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Research Progress in Hemorrhagic Bowel Syndrome

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Hemorrhagic Bowel Syndrome (HBS, also known as Jejunal Hemorrhage Syndrome, bloody gut, or dead gut) is a newly emerging, highly fatal intestinal disease of adult dairy cows in the United States. HBS is characterized by sudden, progressive, and occasionally massive hemorrhage into the small intestine, with subsequent formation of clots within the intestine that create obstruction. Affected segments almost inevitably die, releasing toxins into the cow's bloodstream and abdominal cavity.

Successful treatment of this disease is difficult. Anecdotal reports exist of successful treatment with fluids, laxatives, anti-inflammatory drugs, antibiotics, and surgery; however, it appears that such treatment successes are quite rare. With or without treatment, death of affected cattle usually occurs within several hours to 1-2 days after the onset of clinical signs. At the CSU Veterinary Teaching Hospital, only about 10% of cows affected with HBS have survived.

The disease is seen most commonly in adult dairy cows early in lactation. Thus, factors specific to fresh cow management, nutrition, or the physiologic stress of peak lactation could play a role in the development of this disease. Occasional cases occur in late lactation or the dry period, and heifers are rarely affected. Although HBS usually occurs as a sporadic disease on most dairies, several cows in a herd may be affected in a relatively short period of time (i.e. "clusters" of cases). Anecdotal reports exist of dairies that struggle with this disease on virtually a continual basis.

Research on potential causes: Clostridium perfringens type A is a bacterial organism that has been associated with HBS. This organism has been documented to cause disease in a variety of birds and mammals, including cattle. The primary disease-causing factor for this organism is a potent, lethal toxin called alpha toxin that is released from the organism during rapid growth. The alpha toxin acts as an enzyme that destroys cell membranes. It is lethal to a variety of cells, including intestinal cells and red blood cells.

Some strains of *C. perfringens* type A also carry the beta2 toxin gene; these strains are designated as **A+beta2**. Beta2 toxin is also lethal for intestinal cells. The distribution of *C. perfringens* strains that carry this toxin gene is not well described, but has been identified in the intestine of both healthy and diseased cattle, horses, pigs, sheep, and goats. One or both of these organisms may be isolated from a cow with HBS.

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Winter Dysentery

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Winter Dysentery is a viral disease resulting in very acute onset of profuse watery diarrhea in adult cattle. Winter Dysentery typically occurs in the winter months, most commonly from November through March, in the northern states of the U.S. It has also been observed in beef and feedlot cattle. While outbreaks rarely occur in successive years within a herd, they may occur at intervals of 3 to 15 years.

Winter Dysentery is characterized by its highly explosive epidemic nature within a herd. Animals are often normal the day before and then suddenly break with severe watery diarrhea. Up to 30-50% of the herd can be affected within just a few days and up to 100% within a week. The feces are typically dark green, dark brown, or may appear black. Frank blood will be observed in the feces of some cows. Most animals do not demonstrate an elevated temperature once the diarrhea is present; however, there may be a fever just prior to becoming sick. The cattle are often bright and alert with minimal systemic signs of disease. They generally maintain rumen activity and continue to eat while they are sick. Milk production is

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Important Dates: Mark Your Calendar

March 8 –11, 2005: Western Herd Management Conference – Reno, NV
Contact: http://www.wdmc.org or w.wailes@colostate.edu

October 10 – 24, 2005: A European Dairy Tour – Through Germany and the Netherlands.
Contact: w.wailes@colostate.edu or information@venteuro.com.

Integrated Livestock Management

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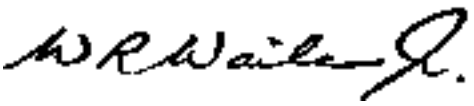
A Message From Your Extension Dairy Specialist.....

After ten years of publishing and mailing the newsletter, The Colorado Dairy News is going electronic! The cost of printing and mailing has become burdensome. With so many people using the Internet it seems far more practical to have the newsletter available electronically. The newsletter will still be produced bimonthly but you must use the internet to read it. This issue (January/February) and the next (March/February 2005) will come to you in hard copy by snail mail.

To receive the May 2005 issue you must register on line. To subscribe to the electronic version, go to:
http://www.ansci.colostate.edu/news/dairynewsubscribe.html >.
The directions are easy to follow.
If you have any questions, please email Ragan Adams at <radams@colostate.edu>.

There are two important events on the calendar that I would like to encourage you to attend. I hope to see you all at the The Western Dairy Management Conference in Reno. This important conference occurs every two years. Please make every effort to attend. Secondly, there is an opportunity to go to Germany and the Netherlands next fall to visit many aspects of their dairy industry. Frank Garry of CSU has helped me line up a wonderful tour. Frank used to work at the University of Munich before he came to CSU so we will get an insider's view. It will be a lot of fun. Please call me for more information.



William R. Wailes, Colorado Extension Dairy Specialist

Commodity Price Quotes

By-Product Feeds	Price/Ton Spot Loads	Price/Ton March/Sept/Clock
Bakery Waste	$95.00	NQ
Blood Meal (Porcine)	$390.00	NQ
Corn Gluten Feed	$92.00	$92.00 - Clock
Corn Hominy	$81.00	$85.00 - March/Sept
Flaked Corn	$102.00	$106.00 - March/Sept
Whole Corn	$87.00	$91.00 - March/Sept
Whole Cottonseed	$155.00	$155.00 - Clock
Distillers Grains	$96.00	$90.00 - March/Sept
Pork - Meat & Bone Meal	$185.00	NQ
Tallow	$0.20/lb	NQ
SBM - 48%	$193.00	$188.00 - March/Sept
Wheat Middlings	$78.00	$78.00 - Clock
Soybean Hulls	$90.00	$95.00 - March/Sept
Canola	$140.00	$142.00 - March/Sept
Corn Gluten Meal	$270.00	NQ

These price quotes are delivery at Greeley, Co

(Winter Dysentery, continued from page 1)

often decreased anywhere from 5 to 30% during an outbreak and production levels may not return fully to expected normal during that lactation. Fortunately, while the number of animals affected is very high, fatal cases are very rare. Most animals recover with basic supportive treatment after about 3 days. The disease will often run its course through a herd within two weeks.

Winter Dysentery is caused by an enteric Coronavirus. Recent studies have shown that the same strains of bovine coronavirus that cause diarrhea in calves also cause Winter Dysentery in adult cattle. It has also been shown that adult cattle with low serum antibody levels to coronavirus are more susceptible to developing disease than those with high antibody levels.

It used to be thought that Winter Dysentery was caused by a bacterium, *Campylobacter fetus* var. *jejuni*. While this bacteria is associated with diarrhea in adult cattle, it is not routinely associated with the explosive breaks of diarrhea classically associated with Winter Dysentery. Bovine viral diarrhea virus (BVDV), Coccidiosis and salmonellosis can also cause herd disease outbreaks of diarrhea that may resemble Winter Dysentery. Since the herd significance and specific treatment and control are different for these diseases, it is important to establish an accurate diagnosis whenever possible. BVDV can be diagnosed by both serology and virus isolation. Bovine coronavirus can be confirmed by both electron microscopy of the feces of affected cattle or by an antigen capture ELISA test of the feces. Coccidiosis can be diagnosed with a fecal floatation test that identifies the coccidia oocysts. Salmonelosis can be diagnosed by fecal cultures.

Treatment for Winter Dysentery should focus on providing basic support for the affected cattle. Most animals will continue to eat and drink and rarely show significant dehydration. If an animal becomes dehydrated, it may be effectively treated with oral fluids. There is no evidence that the use of oral medications shorten the clinical course of the disease or the progression through the herd.

Four factors have been identified as increasing the risk within a herd for Winter Dysentery. These include:
1.The presence of adult cattle with low coronavirus titers.
2.The presence of bovine viral diarrhea virus (BVDV) in the herd.
3.Tiestall or stanchion barn housing compared to free-stall or dry lot facilities.
4.Use of equipment to handle manure and subsequently handle feed.

Some basic herd recommendations can be made that will help prevent Winter Dysentery as well as help decrease other diseases in the herd. Recent studies have demonstrated that experimental coronavirus vaccines can protect against Winter Dysentery in adult cattle. While not specifically tested, the use of calf scour vaccines that contain coronavirus antigens may be helpful in preventing Winter Dysentery in adult cattle. These vaccines are administered to pregnant cattle before calving in order to provide additional immunity to the calf via the colostrum. The immune response stimulated by these vaccines may also help prevent Winter Dysentery in the dams. Routine BVDV control and eradication practices should be established in the herd. This includes periodic serologic surveillance of cattle for BVDV and specific testing of newborn calves to identify and eliminate persistently infected animals. Focus should be directed toward basic hygiene including routine manure removal and preventing the contamination of feed or water sources with manure. Manure handling equipment should either not be used to handle feed or it should be thoroughly disinfected before handling feed. These measures will also help decrease the spread of other enteric diseases such as salmonellosis or Johne's disease.

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The association between these bacteria and HBS is based on these observations: (1) affected cows have positive fecal cultures for these organisms, (2) *C. perfringens* type A and/or type A+beta2 can be isolated from the affected segments of intestine in cows affected by HBS, (3) there is microscopic evidence of intestinal necrosis associated with heavy intestinal growth of one or both of these organisms, and (4) other enteric pathogens associated with hemorrhagic enteritis have, to date, been rarely identified in tissues or intestinal contents of affected cows. Reduced monthly incidence of HBS has occurred following administration of an autogenous *C. perfringens* vaccine to adult cows on certain dairies but data from controlled studies are not available for evaluation of such vaccines on the incidence of, or survival rate for,this disease.

Despite these observations, there are many reasons why we hesitate to consider *Clostridium perfringens* as the sole pathogen causing HBS. *Clostridium perfringens* is widely distributed in the environment, and it is considered to be part of the normal bacterial flora of the intestine of most mammals. In the past, veterinary microbiologists have been reluctant to consider *C. perfringens* type A as an important disease-causing pathogen of livestock because this organism can be readily cultured from the intestine of healthy cattle. This organism proliferates rapidly in the intestine after death, making isolation from necropsy specimens of questionable diagnostic significance. If the lethal toxins of *C. perfringens* can be demonstrated in intestinal contents and/or blood of diseased animals, it is considered more likely that the organism is causing disease, rather than simply acting as a part of the normal gut flora. Until recently, the toxins produced *in vivo* by *C. perfringens* in HBS cases had not been identified, and the genotype and density of colonization of *C. perfringens* isolated from HBS cows and normal dairy cows had not been compared.

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Members of the Integrated Livestock Management Program have recently completed research that investigated these specific questions. We have found that *Clostridium perfringens* types A and A+B_2 can be isolated from multiple sites of the intestinal tract of HBS cows at a significantly higher rate than unaffected herd mates (cows with LDA). In addition, alpha and beta2 toxin production can be demonstrated in the intestine of HBS cows, but not control herd mates with LDA.

It is unclear at present whether proliferation of, and toxin production by, *C. perfringens* occurs as part of the primary insult to the intestine, or if proliferation occurs secondary to another disease process or triggering factor. Hemorrhage into the intestine from another cause could, in theory, initiate secondary proliferation of the ubiquitous *C. perfringens*, as this organism multiplies rapidly when cattle ingest large quantities of soluble protein or carbohydrate. Once the organism proliferates, however, the toxins that it releases during rapid growth could contribute to the degradation of the intestinal wall that is so characteristic of HBS.

An alternate investigation of HBS, headed up by investigators at Oregon State University, focused on characterizing the role of ***Aspergillus fumigatus***, a mold (fungus) that can be found in livestock feeds. Genetic material of this fungal agent can be detected in the blood and intestine of affected cows. A research project involving dairy cows with HBS and dairy cows that have died of other gastrointestinal diseases (the control group) is currently being conducted in Wisconsin, Minnesota, and Oregon. The rates of isolation of *C. perfringens, Salmonella,* and bovine viral diarrhea virus are being compared among cows of these two groups. The rate of detection of *Aspergillus fumigatus* DNA by polymerase chain reaction in the tissues is also being compared. The DNA of this fungal organism was present in the tissues of a significantly greater proportion of cows with HBS than of cows that died of another GI disease. No statistical disparity was found among the two study groups for the presence of *C. perfringens*, although the authors indicated that future data may produce different results on this issue. To date, BVDV and Salmonella have been isolated from only a small fraction of cows affected with HBS.

Aspergillus fumigatus is clearly associated with HBS, and there are currently two hypotheses regarding its participation in this disease: (1) As a primary contributor to the intestinal lesion, or (2) As an agent that impairs the cow's immune system, thereby facilitating or inciting whatever disease process triggers HBS. Anecdotal reports suggest that the incidence of HBS can be reduced on dairies following the introduction of a feed supplement (Omnigen AF®) into the ration. Controlled studies on the efficacy of this product for HBS prevention are pending. When fed to immunosuppressed sheep and cows, this product has recently been demonstrated to improve certain indicators of immune function in neutrophils, a type of white blood cell that plays a critical role in an animal's defense against infection.

Preventive strategies for HBS remain somewhat speculative, given that our understanding of this disease is incomplete. It may be best to first consider all proposed causes or risk factors (e.g. bacteria, fungi, reduced host disease resistance) and take measures to mitigate these potential risk factors. In so doing, one should consider: 1) identifying and correcting management and environmental factors that might impair cow immunity, 2) performing a careful partial budget analysis of the cost of specific preventive measures, and 3) deciding upon which specific corrective measure(s) might be most justified for a particular dairy.

The authors currently recommend a thorough analysis of transition and fresh cow management to identify problems with cow comfort, nutrition, and disease control that might impact disease resistance during peak lactation. Ration formulation and feed management should be reviewed, focusing on effective fiber and soluble carbohydrate content to limit potential dietary influences on gut flora, assessment of feed bunk and pen management to maintain consistent feed and nutrient intake, and a review of commodity handling and silage management to limit spoilage and mold formation. Since these critical areas impact numerous facets of cow health other than HBS, identification and correction of problems in these areas will likely provide an overall benefit to cow health. Potential use of feed additives or vaccines directed against specific, potential contributory pathogens (i.e. *Aspergillus* and *Clostridium perfringens*) should be considered carefully, with the costs of the proposed interventions and their potential efficacy weighed against the costs of the disease.

Current studies at CSU concentrate upon a comparison of the characteristics of *C. perfringens* isolated from HBS cows with characteristics of the same organism isolated from normal cows. Preliminary genetic analysis of isolates taken from HBS cases indicates that these bacteria appear to be genetically diverse. However, toxin production is greater in *C. perfringens* isolated from animals with HBS than in the same organism isolated from herd mates without HBS. Variation in the amount of alpha toxin produced is known to occur among different isolates of *C. perfringens* type A. What induces these changes in *C. perfringens* in cows with HBS? Is it the presence of blood in the intestine? A feed change? A signal from *A. fumigatus*? It is apparent that both *Aspergillus fumigatus* and *Clostridium perfringens* types A and A+beta2 can be demonstrated in the tissues and/or blood of cows affected with HBS. Do these bacterial and fungal agents act together or independently? These questions are under investigation, and hopefully, greater understanding of the pathogenesis of this troublesome disease is forthcoming. The funding provided to the ILM by dairy producers has helped us make progress in our research on HBS, and the authors wish to express their sincere appreciation for that support.