmonella* are at risk of acquiring infections.¹² Thus, anyone working with animals with salmonellosis should take appropriate precautions, such as wearing disposable gloves, designated boots and coveralls, and frequent and thorough hand washing. *Salmonella* also presents a significant foodborne public health risk.¹¹

This case report describes an outbreak of acute calf diarrhea on a large commercial calf and heifer raising operation.

Case Study

The Raising Unit History

The calf and heifer raising operation was located in eastern New York state and began operation in November 1999. It was designed to house 1,000 animals, aged two days to 11 months. The operation was enrolled in the New York State Cattle Health Assurance Program (NYSCHAP) in October of 2000 (http:// nyschap.vet.cornell.edu/).

Source dairies that sent calves to be raised at the facility retained ownership of the calves. Source farms were instructed to follow specific protocols regarding drycow vaccinations, calving facilities and colostrum management. Specific protocols were developed to help source farms reduce their risk for Johne's disease and bovine virus diarrhea (http://nyschap.vet.cornell.edu/). Calves were picked up during the first week of life in a specially designed trailer which had individual pens that were cleaned, pressure washed and disinfected after each load of calves.

Upon arrival at the raising unit, a physical examination was performed in a dedicated receiving room. Blood samples were collected to test for persistent infection with bovine viral diarrhea virus (BVDV) via polymerase chain reaction (PCR), as well as serum total protein measurement by refractometry. All calves found persistently infected (PI) with BVDV were euthanized when positive test results were received at three weeks of age. In the two years previous to the outbreak, two PI calves were detected and euthanized; none were detected during the outbreak. When the serum total protein was below 5.0 g/dl, or if pre-existing illness was detected, the raising unit reserved the right to return the calf to the source farm. An intranasal IBR-PI, vaccination was given, along with injections of B-vitamins, vitamin E and selenium.

After admission, calves were moved to a "wet-calf barn" with individual pens that prevented direct contact. Sidewalls of the pen were a solid, non-porous material. The front of the pen was a "bar" construction, with an opening to allow the calf access to feed and water. The rear panel was mesh wire, with 2.5 inch x 2.5-inch (6.3cm x 6.3cm) openings. Pens were arranged in four rows of 12 pens in each barn, with calves from two rows facing each other, 5 ft (1.5m) apart (Figure 1).

This barn measured 50 by 60 ft (15 x 18m), with 10 ft (3 m) sidewalls and an 8-inch (20cm) ridge opening. A cap, 8 inches (20 cm) above the ridge, covered the ridge opening. A conventional "A" roof with translucent panels allowed sunlight in. Curtains on the sidewalls could be positioned to facilitate or restrict air movement (Figure 2).

Wet-calf barns were filled as calves arrived at the operation, and then maintained as a closed group until calves were weaned and moved. Three identical "wetcalf" barns existed, referred to as barn one, two and



Figure 1. Wet-calf barn pens illustrating the distance between pens and pen construction.

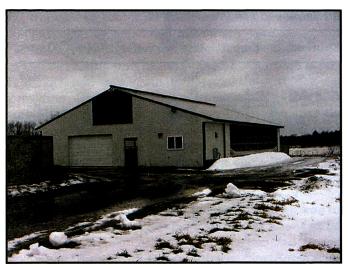


Figure 2. Wet-calf barn.

three. As calves were admitted, opposing pens were filled, so that calves of similar age faced each other. Most of the time, the entire barn was filled within two weeks.

Calves were fed a 28% protein, 20% fat milk replacer at 2.2 lb (1 kg) of powder per day by bucket. A 22% protein calf starter grain mix was provided free choice. Calves were weaned at approximately five weeks of age; gain was typically 1.5 lb (0.7 kg) per day from admission to weaning. After weaning, the 22% protein grain mix was fed to a maximum rate of 7 lb (3.2 kg) per day until three months of age. A total mixed ration was fed from that age until 11 months of age, when calves were either discharged or transferred to another unit for further conditioning and breeding.

Original management protocols called for workers to enter the wet-calf barns wearing boots free of visible dirt, and dip their feet in a disinfectant solution. Various products had been used, including chlorhexidine, peroxygen^a and phenol solution^b, diluted according to label directions. Workers wore disposable rubber gloves when feeding or providing care. Sick animals were treated last, and gloves were changed between sick animals.

At approximately five weeks of age, calves in two adjacent rows facing each other were weaned in groups, leaving half of the barn empty, and moved into group pens with eight calves per pen. Later, pens were combined to make larger groups, and finally calves were put into free-stall housing.

After a "half-barn" of calves were moved, pens were removed to be cleaned, pressure washed and a disinfectant applied, while the other half of the barn still had calves in it. Either chlorhexidine, peroxygen, or phenol product was used for disinfection, following label directions. Barn floors and walls were also cleaned and disinfected, first by scrubbing and then pressure washing. Pens were then replaced, fresh straw and sawdust bedding was added, and new calves admitted. Following these protocols, pre-weaning death loss was 2.5% (16/631; 95% exact binomial confidence interval, $1.5 - 4.0\%^{d}$) from January 1 through July 31, 2001 (pre-outbreak).

Description and Results of the Outbreak

For the purpose of this report, a case of salmonellosis was defined as a calf with a rectal temperature above 102.5°F (39.2°C), bloody diarrhea and weakness with or without confirmatory bacteriologic culture of *Salmonella*. All calves at the outset of the outbreak had bacteriologic confirmation, as did most subsequent suspect cases. All cases were identified by one of two people, adding consistency to the case definition and enumeration.

On August 19, 2001, a three-day-old calf, housed in barn three, had a rectal temperature of 104.2° F (40.1°C) and bloody diarrhea. Feces was submitted to the New York State Animal Health Diagnostic Lab for culture, and yielded *Salmonella* group C2.¹ The calf was treated with oral electrolytes and ceftiofur, administered subcutaneously at 1.0 mg/lb (2.2 mg/kg) every 12 hours for four days, and recovered uneventfully. This calf originated from source farm 12, and had a serum total protein of 5.4 gm/dl at the time of admission.

Over the next two weeks, 11 calves in barn three developed signs compatible with salmonellosis, and eight died. Six of the eight calves had total protein values less than 5.0 gm/dl at admission. Rectal swabs were submitted for culture from the 11 calves; all yielded *Salmonella* serogroup C2, with no other pathogenic bacteria cultured.

E. coli was isolated from the calves, but genotyping revealed no pathogenic adhesins or toxins. The *Salmonella* isolates were eventually confirmed to be serotype *S.* Newport by the National Veterinary Services Laboratories, Veterinary Services, Animal and Plant Health Inspection Service, United States Department of Agriculture (NVSL, VS, APHIS, USDA), Ames, Iowa. In addition, fresh feces were submitted for detection of viruses and protozoa, but none were found.

Antimicrobial susceptibility testing showed resistance to most antimicrobials tested except for trimethoprim/sulfamethoxazole (TMS), aminoglycosides and enrofloxacin; the latter two were not used. Some isolates showed resistance to all drugs in the test screen available for treatment, including TMS, as the outbreak progressed (Table 1). Most calves were treated extralabel with TMS, with appropriate supervision by the attending veterinarian. A dosage of 160 mg of trimethoprim and 800 mg of sulfamethoxazole was given orally every 12 hours for seven days.

Most early disease was limited to barn three, but as barn one was populated a similar disease pattern occurred, and one calf died. A similar pattern occurred

 Table 1.
 Susceptibility testing¹ for selected antimicrobials to Salmonella isolates.^{2,3}

Date of culture	Sample source	TMS	Ceft	Flor	Gent	Enro	Amp	Sulfas	Neo	Oxytet
8-23-01	calf	s	R	R	s	s	R	R	s	R
8-28-01	calf	s	R	R	s	s	R	\mathbf{R}	s	R
8-29-01	calf	s	R	R	s	s	R	\mathbf{R}	R	R
9-11-01	calves	S	R	\mathbf{R}	s	s	R	\mathbf{R}	s	R
10-5-01	calves	s	R	\mathbf{R}	R	s	R	\mathbf{R}	s/R	R
10-23-01	calf	R	R	R	R	s	R	\mathbf{R}	R	R
10-24-01	calves	S	\mathbf{R}	\mathbf{R}	s/R	s	\mathbf{R}	\mathbf{R}	R	\mathbf{R}
10-30-01	calf	S	\mathbf{R}	\mathbf{R}	R	s	\mathbf{R}	\mathbf{R}	\mathbf{R}	\mathbf{R}
10-31-01	calves	S	R	R	s/R	s	R	\mathbf{R}	R	\mathbf{R}
11-6-01	calf	S	R	R	Ι	s	R	\mathbf{R}	\mathbf{R}	R
11-9-01	calf	S	R	\mathbf{R}	s	s	\mathbf{R}	\mathbf{R}	s	R
11-27-01	calf	R	\mathbf{R}	\mathbf{R}	R	s	\mathbf{R}	\mathbf{R}	\mathbf{R}	\mathbf{R}
11-27-01	calves	R	R	R	R	s	\mathbf{R}	\mathbf{R}	\mathbf{R}	\mathbf{R}
12-11-01	calf	R	R	R	Ι	s	R	R	\mathbf{R}	R
12-20-01	environment	s	R	R	Ι	s	R	R	R	\mathbf{R}
12-21-01	calves	R	R	R	R	s	R	\mathbf{R}	R	\mathbf{R}
1-9-02	heifers	R	\mathbf{R}	R	s	s	R	R	R	\mathbf{R}
1-23-02	heifers	R	R	R	Ι	s	R	R	s/R	\mathbf{R}
1-30-02	calves	R	R	R	R	s	R	R	$\mathbf R$	\mathbf{R}
2-1-02	heifers	R	\mathbf{R}	R	Ι	s	R	R	R	\mathbf{R}

¹Performance Standards for Antimicrobial Disk and Dilution Susceptibility Tests for Bacteria Isolated from Animals; Approved Standard, Second Edition, Volume 22, Number 6 (NCCLS, 940 West Valley Road, Suite 1400, Wayne, PA 19087-1898, USA). ²Some breakpoints are not necessarily validated for *Salmonella*, e.g. ceftiofur, and performance of susceptibility testing does not imply treatment recommendations.

³s = sensitive, I= intermediate, R=resistant, TMS=trimethoprim/sulfamethoxazole; Ceft=ceftiofur; Flor=florfenicol; Gent=gentamicin; Enro=enrofloxacin; Amp=ampicillin; Sulfas=sulfathiazole, sulfadimethoxine or sulfachloropyridazine; Neo=neomycin; Oxytet=oxytetracycline.

as barn two was filled with new calves and as a new population of calves were housed in barn three. At times, the population of calves in these barns was decreased to reduce the density of susceptible animals.

The morbidity pattern suggested that calves from source farm 12 were infected at the time of admission. Fecal cultures from calves, less than four days old while still on the source farm (farm 12), confirmed the link between the source farm and the outbreak on the raising unit.

To avoid bringing infected calves into the facility, the raising unit staff began delaying admission of animals from source farm 12 until calves were approximately one week of age. During that time their temperature and manure were monitored closely. If signs of *Salmonella* infection were noted, treatment was instituted, and admission was further delayed until the calf returned to clinically normal health. Once admitted, calves from farm 12 were housed in hutches 30 feet (10 m) away from any other animals.

From September 26 to October 19, 2001, very few calves with clinical signs consistent with salmonellosis, and no deaths, were noted. It was believed that the outbreak was under control and essentially over. This assumption proved to be false. On October 19th, a five-day-old calf in barn one developed clinical signs of *Salmonella* infection. Within a few more days, most calves in this barn were ill, and six animals died. As the next barn was filled with calves, workers cared for the new calves before going to barn one. Boots and clothing were changed after working in barn one. Despite this effort, salmonellosis soon developed in the barn being filled with new animals. The decision was then made to refuse all calves from source farm 12, and to reject any calf with serum total protein below 5.0 gm/dl.

At this time, outside consultation was sought and a visit to the raising unit was made to investigate the outbreak and make management recommendations.

On October 30th, management protocols were reviewed. In addition, environmental samples were collected by dragging 4x4 swabs drenched in sterile double strength skim milk in the transporting trailer, admission room floor, gravel walkways between barns, calf barn floors, floors in rooms where milk replacer was prepared and one foot bath located at the entrance to the raising unit. This footbath contained a peroxygen^a disinfectant mixed according to label directions. All swabs, except the ones from the transporting trailer, were positive for *Salmonella* serogroup C2. Suggestions for changes in protocols designed to reduce spread of the pathogen by personnel movement were made, and the human health risks were stressed.

The raising unit began filling another barn on October 29th, and once again it appeared the outbreak was subsiding. Disease problems were minimal as this barn was filled, and only one death occurred. Because the number of incoming calves was reduced at this time, it took three weeks to fill this barn. This allowed time for the staff to completely empty barn one. It was thoroughly cleaned and sanitized with a phenol disinfectant^b, and sat empty for three days.

On November 12th, environmental testing was repeated, collecting samples from similar locations as before, including a footbath that had been emptied, rinsed, and made fresh only a few minutes earlier. *Salmonella* was again cultured from most locations, including the footbath. This footbath solution had just been changed and a peroxygen disinfectant^a had been added according to label directions.

On November 19th, new calves were put into barn one, and filled by December 6th, but by that time new cases of salmonellosis were noted. At this time, the staff suspected that infected calves were coming from a second source farm because some animals began showing signs within one or two days after arrival. Pre-admission culture results from all calves from this farm confirmed this suspicion, and no more animals were accepted from this farm.

At this time a new protocol was instituted to discontinue using footbaths. Instead, separate boots and coveralls were maintained in each wet-calf barn, and were worn only in that barn. Also, "calf-carts" used to move calves from the receiving room were no longer brought into the barns with new calves to avoid *Salmonella* contamination from the tires that were contaminated on walkways. Instead, a staff member outside the barn would lift the calf out of the cart and place it inside the barn through an open door to an inside staff member.

Also at this time, additional calf hutches were purchased, and all new calves were placed in these hutches for two weeks. This allowed the next barn in the rotation to remain empty after cleaning and disinfecting. Swabs taken from concrete floors in this barn after initial sanitizing with phenol demonstrated large numbers of *Salmonella* organisms, so it was disinfected again using a glutaraldehyde disinfectant.^c Swabs taken for culture after the second disinfection showed *Salmonella* was still present, but in very low numbers. New calves were placed in barn three beginning on December 21.

Between August 1 and December 31, 2001, preweaning mortality was 10% (40/404) on the raising unit. Thirty-six (8.9 %; 95% exact binomial confidence interval, $6.3 - 12.1^{d}$) of the deaths were calves that were

either culture-positive for Salmonella and/or had signs consistent with our case definition of salmonellosis. After the outbreak (January 1 - October 1, 2002) the pre-weaning mortality decreased to 1.4% (9/533). Figure 3 shows the number of calves treated at least once for signs consistent with our case definition of salmonellosis, and the number of deaths by week during the outbreak. The age at first treatment ranged from 1-25 days (median = 8). This was a median of five days after arrival at the rearing unit. The age at death for calves with salmonellosis ranged from 4-28 days (median = 14). At least seven of the dead calves representative of salmonellosis cases received a full necropsy. All were fluorescent antibody negative for BVDV, rotavirus and coronavirus, and all were culture-negative for pathogenic bacteria. The final diagnosis for these calves was salmonellosis.

Farm records of calves admitted from August 1, 2001 to December 31, 2001 showed calves with a serum total protein less than 5.0 gm/dl had an odds of dying 2.3 times higher (95% confidence interval, $1.01 - 5.14^d$) than those calves with total protein greater than or equal to 5.0 gm/dl (Table 2). Results from a logistic regression analysis controlling for herd effects showed a similar association between low total protein and mortality (odds ratio = 2.3; 95% confidence interval, 0.9 - 5.9).^e

After December 21, 2001, no new clinical cases occurred anywhere in the facility. Environmental swabs taken in February of 2002, from the same sites as earlier, cultured no *Salmonella* pathogens.

Discussion

Design of the calf barns at this raising unit did not provide adequate isolation between susceptible calves once a virulent pathogen was introduced. While calves could not touch each other, they were housed in close proximity. Pens were open at the front and rear to allow cross-ventilation, but this same feature may have

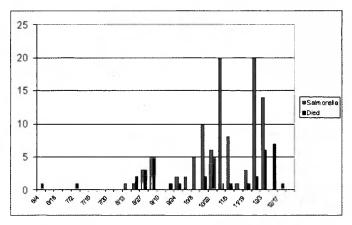


Figure 3. Epidemic curve of clinical salmonellosis cases and deaths before weaning from all causes.

Two-by-two table of calf mortality attributed
to salmonellosis by measure of transfer of
passive immunity as determined by refrac-
tometry with a cut-point of 5.0 g/dl.

Serum total protein			
(gm/dl)	Died	Survived	Total
< 5.0	9	47	56
≥ 5.0	27	321	348
Total	36	368	404

Odds ratio: 2.28 (95% confidence interval: 1.01 - 5.14)^d Attributable fraction: 56.1 (95% confidence interval: 0.9 - 80.5)^d

allowed aerosol and splashing movement of pathogens. Preweaning morbidity during the outbreak ranged from 25 to 95% once the disease appeared in a barn, and preweaning mortality ranged from 2 to 31%. At the same time, none of the 32 calves raised in individual hutches suffered any serious illness, despite being within 30 feet of infected barns, and cared for by the same staff personnel during the worst of the outbreak. A raising unit under the author's (CEG) supervision at another site was designed with all 48 pens arranged as one sheltered row, with 50 feet (15m) between rows. Contagious disease, including *Salmonella* serogroup C2, has been introduced at this facility with virtually no apparent transmission among calves.

The raising unit's protocol for moving weaned calves consisted of moving "half barn" at a time. However, calves admitted previously were present in the other two rows. In some cases, these calves were the next group to be moved, and thus several weeks old. While this practice is an obvious weakness in the biosecurity program, calf losses were 2.5% during the seven months prior to the outbreak, and 1.4% during the 9 months following the outbreak, much better than the national average of 10.5%.²⁰ In addition, changing the protocol so that an entire barn was emptied, disinfected and left empty for three days did not prevent new cases from occurring. The practice of moving "halfbarns", while not desirable, was used successfully before the introduction of S. Newport. Emptying the barn completely before refilling was not sufficient to prevent new cases once the outbreak started.

Multiple culture-positive environmental sites throughout the raising unit suggest that workers and equipment movement had carried *Salmonella* around the "wet-calf" area. Similar samples taken after thorough cleaning and disinfecting one barn showed the pathogen remained. Even after the barn was empty for two weeks, and disinfected with an extremely potent agent,^c cultures from various sites in this barn still revealed the presence of small numbers of pathogens. Biofilms are believed to protect some pathogens from disinfectants. Research that simulated a barn floor showed that *Salmonella* can survive in this environment for 5.5 years.¹³ This suggests it is very difficult to to-tally eliminate S. Newport with practical on-farm cleaning and disinfecting processes.

In retrospect, it appears that biosecurity protocols used at the raising unit were adequate until a virulent pathogen was introduced and/or some other undetected management change occurred. Once introduced, a weakness in building design could contribute to extensive spread of the disease within the barn. Additionally, it is clear that dipping boots in footbaths, even when visibly clean, was not effective for preventing the spread of *Salmonella*. In fact, a "pretty clean" footbath was culture-positive, as well as the one where the disinfectant had just been changed. Changes made to keep separate boots and clothing in each wet-calf barn appeared to reduce the spread of *Salmonella*.

Despite the inability to eliminate the pathogen, the Salmonella outbreak ended quickly once the second infected source farm was identified and calves from it no longer admitted. This suggests that many calves can resist a pathogen challenge as long as the infectious dose of organisms is relatively low, their immunity is adequate and the biosecurity plan limits the spread of the pathogen. However, if a calf shedding the organism is admitted to the population and sheds a sufficient dose of pathogens, then additional sick animals are likely if they are susceptible and transmission can readily occur. Although the reason for the decrease in new cases in this outbreak is unknown, it is possible that stopping the introduction of calves from infected farms played a pivotal role. A previous report suggests that identification of pre-infected animals can be helpful in controlling an outbreak.¹

Serum total protein levels for all calves were determined at admission, and the raising unit reserved the right to reject those with levels below 5.0 gm/dl. Calves were not rejected for this reason prior to and during the early stages of the outbreak, but as the problem worsened calves with low serum total protein were refused. Farm records of calves admitted from August 1, 2001 to December 31, 2001 showed that the odds of calves dying was 2.28 times higher if the serum total protein was less than 5.0 gm/dl compared to those with a total protein greater than 5.0 gm/dl (Table 1).

It has been reported that if more than 15% of calves in a high-density calf environment are immunodeficient, then the entire group of animals is very susceptible to an infectious disease outbreak.¹ Further, it has been shown that serum protein concentrations <5.0gm/dl, as measured by refractometry, were associated with increased mortality on a dairy replacement facility with endemic salmonellosis. ¹⁸ Prior to the decision to reject any calves with serum total protein levels below 5.0 gm/ dl, 11% of all incoming calves were below that threshold. However, 31% of the calves in the barn where the outbreak first started were below the threshold, which may have been a factor that facilitated the outbreak.

Concern was expressed that some infected calves that recovered clinically would remain infected in a subclinical carrier state. Serial culture of pooled fecal samples was used to identify heifers shedding Salmonella in their feces. Five pooled samples, made up of manure from six animals, were submitted for culture before they were released from the facility at approximately 11 months of age. This practice was applied to all animals admitted into the facility during the outbreak, from August 1 to December 31, 2002. These pooled samples were collected approximately every other day until five were obtained. If any of the pooled samples were positive for Salmonella, then each individual was cultured. Using this method, four animals were found to be shedding Salmonella, and were sold for slaughter. All four appeared clinically normal, and were normal size.

S. Newport can develop resistance to antibiotics labeled for use in dairy cattle, as was true in this case. Early in the outbreak, the decision was made to use extra-label antibiotics under the supervision of the attending veterinarian. However, enrofloxacin was not used due to the FDA prohibition of extra-label usage of this drug. In addition to trimethoprim/sulfamethoxazole (TMS), trimethoprim/sulfadiazine was used both as an intravenous injection and as an oral paste, following the dosages recommended for horses. This was done when one group of tests suggested greater sensitivity to this product. A number of supportive therapies, including intravenous and oral electrolytes, B-vitamins and nonsteroidal anti-inflammatory drugs were administered to some animals.

Use of an autogenous bacterin was not favorably considered because efficacy data were lacking. One study showed that cattle vaccinated with an autogenous bacterin made for the serogroup on that dairy had similar fecal shedding, mortality and milk production as unvaccinated controls.⁷

At one point all incoming calves were administered trimethoprim/sulfadiazine paste for the first five days in the unit, but this did not appear efficacious. In addition, in mid-November isolates from calves and heifers were no longer susceptible to TMS. Increased use of TMS likely caused *Salmonella* to develop resistance. In our opinion, the focal point for *Salmonella* control must be on prevention rather than antimicrobial treatment.

On several occasions consideration was given to refusing additional calves until the *Salmonella* outbreak

ended and the pathogen load in the environment was reduced. However, the raising unit had signed contracts to provide care and housing for all calves from several farms. Many of these farms no longer had facilities and/ or personnel to raise their own replacements. Therefore the decision was made to continue accepting new animals, while working to stop the outbreak.

Conclusions

This case suggests the following recommendations for the management of a dairy replacement-raising facility that assembles neonatal calves from several source farms:

- 1. Determine the adequacy of passive transfer of immunity by testing serum total protein by refractometry or an equivalent test. In this outbreak, calves with total protein below 5.0 gm/dl were at significantly greater risk of death than those above this level.
- 2. If *Salmonella* is associated with epidemic scours, testing incoming calves for pre-existing infection may aid in decreasing the environmental dose of pathogens and breaking the transmission cycle. This practice was temporally related to mitigating the outbreak on this raising unit.
- 3. Antimicrobial resistance may develop rapidly during the course of an outbreak. Here, resistance to TMS occurred near the time it was administered to all calves at arrival at the raising unit. Relying on antimicrobials to manage Salmonella may only be useful in the short-term, especially when dealing with multi-drug resistant S. Newport.
- 4. Do not depend on footbaths to kill pathogens, especially *Salmonella*. It was cultured from multiple footbaths on this raising unit during the outbreak. Instead, providing separate boots, clothing and rubber gloves to be worn in each calf barn and adhering to traffic flow patterns that do not allow cross-contamination of multiple areas will likely be more effective for managing the risk associated with epidemic salmonellosis.

Adhering to these guidelines should minimize the introduction and spread of *Salmonella* or other contagious pathogens in a calf and heifer-raising unit.

Acknowledgements

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Footnotes

^aVirkon S[®], Antec International, Sudbury, UK

^b1-Stroke Environ[®], Steris Co., St. Louis, MO

^eAldacide[®] 200, Russell Co. Laboratories, Longmont, CO ^dEpi-Info Version 6.04 2001, Centers for Disease Contral and Provention Atlanta CA

trol and Prevention, Atlanta, GA

°PROC Genmod, SAS version 8.2 for Windows, SAS Institute Inc. Cary, NC

References

1. Bender J: Reducing the risk of *Salmonella* spread and practical control measures in dairy herds. *Bov Pract* 28:62-65, 1994.

2. Fecteau M-E, House JK, Kotarski SF, *et al*: Efficacy of ceftiofur for treatment of experimental salmonellosis in neonatal calves. *Am J Vet Res* 64:918-925, 2003.

3. Gay JM, Hunsaker ME: Isolation of multiple Salmonella serovars from a dairy two years after a clinical salmonellosis outbreak. J Am Vet Med Assoc 203:1314-1320, 1993.

4. Giles N, Hopper SA, Wray C: Persistence of S. typhimurium in a large dairy herd. Epidem and Infect 103:235-241, 1989.

5. Glock RD, Schwartz KJ: Salmonellosis: A growing problem for food animal producers. *Large Animal Vet* 48:12-14, 1993.

6. Hardman M, Wathes CM, Wray C: Transmission of salmonellae among calves penned individually. *Vet Rec* 129:327-329, 1991.

7. House J, Ontiveros M, Blackmer N, et al: Evaluation of an autogenous Salmonella bacterin and a modified live Salmonella serotype Choleraesuis vaccine on a commercial dairy farm. Am J Vet Res 62:1897-1902, 2001.

8. Huston CL, Wittum TE, Love BC, Keen JE: Prevalence of fecal shedding of Salmonella spp in dairy herds. J Am Vet Med Assoc 220(5):645-649, 2002.

9. Huston CL, Wittum TE, Love BC: Persistent fecal Salmonella shedding in five dairy herds. J Am Vet Med Assoc 220(5):650-655, 2002.

10. McDonough PL: Salmonellosis: Diagnostic approach to disease control and epidemiology in the bovine animal. *Proc Am Assoc Bov Pract* 27:61-68, 1995.

11. Mead PS, Slutsker L, Dietz V, *et al*: Food-related illness and death in the United States. *Emerging Infectious Diseases* 5:607-625, 1999. 12. Olsvik O, Sorum H, Birkness K, *et al*: Plasmid characterization of *Salmonella typhimurium* transmitted from animals to humans. *J Clin Micro* 22:336-338, 1985.

13. Plym-Forshell L, Eskebo I: Survival of salmonellas in urine and dry faeces from cattle-An experimental study. *Acta Veterinaria Scandinavica* 37:127-131, 1996.

14. Radostits OM, Blood DC, Gay CC: Veterinary Medicine, ed 9. London, Saunders, 2000.

 Rankin SC, Aceto H, Cassidy J, et al: Molecular characterization of cephalosporin-resistant Salmonella enterica serotype Newport isolates from animals in Pennsylvania. J Clin Micro 40: 4679-4684, 2002.
 Rebhun WC: Diseases of Dairy Cattle. Phildelphia, Williams & Wilkins, 1995.

17. Smith BP: Large Animal Internal Medicine, ed 3. St. Louis, C.V. Mosby Company, 2002.

18. Tyler J, Hancock D, Wiksie S, *et al*: Use of serum protein concentration to predict mortality in mixed-source dairy replacement heifers. *J Vet Intern Med* 12:79-83, 1998.

19. USDA. Part I: Reference of Dairy Health and Management in the United States, 2002. Fort Collins, CO: USDA: APHIS:CEAH 82, 2002. 20. USDA. Part II: Changes in the United States Dairy Industry 1991-2002. Fort Collins, CO: USDA: APHIS:CEAH, 2002.

21. USDA. What veterinarians and producers should know about multidrug-resistant *Salmonella* Newport. Fort Collins, CO: USDA: APHIS: CEAH 4, 2002.

22. Warnick LD, Kaneene JB, Ruegg PL, *et al*: Evaluation of herd sampling for Salmonella isolation on midwest and northeast US dairy farms. *Prev Vet Med* 60(3):195-206, 2003.

23. Warnick LD, Crofton LM, Pelzer KD, et al: Risk factors for clinical salmonellosis in Virginia, USA cattle herds. *Prev Vet Med* 49:259-275, 2001.

24. Wells SJ, Fedorka-Cray PJ, Dargatz DA, et al: Fecal shedding of Salmonella spp. by dairy cows on farm and at cull cow markets. J Food Protect 64:3-11, 2001.

25. Wray C, Wray A: Salmonella in Domestic Animals. Wallingford, UK, CAB International, 2000.