

# Preventing Calf Scours with the Sandhills Calving System

David R. Smith, DVM, PhD  
Institute of Agriculture and Natural Resources  
Department of Veterinary and Biomedical Sciences  
University of Nebraska –Lincoln

## INTRODUCTION

Diarrhea is one of the most likely reasons young beef calves become sick or die.(45) Besides its detriment to calf health and well-being, calf scours is costly to cattle producers due to poor calf performance, death, and the expense of medications and labor to treat sick calves. (3, 41) In addition, catching and treating young calves puts herd owners and their employees at risk of injury, and many producers become disheartened after investing long hours to treat scouring calves during an already exhausting calving season.

Calf scours is a complex disease, with many interrelated causes.(1, 2, 37) Agent, host, and environmental factors collectively explain scours, and these factors interact dynamically over the course of time. Cattle producers and their veterinarians must understand the relationships between these factors within the production system to control the disease or prevent its occurrence.(6)

## AGENT FACTORS

Numerous infectious agents have been recovered from calves with diarrhea.(1, 6, 2, 10, 24, 43, 4, 27, 25, 22) Common agents of calf diarrhea include bacteria such as *Escherichia coli* and *Salmonella*, viruses such as rotavirus and coronavirus, and protozoa such as cryptosporidia. Bovine rotavirus, bovine coronavirus and cryptosporidia are common to most cattle herds, and can be recovered from calves in herds not experiencing calf diarrhea.(6) Knowing the name of an agent recovered from a calf with scours may explain the immediate cause of the calf's illness or death, but that knowledge rarely explains the outbreak, or provides a solution for treatment, control, or prevention. Further, it is typical that multiple agents can be recovered from herds experiencing outbreaks of calf diarrhea; suggesting that even during outbreaks more than one agent may be involved. The adult cow herd commonly serves as the source of pathogens from one year to the next.(14, 12, 13, 23, 47, 35)

## HOST FACTORS

Calves obtain passive immunity against common agents of calf diarrhea after absorbing antibodies from colostrum or colostrum supplements shortly after birth.(7, 8, 9) The amount of antibodies absorbed is determined by the quality and quantity of colostrum the calf ingests, and how soon after birth it is ingested. The presence of antibodies in colostrum requires prior exposure of the dam to the agent. Vaccines are sometimes used to immunize the dam against specific agents, and some commercially available colostrum supplements

contain polyclonal or monoclonal antibodies directed against specific agents. Unfortunately, the use of vaccines or colostrum supplements has not always prevented outbreaks of calf diarrhea.

Calves typically become ill or die from diarrhea within one to two weeks of age.(11, 1, 43, 10) The narrow range of age within which calf diarrhea occurs is not explained solely by the incubation period of the agents. Experimentally, diarrhea is observed in calves which have not received colostrums within a few days of the pathogen challenge regardless of age.(16, 17, 36) Calves may have an age-specific susceptibility to diarrhea agents that occurs as maternal immunity is waning and before the calf is fully capable of developing an active immune response.(7)

Regardless of the reason, the first seven to 14 days of age defines the age of susceptibility as well as the age calves are most likely to become infective and shed the agents in their feces.(18, 44, 29, 30, 35, 26) This is important because in some calving systems the number of susceptible and infective calves can change dynamically with time. At times the number of potentially infective calves may greatly outnumber the number of susceptible calves resulting in widespread opportunity to transfer a disease-causing dose of pathogens.

The dam's age also explains a young calf's risk for diarrhea. Calves born to heifers are at higher risk for diarrhea and have lower maternal antibody levels than calves born to older cows.(39) Calves born to heifers are probably more susceptible to disease because heifers produce a lower volume and quality of colostrum, may have poor mothering skills, and are more likely to experience calving difficulty.(32, 31)

## **ENVIRONMENTAL FACTORS**

The environment may influence both the level of pathogen exposure and the ability of the calf to resist disease. Exposure to pathogens may occur through direct contact with other cattle or via contact with contaminated environmental surfaces. Keeping the environment clean has long been recognized as important for controlling calf diarrhea,(20, 46) but doing so is often a challenge. An effective contact is an exposure to pathogens of a dose-load or duration sufficient to cause disease. Crowded conditions increase opportunities for effective contacts with infected animals or contaminated surfaces. Ambient temperature (e.g. excessive heat or cold) and moisture (e.g. mud or snow) are important stressors that impair the ability of the calf to resist disease and may influence pathogen numbers as well as opportunities for them to be consumed.

## **TEMPORAL FACTORS**

Host susceptibility, pathogen exposure, and pathogen transmission occur dynamically over time within the calving season.(6) Although the adult cow-herd likely serves as the source of calf scour pathogens from year to year,(14, 12, 13, 23, 47, 35) the average dose-load of pathogen exposure to calves is likely to increase over time within a calving season because calves infected earlier serve as pathogen-multipliers and become the primary source of exposure to younger susceptible calves. This multiplier-effect can result in high calf-

infectivity and widespread environmental contamination with pathogens.(5) Each calf serves as growth media for pathogen production; growing the number of pathogens it received to even greater numbers.(18, 44, 36) Therefore, calves born later in the calving season may receive larger dose-loads of pathogens, and, in turn, may become relatively more infective by growing even greater numbers of agents. Eventually the dose-load of pathogens overwhelms the calf's ability to resist disease. These factors alone or in combination may explain observations that calves born later in the calving season are at greater risk for disease or death (Smith et al., unpublished).(11)

### **BIOCONTAINMENT OF CALF DIARRHEA**

Biosecurity is the sum of actions taken to prevent introducing a disease agent into a population (pen, herd, region), and biocontainment describes the actions taken to control a pathogen already present in the population.(15) In theory, outbreaks of calf diarrhea could be prevented by eliminating the pathogens, increasing calf resistance, or altering the production system to reduce opportunities for pathogen exposure and transmission. However, the endemic nature of the common pathogens of calf diarrhea makes it unlikely that cattle populations could be made biosecure from these agents. Maternal immunity from colostrum is clearly important to calf susceptibility to scours pathogens,(28, 37) but that protection decreases with time(7) and managers of extensive beef cattle systems have limited practical opportunities to improve calf ingestion and absorption of colostral antibodies. In addition, vaccines are not available against all pathogens of calf diarrhea, may not provide sufficient cross-protection,(26) and pathogens may evade the protection afforded by vaccination by evolving away from vaccine strains.(21) For these reasons, a biocontainment approach to control calf diarrhea seems more useful.(15, 19)

Disease causing exposure to pathogens can be prevented by physically separating animals, reducing the level of exposure (e.g. through the use of sanitation or dilution over space), or minimizing contact time. These principles have been successfully applied in calf hutch systems on dairies.(38) Various biocontainment systems for beef herds have been developed to prevent calf diarrhea.(34, 42, 33) Each of these strategies are designed to manage cattle in a system that prevents calves from having effective contacts with pathogens by reducing opportunities for exposure and transmission.

### **THE SANDHILLS CALVING SYSTEM**

The management actions defined as the Sandhills Calving System prevent effective contacts among beef calves by: 1) segregating calves by age to prevent direct and indirect transmission of pathogens from older to younger calves, and 2) scheduled movement of pregnant cows to clean calving pastures to minimize pathogen dose-load in the environment and contact time between calves and the larger portion of the cow herd. The objective of the system is to re-create the more ideal conditions that exist at the start of the calving season during each subsequent week of the season. These more ideal conditions are that cows are calving on ground that has been previously unoccupied by cattle (for at least some months), and older, infective calves are not present.

The Sandhills Calving System uses larger, contiguous, pastures for calving, rather than high animal-density calving lots. Cows are turned into the first calving pasture (Pasture 1) as soon as the first calves are born. Calving continues in Pasture 1 for two weeks. After two weeks the cows that have not yet calved are moved to Pasture 2. Existing cow-calf pairs remain in Pasture 1. After a week of calving in Pasture 2, cows that have not calved are moved to Pasture 3 and cow-calf pairs born in Pasture 2 remain in Pasture 2. Each subsequent week cows that have not yet calved are moved to a new pasture and pairs remain in their pasture of birth. The result is cow-calf pairs distributed over multiple pastures; each containing calves within one week of age of each other. Cow-calf pairs from different pastures may be commingled after the youngest calf is four weeks of age and all calves are considered low-risk for neonatal diarrhea.

It can be difficult to manage many cattle groups in intensive grass management systems; therefore, the Sandhills Calving System in these herds is modified to reduce the number of groups. Cattle are moved to different pastures throughout the calving season as appropriate for forage utilization; however, every 10 days, or whenever 100 calves are born, the herd is divided by sorting cows that had not calved from the cow-calf pairs of the preceding group. In this manner, fewer cattle groups are required, although the number of calves within any pasture group never exceeds 100, and all calves within a group are within 10 days of age of each other.

The Sandhills Calving System prevents effective contacts by using clean calving pastures, preventing direct contact between younger calves and older calves, and preventing later born calves from being exposed to an accumulation of pathogens in the environment. The specific actions to implement the system may differ between herds to meet the specific needs of each production system. Key components of the systems are age-segregation of calves, and the frequent movement of pregnant cows to clean calving pastures. Age segregation prevents the serial passage of pathogens from older calves to younger calves. The routine movement (every seven to 10 days) of pregnant cows to new calving pastures prevents the build up of pathogens in the calving environment over the course of the calving season, and prevents exposure of the latest born calves to an overwhelming dose-load of pathogens.

Development of a ranch-specific plan for implementing the Sandhills Calving System must take place well in advance of the calving season, in some circumstances in consultation with a range specialist. Available pastures must be identified and their use coordinated with the calving schedule. Water, feed, shelter and anticipated weather conditions must be considered. The size of the pastures should be matched to the number of calves expected to be born in a given week. Use of the pastures must not be damaging to later grazing.

The Sandhills Calving System may offer additional benefits to labor management. For example, there may be some efficiency because cattle movement could be scheduled once a week as labor is available. Moving cows without calves to a new pasture is often easier than sorting and moving individual cow-calf pairs. Also, the workload is partitioned between pasture groups such that cows at risk for dystocia are together in one pasture while calves at risk for diarrhea are in another. Information from pregnancy examination, when available, enables sorting cows into early and later calving groups. Cows expected to calve later in the

season can be maintained elsewhere and added to the calving pasture as appropriate, thereby reducing the number of cattle moving through the initial series of pastures.

Ranchers using the Sandhills Calving System have observed meaningful and sustained reductions in sickness and death due to calf scours, and greatly reduced use of medications.(40) Although the system was tested and initially adopted in ranches typical of the Nebraska Sandhills, it has been useful elsewhere because the principles on which it is based are widely applicable.

## CONCLUSIONS

Understanding the complex interactions that cause calf diarrhea is the basis for developing strategies for control and prevention. The common pathogens of calf diarrhea are common to most cattle herds, and it is unlikely that cattle could be made biosecure from these agents. Managers of extensive beef cattle systems have few opportunities to improve rates of colostrum uptake and absorption, and vaccines are not always protective. Colostral immunity wanes, making calves age-susceptible and age-infective. Each calf serves as growth media for pathogen production; amplifying the dose-load of pathogen it received and resulting in high calf-infectivity and widespread environmental contamination over time in a calving season. For these reasons it is logical to apply biocontainment strategies to prevent effective transmission of the pathogens causing diarrhea. Cattle management systems based on an understanding of infectious disease dynamics have successfully reduced sickness and death due to calf diarrhea.

## REFERENCES

1. Acres, S. D., C. J. Laing, J. R. Saunders, and O. M. Radostits. 1975. Acute undifferentiated neonatal diarrhea in beef calves. I. Occurrence and distribution of infectious agents. *Can. J Comp Med.* 39:116-132.
2. Acres, S. D., J. R. Saunders, and O. M. Radostits. 1977. Acute undifferentiated neonatal diarrhea of beef calves: the prevalence of enterotoxigenic *E. coli*, reo-like (rota) virus and other enteropathogens in cow-calf herds. *Can. Vet J* 18:274-280.
3. Anderson, D. C., D. D. Kress, M. M. Bernardini, K. C. Davis, D. L. Boss, and D. E. Doornbos. 2003. The effect of scours on calf weaning weight. *The Professional Animal Scientist* 19:399-403.
4. Athanassious, R., G. Marsollais, R. Assaf, S. Dea, J.-P. Descoteaux, S. Dulude, and C. Montpetit. 1994. Detection of bovine coronavirus and type A rotavirus in neonatal calf diarrhea and winter dysentery of cattle in Quebec: Evaluation of three diagnostic methods. *Can Vet J* 35:163-169.
5. Atwill, E. R., E. M. Johnson, and M. G. Pereira. 1999. Association of herd composition, stocking rate, and duration of calving season with fecal shedding of *Cryptosporidium parvum* oocysts in beef herds. *J Am. Vet Med. Assoc.* 215:1833-1838.
6. Barrington, G. M., J. M. Gay, and J. F. Evermann. 2002. Biosecurity for neonatal gastrointestinal diseases. *Vet Clin. North Am. Food Anim Pract.* 18:7-34.
7. Barrington, G. M. and S. M. Parish. 2001. Bovine neonatal immunology. *Vet Clin. North Am. Food Anim Pract.* 17:463-476.
8. Besser, T. E. and C. C. Gay. 1994. The importance of colostrum to the health of the neonatal calf. *Vet Clin. North Am. Food Anim Pract.* 10:107-117.
9. Besser, T. E., C. C. Gay, T. C. McGuire, and J. F. Evermann. 1988. Passive immunity to bovine rotavirus infection associated with transfer of serum antibody into the intestinal lumen. *J Virol.* 62:2238-2242.
10. Bulgin, M. S., B. C. Anderson, A. C. Ward, and J. F. Evermann. 1982. Infectious agents associated with neonatal calf disease in southwestern Idaho and eastern Oregon. *J Am. Vet Med. Assoc.* 180:1222-1226.

11. Clement, J. C., M. E. King, M. D. Salman, T. E. Wittum, H. H. Casper, and K. G. Odde. 1995. Use of epidemiologic principles to identify risk factors associated with the development of diarrhea in calves in five beef herds. *J Am. Vet Med. Assoc.* 207:1334-1338.
12. Collins, J. K., C. A. Riegel, J. D. Olson, and A. Fountain. 1987. Shedding of enteric coronavirus in adult cattle. *Am J Vet Res* 48:361-365.
13. Crouch, C. F. and S. D. Acres. 1984. Prevalence of rotavirus and coronavirus antigens in the feces of normal cows. *Can J Comp Med* 48:340-342.
14. Crouch, C. F., H. Bielefeldt Ohman, T. C. Watts, and L. A. Babiuk. 1985. Chronic shedding of bovine enteric coronavirus antigen-antibody complexes by clinically normal cows. *J Gen Virol* 66:1489-1500.
15. Dargatz, D. A., F. B. Garry, and J. L. Traub-Dargatz. 2002. An introduction to biosecurity of cattle operations. *Vet Clin. North Am Food Anim Pract.* 18:1-5.
16. El-Kanawati, Z. R., H. Tsunemitsu, D. R. Smith, and L. J. Saif. 1996. Infection and cross-protection studies of winter dysentery and calf diarrhea bovine coronavirus strains in colostrum-deprived and gnotobiotic calves. *Am J Vet Res* 57:48-53.
17. Heckert, R. A., L. J. Saif, J. P. Mengel, and G. W. Myers. 1991. Mucosal and systemic antibody responses to bovine coronavirus structural proteins in experimentally challenge-exposed calves fed low or high amounts of colostrum antibodies. *Am. J Vet Res.* 52:700-708.
18. Kapil, S., A. M. Trent, and S. M. Goyal. 1990. Excretion and persistence of bovine coronavirus in neonatal calves. *Arch Virol* 115:127-132.
19. Larson, R. L., J. W. Tyler, L. G. Schultz, R. K. Tessman, and D. E. Hostetler. 2004. Management strategies to decrease calf death losses in beef herds. *J. Am. Vet. Med. Assoc.* 224:42-48.
20. Law, J. 1916. Diseases of young calves. p. 245-261. In Special Report on Diseases of Cattle. United States Department of Agriculture, Bureau of Animal Industry, Washington DC.
21. Lu, W., G. E. Duhamel, D. A. Benfield, and D. M. Grotelueschen. 1994. Serological and genotypic characterization of group A rotavirus reassortants from diarrheic calves born to dams vaccinated against rotavirus. *Vet Microbiol.* 42:159-170.
22. Lucchelli, A., S. A. Lance, P. B. Bartlett, G. Y. Miller, and L. J. Saif. 1992. Prevalence of bovine group A rotavirus shedding among dairy calves in Ohio. *Am J Vet Res* 53:169-174.
23. McAllister, T. A., M. E. Olson, A. Fletch, M. Wetzstein, and T. Entz. 2005. Prevalence of *Giardia* and *Cryptosporidium* in beef cows in southern Ontario and in beef calves in southern British Columbia. *Can. Vet J* 46:47-55.
24. Mebus, C. A., E. L. Stair, M. B. Rhodes, and M. F. Twiehaus. 1973. Neonatal calf diarrhea: propagation, attenuation, and characteristics of coronavirus-like agents. *Am J Vet Res* 34:145-150.
25. Morin, M., S. Lariviere, and R. Lallier. 1976. Pathological and microbiological observations made on spontaneous cases of acute neonatal calf diarrhea. *Can. J Comp Med.* 40:228-240.
26. Murakami, Y., N. Nishioka, T. Watanabe, and C. Kuniyasu. 1986. Prolonged excretion and failure of cross-protection between distinct serotypes of bovine rotavirus. *Vet Microbiol.* 12:7-14.
27. Naciri, M., M. P. Lefay, R. Mancassola, P. Poirier, and R. Chermette. 1999. Role of *Cryptosporidium parvum* as a pathogen in neonatal diarrhoea complex in suckling and dairy calves in France. *Vet Parasitol.* 85:245-257.
28. Nocek, J. E., D. G. Braund, and R. G. Warner. 1984. Influence of neonatal colostrum administration, immunoglobulin, and continued feeding of colostrum on calf gain, health, and serum protein. *J Dairy Sci.* 67:319-333.
29. Nydam, D. V., S. E. Wade, S. L. Schaaf, and H. O. Mohammed. 2001. Number of *Cryptosporidium parvum* oocysts of *Giardia* spp. cysts by dairy calves after natural infection. *Am J Vet Res* 62:1612-1615.
30. O'Handley, R. M., C. Cockwill, T. A. McAllister, M. Jelinski, D. W. Morck, and M. E. Olsen. 1999. Duration of naturally acquired giardiasis and cryptosporidiosis in dairy calves and their association with diarrhea. *J Am Vet Med Assoc* 214:391-396.
31. Odde, K. G. 1988. Survival of the neonatal calf. *Vet Clin. North Am Food Anim Pract.* 4:501-508.
32. Odde, K. G. 1996. Reducing neonatal calf losses through selection, nutrition and management. *Agri-Practice* 17:12-15.
33. Pence, M., S. Robbe, and J. Thomson. 2001. Reducing the incidence of neonatal calf diarrhea through evidence-based management. *Compendium on Continuing Education for the Practicing Veterinarian* 23:S73-S75.
34. Radostits, O. M. and S. D. Acres. 1983. The control of acute undifferentiated diarrhea of newborn beef calves. *Vet Clin. North Am. Large. Anim Pract.* 5:143-155.

35. Ralston, B. J., T. A. McAllister, and M. E. Olson. 2003. Prevalence and infection pattern of naturally acquired giardiasis and cryptosporidiosis in range beef calves and their dams. *Vet Parasitol.* 114:113-122.
36. Saif, L. J., D. R. Redman, P. D. Moorhead, and K. W. Theil. 1986. Experimentally induced coronavirus infections in calves: Viral replication in the respiratory and intestinal tracts. *Am J Vet Res* 47:1426-1432.
37. Saif, L. J. and K. L. Smith. 1985. Enteric viral infections of calves and passive immunity. *J Dairy Sci.* 68:206-228.
38. Sanders, D. E. 1985. Field management of neonatal diarrhea. *Vet Clin. North Am. Food Anim Pract.* 1:621-637.
39. Schumann, F. J., H. G. Townsend, and J. M. Naylor. 1990. Risk factors for mortality from diarrhea in beef calves in Alberta. *Can. J Vet Res.* 54:366-372.
40. Smith, D. R., D. M. Grotelueschen, T. Knott, and S. Ensley. 2004. Prevention of neonatal calf diarrhea with the sandhills calving system. *Proc Am Assoc Bov Pract* 37:166-168.
41. Swift, B. L., G. E. Nelms, and R. Coles. 1976. The effect of neonatal diarrhea on subsequent weight gains in beef calves. *Vet Med. Small Anim Clin.* 71:1269, 1272-
42. Thomson, J. U. 1997. Implementing biosecurity in beef and dairy herds. *Proc Am Assoc Bov Pract* 30:8-14.
43. Trotz-Williams, L. A., B. D. Jarvie, S. W. Martin, K. E. Leslie, and A. S. Peregrine. 2005. Prevalence of *Cryptosporidium parvum* infection in southwestern Ontario and its association with diarrhea in neonatal dairy calves. *Can. Vet J* 46:349-351.
44. Uga, S., J. Matsuo, E. Kono, K. Kimura, M. Inoue, S. K. Rai, and K. Ono. 2000. Prevalence of *Cryptosporidium parvum* infection and pattern of oocyst shedding in calves in Japan. *Vet Parasitol.* 94:27-32.
45. USDA. 1997. Part II: Reference of 1997 beef cow-calf health and management practices.
46. Van Es, L. 1932. White scours. p. 504-513. *In* The Principles of Animal Hygiene and Preventive Veterinary Medicine. John Wiley and Sons, Inc., New York.
47. Watanabe, Y., C. H. Yang, and H. K. Ooi. 2005. Cryptosporidium infection in livestock and first identification of *Cryptosporidium parvum* genotype in cattle feces in Taiwan. *Parasitol. Res.* 97:238-241.