Beef Sessions

Moderator: Buzz Iliff

Review: Pre-weaning Calf Pneumonia and Management Considerations in Beef Cattle Operations

Gregg A. Hanzlicek[1], *DVM;* **Brad J. White**[1], *DVM, MS;* **Mark F. Spire**[2], *DVM, MS, DACT*
[1]*Department of Clinical Sciences, Kansas State University, Manhattan, Kansas 66506*
[2]*Intervet / Schering Plough, Manhattan, Kansas 66502*

Abstract

Pre-weaning calf health continues to be an important factor in the profitability of beef-cattle operations. Dystocia, diarrhea and pneumonia are the most common syndromes reported as contributing to calfhood disease. Risk factors for preweaning morbidity and mortality include age of dam, environmental temperature at birth, period of birth-time within the calving season, and cow/calf population density. Because calves are born agammaglobunemic, passive transfer provided by colostrum consumption continues to be important. Total immunoglobulin absorption is controlled by colostrum IgG concentration, the amount consumed, and the amount of time elapsing from birth to ingestion. Neonatal diarrhea and calf pneumonia can occur at different calf ages, with pneumonia occurring over a longer calf-age range; therefore, management for pneumonia must concentrate on the entire birth-to-weaning time period. A challenge in stimulating immunity to respiratory pathogens is selecting the appropriate vaccine at the most likely time to generate a protective response. Stimulation of active immunity in calves is somewhat dependent on colostrum quality and quantity consumed, the pathogen protected against, and the time period when maternal antibodies decrease to non-interference levels may be unique for each calf. Neutralizing antibody measures suggest that calves vaccinated in the presence of high maternal antibodies do not mount an immune response. However, some studies suggest vaccination, in the presence of maternal antibodies, results in a response not measurable by neutralizing antibodies, but nonetheless protective against clinical illness. The objective of this paper is to describe risk factors and control measures for pre-weaning diseases specifically focused on pneumonia.

Résumé

La santé des veaux avant le sevrage demeure toujours une facette importante de la profitabilité des productions de bœuf et de bétail. La dystocie, la diarrhée et la pneumonie sont les syndromes qui contribuent le plus aux maladies chez les veaux. Les facteurs de risque contribuant à la morbidité et à la mortalité sont l'âge de la mère, la température environnementale à la naissance, la période de l'année au moment du vêlage et la densité de la population des vaches allaitantes. Comme les veaux naissent sans gamma globulines, le transfert passif que permet la consommation de colostrum demeure important. L'absorption d'immunoglobulines totales dépend de la concentration des IgG dans le colostrum, de la quantité consommée et du laps de temps entre la naissance et l'ingestion. La diarrhée néonatale et la pneumonie des veaux peuvent prendre place à des moments différents dans la vie des veaux et la pneumonie peut avoir lieu sur une plus longue période durant la vie du veau. Par conséquent, la régie de la pneumonie doit considérer toute la durée de la période entre la naissance et le sevrage. L'un des défis de la stimulation immunitaire contre les pathogènes respiratoires est le choix du bon vaccin et du moment de l'inoculation le plus propice à la génération d'une réponse de protection. La stimulation de l'immunité active chez les veaux dépend d'une certaine façon de la qualité et de la quantité de colostrum ingéré et du type de pathogènes visés par la protection. De plus, la période de temps durant laquelle la concentration des anticorps maternels s'abaisse à des niveaux ne causant pas d'interférence peut varier d'un veau à l'autre. Les concentrations d'anticorps neutralisants suggèrent que les veaux vaccinés lorsque la concentration des anticorps maternels est élevée ne développent pas une réponse immunitaire. Toutefois, certaines études laissent entendre que la vaccination en présence d'anticorps maternels déclenche une réponse qui n'est pas mesurable avec les anticorps neutralisants mais qui permet quand même une protection contre les maladies cliniques. L'objectif de cet article est de décrire les facteurs de risque et les mesures de contrôle concernant les maladies avant sevrage se rapportant surtout à la pneumonie.

Introduction

Pre-weaning pneumonia can significantly impact livestock operations and individual cattle. The incidence of the disease is lower in the pre-weaning time frame relative to stocker or feeder phases of production, but this syndrome is still present in suckling calves. The preservation of pre-weaning calf health is an important factor in the profitability of cow-calf operations, as the sale of calves at weaning (five to nine months of age) is the primary source of income for cow-calf ranches.[16] Morbidity and mortality, particularly those associated with diarrhea and pneumonia between birth and weaning, influence both the number and final weight of calves sold.[2,18] Profitability in a cow-calf operation is impacted by the efficiency of the system, which can be measured by the pounds of calf sold per female exposed to the bull during the breeding season. While reproduction is the primary driver, disease can decrease the pounds of calves weaned, and the long-term sustainability of an operation is greatly influenced by controlling pre-weaning diseases to minimize impact.

Disease prevention in a population is based on the ability to include all components of the standard epidemiological triad: host, environment, and pathogen risk factors.[34] Incorporating the triad concept into the herd disease management decision-making process can significantly increase efficiency, reduce cost of production, and increase herd profitability.[52] This concept has been effectively proven through novel disease prevention strategies, such as the Sandhills Calving System, to control neonatal diarrhea in beef calves.[41] In the case of pre-weaning pneumonia, questions arise as to the risk of a pneumonic event occurring, the inciting factors associated with an outbreak and approaches to lessen the severity of an outbreak. The objective of this paper is to provide background information on the risk factors for pre-weaning diseases and management approaches to address pre-weaning respiratory disease.

Prevalence and Risk Factors for Preweaning Diseases

The prevalence of pre-weaning morbidity and mortality has been estimated by several studies.[4,48] The NAHMS 97 (National Animal Health Monitoring System) survey reported 5.5% of all calves born alive were lost or died before weaning.[48] This is similar to a Canadian survey reported by Dutil of estimated preweaning mortality rates between 4.9 and 5.6%.[12] The 1997 NAHMs survey reported the rate of death loss by perceived causes included: weather (20.2%), unknown (17.7%), respiratory problems (16.3%), digestive problems (14.4%), calving problems (13.9%), predators (6.4%), and poisoning (1.3%).[48] These findings differ slightly from the 1993 CHAPS study which showed the most frequent cause of calf death reported to be dystocia (33.0%) with digestive and respiratory deaths at 16.4% and 8.8%, respectively.[49] In a fifteen year study involving 798 necropsies of calves dying before weaning, 50.9% of the deaths were due to dystocia, with pneumonia and diarrhea as the second-most common causes.[4] The two primary disease syndromes associated with pre-weaning sickness and death losses are diarrhea and pneumonia. The 1997 NAHMs survey reported that 5.1% of herds had at least a 2% incidence of diarrhea and 14.1% of herds reported at least a 2% incidence of pneumonia.[48] A much lower morbidity rate for pinkeye and foot-rot in pre-weaned calves was also reported at 0.5% and 1.95%, respectively. In a South Dakota study of 752 calves at three different facilities, the prevalence of specific diseases among morbid calves was: diarrhea (21.4%), fever of unknown origin (22.3%), respiratory disease (15.5%), footrot (14.6%), navel ill (10.7%) and other diseases (14.6%). In a 1999 Canadian survey, 57.9% of the herd owners with 40 or more cows reported experiencing a problem with calf diarrhea, and 35.6% reported problems with calf pneumonia.[12]

The conditions for potential disease events during the suckl ing period of a calf's life actually begin at birth. Dystocia is the leading cause of pre-weaning calf death and is also a major contributor toward increased pre-weaning morbidity risk.[37] Heifers are more likely to have dystocia and their offspring are more likely to succumb to perinatal mortality, when compared to cows and their calves.[5] Offspring resulting from dystocia have been reported to have 12.9 (95% CI 8.1, 20.3) greater odds of dying compared to calves born without difficulty.[35] Prevention of dystocia as a management approach will aid in reducing the risk of pre-weaning morbidity and mortality.

Age segregation and timing of the calving season are two of the basic tenants of a pre-weaning pneumonia management program. The timing of calf birth influences risk of mortality from both climatic and potential disease pathogen exposure factors. Low temperatures at birth can increase pre-weaning mortality risk.[35] Recent research illustrates that calves born early in the calving season have a decreased risk of mortality compared to calves born later in the season.[45] Smith *et al* reported that the probability of death due to diarrhea increased for calves born later in the season.[41] This phenomenon is likely related to the level of exposure to pathogens in the environment. Older calves were exposed to a lower level of pathogens early in life (during the high-risk time period for diseases like diarrhea), while calves born later in the season may be exposed to higher levels of pathogens due to an increasing population density. Segregation of calves according to age, timing the calving season to match milder environmental conditions,

and practicing feeding practices that do not allow adult cows to congregate in small areas for long periods of time may decrease the young calf's exposure to high pathogen levels.[2, 22]

Age-associated incidence risk of neonatal diarrhea and pre-weaning pneumonia differ. Diarrheal infections from *Escherichia coli*, rotavirus and coronavirus occur most commonly in calves less than three weeks of age.[36] Saif *et al* suggests age of susceptibility varies by pathogen and has estimated age ranges for several common pathogens, including *E. coli* (< 7 d), rotavirus (7 to > 30 d), coronavirus (7 to > 30 d), and *Cryptosporidium* (7 to 21 d).[36] In contrast, pneumonia pathogens such as bovine herpes virus-1, bovine respiratory syncytial virus, bovine viral diarrhea virus, and *Mannheimia haemolytica* may occur in the broad range of time from birth to nine months of age. Bovine respiratory coronavirus has been reported to play a causative role in outbreaks of young-calf pneumonia, and if acting similarly to coronavirus found in human respiratory disease, it may act on host tissues by escaping the antiviral actions of cytokines.[21,44,54] Pre-weaning bovine respiratory disease (BRD) may occur at any time during this production phase, but research on a single, large herd over 20 years illustrated that the highest BRD incidence rates occurred between calf ages of 70 and 170 days.[43] Because of this wide age disparity, management focus on respiratory disease control needs to be over the entire period from birth to post-weaning. In cow-calf operations this will be challenging as calves usually spend most of this phase in pasture or range environments, making early disease detection and intervention difficult.

Similar to BRD in post-weaned cattle, pneumonia in young calves is a multi-factorial disease complex associated with several risk factors. Muggli-Cockett *et al* examined BRD in over 10,000 head from birth to harvest and found that dam age impacted the risk for onset of BRD. In this work, calves born to two-year old dams had increased risk for pre-weaning pneumonia, but decreased risk in the feedlot phase when compared to calves from older dams.[25] One potential explanation for these findings could be the association between dam age and passive immunity. If younger dams transferred lower levels of passive immunity to their calves, the calves could be at a higher risk for pneumonia. Conversely, relatively low levels of passive immunity may have allowed for development of active immunity, decreasing the disease risk in the feeding phase. In another study, the occurrence of BRD in the pre-weaning phase was not shown to influence the risk of BRD onset in the feeding phase.[42] In a study investigating management practices as risk factors for morbidity and mortality in dairy heifers, it was found diagnosis with scours increased risk of subsequent pre-weaning pneumonia.[40] The increased risk of pneumonia could be due to a previously challenged immune system from the diarrhea incident or the fact that this is a subset of the population already more susceptible to disease. In either case, good general management practices to decrease disease rates from either syndrome (diarrhea or pneumonia) could serve to decrease incidence risk for both in the herd.

Population dynamics plays a significant role in risk to pre-weaning pneumonia. Dutil *et al* identified a positive association between the length of calving season and pre-weaning pneumonia mortality risk in Canadian cow-calf herds.[12] In addition, the owners of larger herds (>40 cows) reported more problems due to respiratory disease.[12] Increased risk due to population density and calving season length may be explained in part by the fact that older animals serve as common carriers of many of the respiratory pathogens (i.e. bovine herpes virus-1) and potentially spread the virus through saliva.[1] As the calving season length increases a relatively higher number of older, potentially disease-harboring, animals will be present in the herd when younger, more susceptible calves are born. In addition, larger herd sizes allow more opportunities for the pathogens to establish carrier animals, and population density may influence pathogen transmission.

Genetics may play a role in pre-weaning BRD onset. Heritability of BRD occurrence has been estimated to be relatively low (0.07 to 0.19) in the pre-weaning phase.[25,42] However, one study did note significant differences between breeds of cattle.[42] These findings indicate there is a genetic influence on animal health; however, concentrating solely on genetically influenced pneumonia resistance will likely result in slow improvements to calf health.

Management Approaches to Controlling Pre-weaning Pneumonia

Colostrum and Passive Immunity

While the immune system begins to develop *in utero* and is capable of responding to antigenic challenge, the bovine fetus is born agammaglobulinemic because the syndesmochorial placenta prevents the mixing of maternal and fetal blood.[3, 33, 38, 51] As a result, the bovine neonate is dependent on the consumption of colostrum for immediate immunological protection. Colostrum is known to contain numerous antibacterial or antiviral components such as IgG, complement, lactoferrin, lipids, mucin, and lactoadherin.[3] Ruminant colostrum is unique because the primary immunoglobulin present is IgG (85% of the total immunoglobulins) and not IgA, as in monogastrics.[3,36] Failure of passive transfer (FPT) occurs when adequate levels of IgG are not absorbed by the calf from colostrum.

Failure of passive transfer is considered to be a major risk factor for pre-weaning morbidity and mortal-

ity. Failure of passive transfer is defined differently by investigators, but the most common criteria is serum concentrations less than 1,000 mg IgG/ dL of serum.[3,17] Using this criteria, within-herd prevalence of FPT in beef calves is reported to be from 11% to 31%.[30] In a study examining 1,568 calves from birth to weaning over three calving seasons, calves classified with failure of passive transfer (IgG ≤ 800mg/dL) were 2.24 times more likely to become ill and 2.7 times as likely to die before weaning.[11] In another study of 263 crossbred beef calves, those with <800mg/dL IgG were at greater risk of pre-weaning morbidity (OR 3.2), neonatal morbidity (OR 6.4) and preweaning mortality (OR 5.4) compared to those with levels > 800 mg/dL.[53] Measuring serum protein values is an alternative method of accessing passive transfer, and a serum protein value of 5.2 g/dL equates to approximately 1,000 mg/dL IgG.[47] Designating failure of passive transfer as serum protein levels < 5.5 g/dL, Courtney *et al* found calves with FPT were 3.07 times more likely to become ill as calves with levels above 5.5 g/dL.[10]

Absorption of IgG is accomplished by enterocytes through the active process called pinocytosis. This process is non-selective, and all macromolecules appear to be absorbed equally well. Absorption is initiated by macromolecules themselves, and as they compete for absorption sites, saturation does occur.[3] This results in a decline of absorptive efficiency as immunoglobulin concentration increases.[46] In order for this macromolecule absorption mechanism to be efficient, fetal cortisol must be present in high levels.[9] One reason premature calves and those removed by elective cesarean section before the birthing process begins, have lower immunoglobulin uptake is they do not experience the normal cortisol release that occurs during the birthing process.[39] This reduced absorptive ability is thought to result from lowered cortisol levels, the immaturity of the intestinal structure, lowered blood glucose and oxygen levels.[39] These findings may explain, at least in part, the increased risk of pre-weaning morbidity and mortality noted in calves that endure a difficult birth.

The calf's ability to absorb immunoglobulins is diminished by 24 hours post-birth, although delayed colostrum feeding can extend this time to 36 hours.[36,51] Although absorption is possible for 24 hours after birth, the optimal absorption occurs during the first six hours. The major influencers of IgG1 absorption are total IgG1 ingested and the period of time from birth to colostrum ingestion.[38] The IgG absorbed from colostrum has a half-life of approximately 11 to 16 days, with most of the neonatal clearance of the immunoglobulin occurring through the intestinal lumen.[6] By some estimation this re-excretion of immunoglobulins into the intestinal lumen does provide some level of protection against enteric diseases.[6] In one experiment, colostrum-derived IgG rotavirus antibodies were present in the feces of calves for at least 22 days post colostrum consumption.[28]

Passive immunity continues to be the primary source of immune protection for the neonatal calf. Anything that interferes with normal parturition or colostrum consumption can reduce neonatal passive protection. Assuring normal parturition, enhancing colostral antibody content, maintaining a sound pre-calving nutritional program to encourage quality colostral production and assuring pairing-up of dams and calves are management steps that can reduce failure of passive transfer in beef cattle operations.[7, 27, 31]

Very few studies have provided concrete timing guidelines for vaccinating young calves against non-enteric diseases in the presence of maternally derived antibodies. If maternal antibodies block a primary immune response to vaccination, one might conclude that the administration of respiratory vaccines should be delayed until traditional weaning, as passive immunity to common bovine respiratory viruses may persist for nine months.[46] In a study by Fulton *et al*, the average time for passively acquired BVD titers to decay to negative status (<16) was 192 days.[18] But because the initial titers ranged from 4 to 16,384, the calculated range to seronegativity was 4 to 299 days after first evaluation, which was approximately at 60 days of age.[18] Other studies have concluded passive immunity to *Mannheimia haemolytica* and *Pasteurella multocida* were at their lowest point 60-90 days of age.[32] A decision to delay primary immunization may be costly as the passive immune status of each calf, the actual level of protection from any given gammaglobulin titer and the date of final antibody disappearance are unknown.[9] The percentage of calves at risk for disease based on these factors varies by the herd immunity and important pathogens for the specific herd.

Neonatal Vaccination and Disease Control

One of the major challenges in pre-weaning management is the timing of vaccination in the presence of maternal antibodies. For each calf there is a unique period of time after birth where maternal IgG is waning but immunocompetence is not complete.[24] This presents a time period where susceptibility to calfhood diseases is elevated, and this phase lasts until active immunity has reached protective levels for the current disease challenge.

All components of the acquired immune system, primarily consisting of memory lymphocytes and antibody, are present at birth. These components are present, even without passive transfer, although at much lower levels than found in adults.[17,21] By some estimation, B-cells make up only 4% of the total lymphocyte population at birth but they increase to approximately 30% by the first week of age.[17] T-cell populations among neonates

are comparable to adult levels, although memory T-cell populations are absent at birth.[20] Serum levels of both T-cells and B-cells decrease at birth as these cells are sequestered in lymphoid tissue.[20] In addition, it appears that peripheral T- and B-cells may not be fully functional until two to three weeks of age.[21] This is in contrast to substantial populations of both $CD4^+$ and $CD8^+$ T lymphocytes in mucosal associated lymphoid tissue in neonatal cattle.[23] These types of cells have been shown to be functional and capable of response to antigens in neonatal ruminants.[26] Traditional studies that have investigated passive transfer interference measure antibody changes after vaccination. More recent studies have evaluated innate and active immunity responses in the presence or absence of maternal antibodies. Cell-mediated and B-cell memory responses have been demonstrated using attenuated vaccines in the presence of maternal antibodies.[13,14] These findings may provide support for the development of novel attenuated vaccination products for use in very young calves to provide a primary memory response. Potential differences between vaccine types were evaluated in a bovine viral diarrhea study where calves with circulating maternal antibodies were vaccinated at seven weeks of age. Calves vaccinated with modified-live (MLV) BVDV mounted a T-cell response, while calves given killed vaccine did not develop a T-cell response.[15] This is suggestive that memory cells were stimulated during a primary immune response in the presence of maternal antibodies.

The route of vaccination administration in young calves may influence post-vaccinal immunity. Vaccines delivered intranasally have been found to be more protective against a clinical challenge compared to parenterally administered products in BHV-1 seronegative three-month-old calves or in calves possessing maternal antibodies.[8] Those calves receiving the intranasal vaccination did not become febrile, were not dyspneic, did not have nasal discharge, were not observed coughing, whereas some of all the calves vaccinated with the intramuscular product were observed with at least one clinical sign.[8] In this study, the route of administration appeared to impact the level of protection against challenge when vaccinating young calves. In another study, two-week-old Holstein-Hereford cross calves, with evidence of maternal antibodies, were given a single intranasal BHV-1 vaccination, then challenged 16 weeks later with live infectious bovine rhinotracheitis (IBR).[29] Although no rise in virus-neutralizing antibodies to BHV-1 was observed in any calf, all intranasally vaccinated calves were protected against pyrexia, and nasal viral shedding was decreased in this group. Based on these studies, intranasal vaccination for BHV-1 appears to provide some protection against clinical challenge even when administered to young calves.

Research examining calves possessing maternal antibodies to BRSV evaluated vaccination with a modified-live BRSV-PI3 intranasal product at three weeks of age, followed by a live BRSV challenge 66 days later. Compared to controls, no vaccinated calf was observed to be in the severe category for all clinical parameters measured except nasal discharge.[50] Measuring virus-neutralizing antibodies in young calves, although a long held standard measurement of immune status, may not provide a complete picture of the actual degree of protection from exposure to potential pathogens. Because the B- and T- cell population numbers and their functionality are decreased in the neonatal calf compared to older calves and adults, it is possible that the time from pathogen exposure to immune response may be prolonged. However, in young calves vaccinated with BHV-1 vaccine the cell-mediated immunity provided a rapid degree of protection to respiratory disease challenge.[19] These new studies strongly suggest that vaccinating young calves, particularly via the intranasal route with modified live vaccines, may be capable of stimulating protection against disease challenge.

Conclusion

Pre-weaning calf pneumonia can negatively affect both the short and long-term profitability of the cow-calf producer. The occurrence of pre-weaning pneumonia is a complex interaction involving exposure of this age group of animal to potential pathogens, the level of passive transfer, and the ability of the calf to generate an active immune response. Calves are born immunologically naïve, and passive transfer afforded by colostrum ingestion remains an important component of health management. Because the level of passive transfer is a function of total immunoglobulin absorption by the calf within a few hours after birth, adequate colostrum production by the dam and timely colostrum consumption by the calf are key areas for management attention. Planning the calving season to employ management practices that aid in reducing total pathogen load such as age segregation of calves, moving feeding areas and rotating pastures are vital components of a disease control program. Designing young calf vaccination programs to take advantage of a developing immune system capable of rapid and protective cell-mediated responses requires an understanding of the timing of vaccine delivery and the type of product used.

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