Conglutinin and immunoconglutinin titers in stressed calves in a feedlot

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Conglutinin is part of a group of serum proteins called collectins. The term collectins describes a group of proteins that have 4 domains. These domains consist of an amino terminus rich in cysteines, followed by a collagen domain that is connected to the carboxy terminal lectin domain, which is connected to the neck protein. These 4 domains are arranged in 2 types of structures. Type-I collectins include the mannose-binding proteins and surfactant apoprotein-A. Type-II collectins include conglutinin, collagenous lectin-43 (CL-43), and surfactant protein-D, which form cruciform arrays. Conglutinin and CL-43 have only been identified in bovids. Structural aspects of collectins have been examined. Mannose-binding proteins, conglutinin, and CL-43 are synthesized in the liver and found mainly in the lungs.

Conglutinin mediates bactericidal activity against gram-negative bacteria in an in vivo system containing adherent leukocytes and intact complement. However, the role that conglutinin plays in innate immunity against acute bovine respiratory disease complex (BRDC) is unknown. One of the most important bacterial agents involved in BRDC is Mannheimia haemolytica serovar A1 (MhA1; formerly Pasteurella haemolytica serovar A1), a gram-negative bacterium. Conglutinin specifically binds to nonreducing terminal N-acetylglucosamine, mannose, and fructose residues of bacteria. Conglutinin also may inhibit certain viruses in vivo and in vitro. Conglutinin acts as a lectin that binds nonspecifically to complex bacterial polysaccharide molecules in a calcium-dependent reaction. Immunoconglutinins are antibodies that have specificity for new antigenic determinants created or exposed when complement is fixed with antigen and antibody.

Imunoconglutinin activity has been associated with increased protection against challenge-exposure with infectious bacteria. Bovine immunconglutinin appears to agglutinate relatively small complexes containing bound complement (ie, C3), which, in turn, makes the complexes more susceptible to phagocytosis. Reactions involving immunoconglutinin are independent of calcium ion concentrations. Because immunoconglutinin contribute to removal of primary antigens, they can be classified as protective antibodies.

It has been determined that total complement activity in serum of feeder calves is influenced by stress associated with marketing. In the study reported here, conglutinin and immunoconglutinin activity of feeder calves undergoing stress associated with marketing were evaluated. We examined the hypotheses that calves in feedlots that remain healthy have higher conglutinin titers are evident in stressed calves that do not develop respiratory tract disease in feedlots, compared with respiratory tract disease, and to determine the increase in immunoconglutinin titers.

Animals—101 mixed-breed beef calves.

Procedure—Calves were processed at 4 farms of origin and allowed to remain with their dams for another 100 days. Calves from each farm were brought to a centrally located order-buyer barn. In a feedlot, 101 calves were assigned to pens and observed daily for clinical signs of acute respiratory tract disease. When sick calves were detected, they were treated with antibiotics and isolated in a pen for 4 days. Conglutinin and immunoconglutinin titers were determined for all calves.

Results—During the 28-day study, 73 calves developed respiratory tract disease, whereas 28 calves remained healthy. Mean conglutinin titers differed significantly among calves from the 4 farms. Significant differences were not detected in conglutinin titers among calves on the basis of sex, morbidity, or vaccination status against Mannheimia haemolytica at each farm, the order-buyer barn, or the feedlot on days 8, 15, and 28 after arrival. Immunoconglutinin titers in calves differed significantly among farms and morbidity status.

Conclusions and Clinical Relevance—Mean conglutinin titers in calves do not appear to be associated with the incidence of acute respiratory tract disease; however, increased immunconglutinin titers appear to be associated with recovery of stressed calves from respiratory tract disease during the first 15 days after arrival in a feedlot. (Am J Vet Res 2000;61:1403–1409)
glutinin titers than those that develop acute BRDC and that calves would have increased immunoconglutinin titers after recovery from acute BRDC. Furthermore, we believed recovery of calves after acute BRDC would be attributable, in part, to inoculation with MhA1 and an increased antibody response to this respiratory tract pathogen. The increase in antibody titers to MhA1 usually is evident during recovery. Also, MhA1 in nasal secretions at the time that calves are febrile and have other clinical signs of BRDC implies that this organism is partly responsible for illness. Therefore, we believed it important in the study to determine the antibody titer against MhA1 and to detect MhA1 in nasal secretions. In addition, we examined the possibility that serum haptoglobin and plasma fibrinogen concentrations, both of which are markers of inflammation, may serve as potential prognostic indicators of the severity of BRDC, similar to the situation in goats with acute respiratory tract disease.20,21 Such indicators could be of value to veterinarians when determining treatment regimens.

Materials and Methods

Animals—Calves (n = 101) used in the study were mixed-breed English beef calves purchased from 4 farms in eastern Tennessee (farms A to D; 26, 21, 28, and 26 calves, respectively). Calves on all farms were housed with their dams in pastures. In the spring, calves were separated briefly from their dams. Rectal temperature of each calf was measured. Calves were treated with ivermectin, and an ear tag was applied. Males were castrated. All calves were vaccinated, using a combination clostridial bacterin and an intranasally administered preparation of a modified-live infectious bovine rhinotracheitis and parainfluenza-3 virus product. Calves with odd-numbered ear tags were given an experimental MhA1 bacterin-toxoid.22 After processing, calves were returned to their dams and allowed to remain in the pastures for another 100 days.

Experimental design—In the fall of that year, 101 calves (51 calves vaccinated with the MhA1 bacterin-toxoid and 50 nonvaccinates) were transported 8 to 32 km from the 4 farms to a centrally located order-buyer barn (OBB). Rectal temperatures were measured, and calves were vaccinated intramuscularly with a modified-live infectious bovine rhinotracheitis and parainfluenza-3 virus product.22 Calves with odd-numbered ear tags were given an experimental MhA1 bacterin-toxoid.22 After processing, calves were returned to their dams and allowed to remain in the pastures for another 100 days.

After 5 days in the OBB, the calves were transported 1,600 km to a USDA-Agricultural Research Service feedlot in Bushland, Tex. On arrival at the feedlot (day 0), rectal temperatures were measured, and calves were allocated to 4 pens. They were given water and a concentrate-alfalfa hay diet and allowed to rest overnight. The next day, calves were weighed, rectal temperatures were measured, and calves were allocated to 12 pens on the basis of sex and MhA1-vaccination status.

Scoring system used for selecting sick calves and a treatment regimen have been described elsewhere.18 Briefly, sick calves were identified by a system in which points were assigned on the basis of clinical signs (nasal and ocular exudate, lethargy, anorexia, and fever of ≥ 40 °C for at least 48 hours). Sick calves were treated with tilmicosin phosphate (10 mg/kg of body weight, IM), and those that remained febrile were given oxytetracycline.22 Treated calves were housed separately from their penmates in a sick pen for 4 days. After that time, if the calves appeared clinically normal, they were returned to their originally assigned pen. At the time when they entered and again when they exited the sick pen, a swab specimen of nasal mucus was collected from each calf for isolation of M haemolytica, and blood samples were processed to yield plasma and serum for determination of fibrinogen and haptoglobin concentrations.

Serologic assay—An indirect hemagglutination antibody (IHA) assay24 was used to determine mean serum titers against MhA1 and MhA6 for each calf from samples obtained when calves were at each farm of origin, at the OBB, and at the research feedlot. Samples were collected at the feedlot on days 8, 15, and 28. The IHA titers were reported as the inverse of the highest dilution that yielded a positive response. Blood samples were collected via jugular venipuncture, and serum was harvested. All serum samples for each calf were assayed on the same day to prevent variation in assay results between days. Sera with known titers were used as control samples.

Bacterial culture and isolation—Specimens of nasal mucus were obtained, using cotton-tipped swabs, from the ventral nasal meatus of each calf. Swab specimens were submitted for bacterial culture and isolation and identification of Mannheimia spp. Swab specimens were obtained from calves at each farm of origin, at the OBB, and at the feedlot on days 1, 8, 15, and 28.22

Conglutinin and immunoconglutinin assays—Titters of conglutinin and immunoconglutinin were measured in serum samples, using a method developed by Barta, with the modification22 that sheep RBC (SRCB) were sensitized with bovine serum (natural anti-SRCB antibody) instead of rabbit anti-SRCB antibody. The SRCB were prepared for assay by initially incubating them in pooled diluted bovine serum (sensitized). Cells then were incubated in a dilution of equine serum complement, resulting in sensitized SRCB (SRCB+). Complement in serum samples was inactivated at 56 °C for 30 minutes; they then were absorbed with nonsensitized SRCB (SRCB–). Serum dilutions were prepared in 12X 75-mm siliconized tubes containing 0.1 ml of isotonic barbitonal-0.147M NaCl buffer (BNB), pH 7.3, that had an ionic strength of 0.094 and an electrical conductivity of 13.8 miliSiemens. To this we added 0.1 ml of SRBC+ or 0.3 ml of BNB. Tubes were incubated in a water bath at 37 °C for 30 minutes and again at 4 °C overnight. Fetal bovine serum and adult bovine serum (stored at –84 °C) were used as negative- and positive-control samples for the assay. Other control samples included tubes that contained BNB and SRBC+ or BNB and SRBC–to ensure lack of spontaneous agglutination. Serum conglutinin activity was determined from the well with the highest dilution that had positive results, as indicated by agglutinated SRBC. Tubes with negative results contained an unagglutinated button of cells. Immunoconglutinin titers were determined by adding 0.8 ml of 0.086M Na2EDTA in 0.15M NaCl to conglutinin assay tubes to dispense agglutinated SRBC. Tubes were incubated at 22 °C for 24 hours to allow for immunoconglutinin-mediated agglutination.

Complement assay—The procedure used was that reported by Renshaw et al22 and modified by Barta and Barta.23 The classical hemolytic complement assay used was described by Purdy et al.22 Complement activity in CH50 units
was determined for each calf from samples obtained at the OBB and the feedlot on days 1, 8, and 28.

Clinicopathologic analyses—Serum haptoglobin,22 and plasma fibrinogen22 concentrations were determined for each calf from samples obtained at the OBB and at the feedlot on day 1. Haptoglobin and fibrinogen concentrations and total number of WBC were determined for each calf when it entered and again when it exited the sick pen. Assays were performed at a veterinary medical diagnostic laboratory.1

Statistical analysis—Data were analyzed, using a repeated-measures ANOVA.22 Variables included in the model were time, treatment, and time-by-treatment interaction. Differences were considered significant at a value of $P \leq 0.05$. When a significant time-by-treatment interaction was detected, data subsequently were analyzed within time (ie, day of sample collection). Significant differences between treatment groups were evaluated further, using the Bonferroni and Dunnett adjusted paired $t$-tests, which allowed pairwise comparison of means for all treatment groups within any day of sample collection. Antibody titers determined by use of IHA assay were reported as geometric means. Analyses were performed for conglutinin and immunoconglutinin titers, using data transformed in log$_2$ format.

Results

Morbidity and mortality—During the first 18 days after arrival in the feedlot, 73 calves developed BRDC, whereas 28 calves remained healthy throughout the study. One calf died while in the feedlot. The number of sick calves in each treatment group, days treated, and total number of sick days were as follows: 7 MhA1 vaccinates, each treated for 6 days for a total of 42 sick days; 34 MhA1 vaccinates, each treated 3 days for a total of 102 sick days; 5 nonvaccinates, each treated for 6 days for a total of 30 sick days; and 27 nonvaccinates, each treated for 3 days for a total of 81 sick days. Eighteen nonvaccinated calves and 10 vaccinated calves remained healthy throughout the study. Vaccination against MhA1 did not have a significant effect on incidence of BRDC.

Rectal temperature—Rectal temperatures were related to stress associated with transportation and mixing at the OBB as well as disease. The MhA1-vaccinated calves had significantly higher rectal temperatures on days 0, 1, and 3 through 7, compared with nonvaccinates. During the first 7 days in the feedlot, sick calves treated for 6 days had significantly higher rectal temperatures, for the most part, than sick calves treated for 3 days.

Conglutinin and immunoconglutinin titers—We did not detect significant differences in mean conglutinin titers between calves on the basis of morbidity, sex, or MhA1 vaccination status on any sample collection day. There was a significant difference in mean conglutinin titers of calves among the 4 farms of origin (titers of 920, 197, 640, and 325 for farms A to D, respectively; Table 1). Results of the Bonferroni adjusted paired $t$-test indicated a significant difference in titers between calves on farms A and B in samples obtained at the farm of origin. Results of the paired $t$-test indicated significant differences in titers between calves from farms A and C, and B and D in samples obtained on day 1 in the feedlot and between calves from farms A and D in samples obtained on day 8 in the feedlot. Significant differences in mean conglutinin concentration of calves from the various farms were evident in samples obtained on all sample collection days, except day 13.

Significant differences in geometric mean immunoconglutinin titers were detected in calves on the basis of morbidity (healthy, sick calves treated for 3 days, and sick calves treated for 6 days) for samples obtained on every sample collection day (Fig 1). Results of the paired $t$-test indicated differences between sick calves treated for 6 days and calves that were healthy or sick calves treated for 3 days. With the exception of samples obtained on day 13, mean

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**Table 1**—Mean conglutinin titers of calves in a feedlot, grouped by farm of origin

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<th>Farm of origin</th>
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<tr>
<td></td>
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<tr>
<td>A (n = 26)</td>
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<td>640*</td>
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<tr>
<td>D (n = 26)</td>
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*Overall significance of $P = 0.01$. †Overall significance of $P = 0.02$. ‡Overall significance of $P < 0.001$.

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**Table 2**—Mean immunoconglutinin titers of calves in a feedlot, grouped according to farm of origin. See Table 1 for key.

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<th>Samples</th>
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<td>C (n = 28)</td>
<td>82*</td>
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<tr>
<td>D (n = 26)</td>
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**Figure 1**—Geometric mean immunoconglutinin titers of calves in a feedlot in samples obtained from calves at the farm of origin (farm), at the order-buyer barn (OBB), and for various days after arrival in the feedlot (1, 8, 15, and 28), on the basis of morbidity status (healthy, sick and treated for 3 days [S3], sick and treated for 6 days [S6]). a, b = Values with different letters differ significantly ($P < 0.05$; Bonferroni paired $t$-test). OBB = Order-buyer barn.
immunoglobulin titers for sick calves treated for 3 days were slightly higher, but not significantly different, from those of calves that were healthy. Mean immunoglobulin titers peaked on day 15. Mean immunoglobulin titers were reported for calves sorted on the basis of farm of origin (Table 2).

Complement concentration (CH 50 units)—A significant ($P \leq 0.008$) difference in mean complement concentration was detected for samples collected at the OBB when calves were grouped on the basis of farm of origin. Mean ($\pm$ SD) complement concentration for calves from farms A to D at the OBB was $97 \pm 35, 73 \pm 37, 68 \pm 27$, and $126 \pm 41$ U/ml, respectively. Results of the Bonferroni paired $t$-test indicated that mean complement concentration was significantly different between calves from farms A and D and A and C. Mean complement concentrations were not significantly different in samples obtained on any of the other sample collection days. Results of the repeated-measures ANOVA did not determine significant differences when sex, vaccination status, and morbidity status were analyzed for each of the sample collection days. Mean complement concentration for 58 steers was lower than that of 38 heifers for each sample collection day. Concentrations for steers were $88 \pm 40$ U/ml at the OBB, $90 \pm 28$ U/ml on day 1, $61 \pm 27$ U/ml on day 8, and $97 \pm 29$ U/ml on day 28, whereas concentrations for heifers were $96 \pm 44$ U/ml at the OBB, $107 \pm 42$ U/ml on day 1, $65 \pm 31$ U/ml on day 8, and $113 \pm 36$ U/ml on day 28.

Clinicopathologic analyses—Mean haptoglobin concentrations were monitored in 73 calves. Concentrations were significantly different among calves in samples collected at the OBB, on day 1, when calves entered the sick pen, and when calves exited the sick pen (Fig 2). Mean haptoglobin concentration of sick calves when entering the sick pen was $189 \pm 110$ mg/dl, which was 2.36-fold higher ($P < 0.001$) than when they exited the sick pen 4 days later after a course of antibiotic treatment ($80 \pm 68$ mg/dl). Results of the repeated-measure ANOVA did not reveal significant differences among calves when concentration for day 1 was treated as a dependent variable. Mean haptoglobin concentrations on day 1 were as follows: sick calves treated for 3 days ($n = 61$), $157 \pm 154$ mg/dl; sick calves treated for 6 days (12), $217 \pm 165$ mg/dl; and calves that were healthy (28), $46 \pm 91$ mg/dl. Again, we did not detect significant differences when concentration for calves at the OBB was treated as a dependent variable. At the OBB, mean haptoglobin concentrations were as follows: sick calves treated for 3 days ($n = 61$), $3 \pm 11$ mg/dl; sick calves treated for 6 days (12), $3 \pm 4$ mg/dl; and calves that were healthy (28), $10 \pm 52$ mg/dl.

We did detect a significant ($P < 0.001$) difference in fibrinogen concentrations among samples obtained on various days; however, there was not a significant difference in mean fibrinogen concentrations on the basis of vaccination status or morbidity status (Fig 3). Mean fibrinogen concentration for the 101 calves was $230 \pm 167$ mg/dl for samples obtained at the OBB and $230 \pm 230$ mg/dl for samples obtained on day 1. Data for 72 sick calves were retrospectively analyzed. Mean total WBC counts were significantly ($P = 0.01$) less for calves when they entered the sick pen, compared with counts for those calves when they exited the sick pen (Fig 4).

Serologic titers—Geometric mean titers, determined by use of IHA testing, did not differ significantly between MhA1 vaccinates and nonvaccinates or between morbid and healthy calves (calves that never became sick during the study). Therefore, MhA1 geometric mean titer for all 101 calves was calculated for sample collection day (on the farm of origin, 5; at the OBB, 9; day 1, 14; day 8, 69; day 15, 72; day 28, 91). The MhA6 geometric mean titer for all 101 calves throughout the study was as follows: on the farm of origin, 4; at the OBB, 6; day 1, 9; day 8, 16; day 15, 15; day 28, 27. The MhA1 geometric mean titer was always 3-fold greater than the MhA6 titer on day 28, regardless of the manner in which calves were grouped for analysis.
Bacterial culture and isolation—*Mannheimia haemolytica* A1 was isolated from 4 calves, and MhA6 was isolated from 2 calves in samples obtained at the farm of origin. In samples obtained at the OBB, MhA1 was isolated from 6 calves, MhA2 from 1 calf, and MhA6 from 1 calf. There were 284 MhA1 isolates and 142 MhA6 isolates recovered from all calves during samples collected on 4 days during their stay in the feedlot.

Discussion

We are unaware of other reports on conglutinin titers in stressed feeder calves. In the study reported here, we did not detect significant differences in mean conglutinin titers in calves on the basis of morbidity status (healthy, sick calves treated for 3 days, and sick calves treated for 6 days). Significant differences in mean conglutinin titers were detected in calves from the various farms of origin on all sample collection days, except day 15. Significant differences in conglutinin titers among calves from the various farms could have been explained by differing breeding and management practices. In cattle, conglutinin concentration is a heritable trait, and those with low concentrations are more susceptible to infections. Also, variations in conglutinin concentration in calves on various farms has been attributed to differences in nutrition, season of the year, hours of daylight, and other environmental factors. It does not appear from the study reported here that conglutinin plays an important role in reducing acute BRDC in stressed feeder calves. However, another report cites the fact that conglutinating activity of ox serum decreases during systemic infections, at parturition, and in connection with abortion. Systemic infections and stress certainly could have been important factors in our study. Stressed weaned calves from the southeastern United States that are moved to feedlots in Texas usually become ill with BRDC during the first 2 weeks after arrival. In the study reported here, 73 calves became sick, and 28 calves apparently did not get sick. Stress involved with marketing and transportation may have induced a decrease in the serum conglutinin titer in the calves that remained healthy. It would be expected that sick calves with BRDC would have a decrease in conglutinin titer while host immunologic reactions were taking place. Only after the end of the infectious process would it be expected that the conglutinin titer would increase in recovering calves. If the conglutinin titers of the calves that remained healthy were decreased as a result of stressors other than infection, it would be exceedingly difficult to prove our hypothesis that stressed calves that remain healthy would have higher conglutinin titers than calves that developed BRDC. In another report, the importance of periparturient and postparturient stress on depression of the immune system (innate and adaptive immunity) were described. Specifically, conglutinin concentration and complement activity were decreased 3 weeks prior to parturition, and they reached their minimum at time of parturition. That study also revealed genetic differences among sire progeny groups that affected immune assays. Thus, it would appear that numerous varied stressors can cause a decrease in conglutinin activity.

Results of our study, which revealed that calves that remained healthy did not have significantly higher conglutinin titers than calves with active infections, were unexpected. In an in vivo study, investigators used intact complement and conglutinin to document efficient killing of *Escherichia coli*. A subsequent study by Laursen et al confirmed results of the previous study on killing of *E coli*. Again, a conglutinin-mediated enhancement of killing of *E coli* with an exceptional selectivity for the high mannose group presented on iC3b was observed. Conglutinin may preferentially attach to iC3b that is covalently attached to another protein or carbohydrate via an activated thio ester. The other carbohydrate or protein can be provided by bacteria, which are then inactivated. The structural basis of this phenomenon is unknown, but the biological implication appears to be that binding to the carbohydrate will not happen unless the presentation is altered through activation via an inflammatory process.

Although not significantly different, mean conglutinin titer of steers was less than that of heifers on each sample collection day. This apparent sex difference concerning mean conglutinin titer is similar to results obtained in other studies of complement. Overall, when conglutinin titers of stressed feeder calves were measured on the farm of origin and throughout the feedlot period, they fluctuated in a manner similar to that for concentrations of complement.

The hypothesis that mean conglutinin titers of stressed healthy calves that remained healthy would be significantly higher than those of calves that became ill appears not to be valid under the conditions of the study reported here. However, sick calves treated for 6 days had lower conglutinin titers, but not significantly so, than healthy calves.

The mean immunoconglutinin titers of calves increased over time, and it appears that these titers were at a maximum on day 15 in the feedlot, whereas the MhA1 titer was at a maximum on day 28 in the feedlot. Thus, it appears that the 2 assays were not measuring the same antibody. The hypothesis that there would be an increase in immunoconglutinin titer
as calves recovered from BRDC appears to be correct. However, this immunity was not always solid, because many calves that did not become sick frequently harbored MhA1. Thus, subclinical *M. haemolytica* infections probably develop even in calves that remain healthy. Also, MhA1 titers increased in vaccinates and nonvaccinates, regardless of morbidity status. This implies that titers increased in calves as a result of MhA1 exposure in nonvaccinates that did not become overtly sick.

In the study reported here, mean fibrinogen concentrations did not appear to be a good marker for inflammation in calves with BRDC. Fibrinogen tests are sensitive, and concentrations appeared to increase at the OBB as a result of stress alone (263 mg/dl). It also appeared that fibrinogen remained in the plasma for a long time. There was little difference in the fibrinogen concentration in the plasma of calves when they entered the sick pen (769 mg/dl) and when they exited the sick pen 4 days later (838 mg/dl). Fibrinogen concentrations did not differ significantly between sick and healthy calves.

For our purposes, mean haptoglobin concentrations appeared to be the best marker for inflammation in calves with BRDC. Seventy-two calves had significantly higher mean serum haptoglobin concentrations (189 mg/dl) when they entered the sick pen than when they exited the sick pen 4 days later (80 mg/dl) after 3 days of antibiotic treatment. Calves that remained healthy had low mean concentrations of haptoglobin. Calves at the OBB prior to developing sickness also had low mean haptoglobin concentrations.

There were significant differences in complement concentrations in calves from various farms of origin; however, complement concentrations did not differ significantly on the basis of sex, vaccination status, and morbidity status for each sample collection day. Mean complement concentrations were lower in steers than in heifers, which is in agreement with a previous report. Patterns of *M. haemolytica* isolates recovered from mucus in nasal swab specimens were typical in that few isolates were recovered from samples obtained at the farm of origin or in the OBB. The number of bacterial isolates increased dramatically after 8 days in the feedlot. It should be mentioned that 142 MhA6 nasal isolates were recovered from calves during 4 sample collection days in the feedlot, and these isolates appeared to have spread from 2 calves in which MhA6 was recovered in samples obtained on the farm of origin. These MhA6 isolates are important, because MhA6 isolates have been recovered from the lungs of feeder calves that died. These isolates also are important for 2 additional reasons. The first reason is that veterinary diagnostic laboratories do not routinely serotype isolates recovered from the lungs of feeder calves that have died, and the second reason is that vaccine manufacturers only include the MhA1 serovar in their vaccines. There is little information concerning cross-protection between *M. haemolytica* serovars. However, we have documented in goats that MhA1 may cross-protect against challenge-exposure with MhA6, but MhA6 will not cross-protect against challenge-exposure with MhA1.

We were unable to document that bovine conglutinin titers played an important role in innate immunity against MhA1 organisms. Immunoconglutinin titers probably play a minor role in specific acquired immunity against MhA1. Additional studies should be conducted to determine whether conglutinin plays a larger role in innate immunity against pasteurellosis than we were able to determine.

The cause of BRDC in stressed feeder calves is so complex that even when efficacious MhA1 vaccines are given in an approved manner, the results are often disappointing. It is known that calves whose farm of origin is in the southeastern United States are often poorly managed in regard to the administration of virus vaccines while on the farm of origin. Also, all of the pathogenic viruses associated with BRDC may have not been identified. The mean total WBC count of the calves at the time they entered the sick pen, which was in the low end of the reference range, may have been attributable to some type of virus infection that affects nonimmune calves during this time of maximum stress. Four days later, there was a significant increase to within the reference range for mean total WBC counts when the calves were considered healthy. Of the 3 inflammatory C-reactive proteins of stressed feeder calves that were determined in this study, haptoglobin holds the most promise to be predictive of BRDC severity. Four days after entering the sick pen for antibiotic treatment, a significant decrease in haptoglobin concentration appeared to be a good indicator of a positive (beneficial) response of the calves to treatment. Fibrinogen concentration does not appear to be a useful marker, because fibrinogen may linger in the blood too long to be a practical predictive indicator for the short period available for treating sick calves. When any of these inflammatory proteins are used to monitor calves, it should be remembered that stress alone (even in calves that remain healthy) will increase from an OBB to entry into a feedlot. A minimum of 2 blood samples would be required to make a clinical judgement, and these samples would have to be collected at an appropriate interval to allow an accurate assessment for treatment of BRDC.

References

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