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DAIRY CALF PNEUMONIA

The Disease and Its Impact

Trevor R. Ames, DVM, MS

Pneumonia of dairy calves or enzootic calf pneumonia is an important component of the bovine respiratory disease complex[31]; also included in this complex are shipping fever of feedlot cattle and acute respiratory distress syndrome or atypical interstitial pneumonia.[31] Dairy calf pneumonia (DCP) has traditionally been described as affecting calves from 2 to 6 months of age.[11] Recent prospective studies examining cohorts of calves have found calves may be affected with DCP as early as 2 weeks of age,[56] with peak incidence occurring at 5 to 6 weeks.[44, 57] Biss et al found that 53 of 26,765 dairy calves aged 4 to 14 days were condemned at slaughter for lesions of enzootic pneumonia.[8] Of the 370 carcasses reported condemned, enzootic pneumonia was second only to navel ill in reason for condemnation.[8] These slaughter condemnation data further reinforces the concern that DCP may start much earlier in life than previously recognized. Virtala et al[56] found that veterinary-diagnosed DCP occurred at a younger age than did caretaker-diagnosed DCP. Studies relying solely on owner diagnosis of DCP[57] have usually found later ages of disease onset than those using veterinary diagnosis.[56]

Pneumonia of dairy calves occurs both as endemic diseases and as outbreaks of respiratory disease.[4, 44] Chronic endemic disease is the most common manifestation of this disease, and as a result, pneumonia of dairy calves is often called *enzootic calf pneumonia.* This distinction between enzootic and epizootic DCP may be especially important in reference to cause, as different causes are more important in each form of the disease.[4, 44] DCP is manifest as calves with fever, nasal discharge,

From the Department of Clinical and Population Sciences, College of Veterinary Medicine, University of Minnesota, St. Paul, Minnesota

lacrimination, cough, increased respiratory rate, depression, rough hair coats, poor weight gains, and partial anorexia. Auscultation will reveal abnormal lung sounds, including increased bronchial sounds, crackles, and wheezes, with evidence of consolidation heard in advanced cases. Cranial tracheal compression may induce a cough response. Advanced cases of DCP may exhibit dyspnea and emaciation. Virtala et al[56] found that the earliest sign of DCP was often fever, and that signs referable to respiratory system followed this initial fever. Early symptoms of DCP remain subclinical to many caretakers, raising concern that more established refractory disease will be present when it is recognized by the caretaker. Acute outbreaks of DCP often produce more recognizable disease for the caretaker, as multiple animals are depressed and febrile, with some calves having symptoms referable to the respiratory system.

The postmortem findings in calves dying of endemic DCP or enzootic calf pneumonia often suggest a chronic progressive lesion.[44] Similar lesions are often found in calves that are condemned at slaughter.[8, 40, 55] Lesions commonly found include significant pulmonic involvement and pleuritis. Pulmonic lesions are often purulent or exudative in nature, and are primarily a bronchopneumonia with cranioventral distribution.[44, 55] Necrosis or abscessation may be present in some cases, as can bronchiectasis. Calves dying acutely during an outbreak may have lesions more consistent with atypical interstitial pneumonia, such as seen with bovine respiratory syncytial virus, as this agent is most commonly associated with outbreaks of DCP.[4]

CAUSATIVE FACTORS

Dairy calf pneumonia, like many other multifactorial diseases, can rarely be blamed on a single causal agent or management practice. As with other multifactorial diseases, a triad of causal agents, calf factors, and environmental factors is commonly suggested.

Etiologic Agents

A wide range of infectious agents has been implicated in DCP. Viral agents include bovine respiratory syncytial virus (BRSV),[4, 44] parainfluenza-3 virus (PI-3),[10, 54] bovine adenovirus,[13] bovine corona virus,[47] and bovine rhinovirus.[38] Other more common viruses, such as bovine viral diarrhea virus (BVDV) and bovine herpesvirus-1 (BHV-1), are rarely involved in DCP unless a farm problem involving these viruses in the adult cattle is occurring.[4, 44] BRSV has been identified as the most common cause of outbreaks of DCP in a number of studies.[4, 44] Other nonviral agents may become involved secondary to initial viral involvement.

The role of chlamydial agents, such as *Chlamydia psitacci*, in DCP remains a potential, but rarely documented, possibility.[37] Recently, my-

coplasmal agents, such as *Mycoplasma bovis* and *Mycoplasma dispar*, have been recognized as important primary pathogens resulting in DCP.[1, 10, 56] *M. bovis* especially has been documented in calves with pneumonia and tenosinovitis in North America whereas other *Mycoplasma* spp have been found more commonly in Europe.[49]

With all nonbacterial agents, questions arise as to the causal relationship of isolating or detecting an agent in the lung of a calf with respiratory diseases. Prospective studies that examine seroconversion to various respiratory pathogens in calves experiencing respiratory disease may provide some useful information. Three studies have followed calves over the first 3 to 4 months of life, monitoring the serologic response to the respiratory viruses (BRSV, PI-3, BVDV, BHV-1).[44, 54, 56] Seroconversion to BHV-1, BVDV, PI-3, or BRSV was a rare occurrence for any of the viruses in healthy and pneumonic calves, and was not found to be significantly correlated with cases of respiratory disease.[44, 56] These studies would suggest that respiratory viruses are not commonly associated with enzootic calf pneumonia or endemic DCP. Seroconversion to BRSV was documented on farms where outbreaks of DCP occurred.[44] This is consistent with previous work that demonstrated that BRSV was the causal agent most commonly associated with the outbreaks of DCP.[4]

Prospective studies have examined the serologic response to *M. bovis* and *M. dispar* in dairy calves over the first 3 to 4 months of life.[54, 56] One study found calves with higher passive antibody titers to *M. dispar* in the first month of life, had fewer cases of pneumonia, and better growth rates than did calves with low antibody titers to *M. dispar*.[54] This would imply that high antibody titers to *M. dispar* protects calves from DCP and subsequent poor growth. Virtala et al[56] found that seroconversion to *M. dispar* was a common event in dairy calves that occurred in both clinical cases and healthy calves. The role of mycoplasmal agents as initiators of DCP needs further documentation, but research to date suggests these agents may play an important initiating role.

Bacteria most commonly associated with DCP include *Pasteurella multocida, Pasteurella haemolytica*, and *Haemophilus somnus*.[4, 10, 44] Bacterial agents, such as *Actinomyces pyogenes*, may be found in pneumonic calf lungs. This is not considered a primary component of the respiratory disease complex, but rather a secondary invader of necrotic or diseased lung. *Salmonella* sp or *Escherichia coli* may also be found in pneumonic lung, but again these are bacteria which gain hematogenous access to the lung as a complication of sepsis, and are not considered part of the respiratory disease complex. *P. multocida* is commonly cited as the bacteria most often isolated from DCP.[4, 10] This may to some extent reflect the opportunistic nature of this organism to overgrow lung previously damaged by other bacteria, such as *P. haemolytica*.[2] A synergistic relationship between *Mycoplasma* spp and *P. haemolytica* has been suggested.[10, 23] Some authors believe that a synergistic relationship may also apply to *P. multocida* and *Mycoplasma* spp as well.[56] Further evidence of this synergism is suggested by the finding that it is more common to find

mycoplasma in conjunction with other bacteria than alone.[56] Synergism, such as that between *Mycoplasma* spp and *Pasteurella* spp, would not be unexpected with a multifactorial disease, such as DCP.

Prospective studies looking at serologic response to bacterial pathogens and its association with DCP have found calves with higher titers to *P. haemolytica* in the first month of life had less chance of respiratory disease and better growth rates.[54] Higher colostral titers against *P. haemolytica* would appear to be protective for DCP and resultant poor growth, which further confirms the importance of this bacteria as a causal agent in DCP. *H. sommus* is infrequently found in DCP,[4, 43, 54] but rarely in some farms it will be the only bacteria found.

Calf Factors

The respiratory defenses of the calf lung include aerodynamic filtration, particle removal, adhesion resistance, secretory defenses, and cellular defenses. The physical respiratory defenses (filtration, removal, adhesion resistance) can be compromised by inhaled noxious gases, temperature extremes, dehydration and viral infections causing impairment through damage to the mucosal lining of the upper respiratory tract, or by increased viscosity of respiratory secretions.[7, 10, 27] Noxious gases, such as ammonia, methane, hydrogen sulfide, or carbon dioxide, which become increased from inadequate manure handling or poor ventilation, can also impair secretory defenses via damage to the mucosal lining as well as impair cellular defenses by direct effect on alveolar macrophages.[30] Viral infection may also damage the mucosal lining and impair production of secretory defenses, such as lysozymes, lactoferin, complement, or secretory immunoglobin. Viral infections with agents such as BRSV, PI-3, and BHV-1 can also have a direct effect on cellular defenses, including alveolar macrophages, and for some viruses, the neutrophils.[30] Some viral agents, such as BVDV, may even impair pulmonary intravascular macrophages or lung lymphocytes in addition to alveolar macrophages and neutrophils.[35] Stress due to overcrowding, temperature extremes, commingling, surgical procedures, or vaccination may impair cellular defenses, immunoglobin production, and enhance bacterial adherence.[30, 61]

The specific immune response can be enhanced by passive transfer of colostrally-derived antibodies or by direct vaccination of the calf. Numerous studies have examined the importance of failure of passive transfer in the DCP morbidity or mortality.[36, 44, 54, 56] In one study, postcolostral immunoglobin levels were found to be negatively correlated with cases of pneumonia and positively correlated with growth rate.[54] Davidson et al[19] found that calves with low immunoglobin levels were treated for pneumonia earlier and longer than calves with high immunoglobin levels, and that in some groups, low immunoglobin levels were associated with increased respiratory morbidity. Other studies have failed to show an association between immunoglobin levels or

failure of passive transfer and the occurrence of respiratory disease or calf mortality.[36] One study concluded that unless large numbers of calves are examined it may be difficult to detect a significant difference in calf morbidity or mortality in calves with failure of passive transfer.[44] Rea et al[36] concluded that calves with lower passive transfer values had increased risk of death, but that failure of passive transfer is not an infallible predictor of mortality. The morbidity and mortality attributable to DCP accounts for less than half of the total calf morbidity and mortality typically reported. As a result, the effect of the immunoglobin levels on DCP-related morbidity and mortality will be even more difficult to document than total calf morbidity and mortality. An additional explanation for the difficulty in documenting a correlation between immunoglobin levels and DCP is that a significant amount of DCP occurs in calves over 2 months of age, and immunoglobins to many pathogens of DCP have waned by 2 months of age.[14]

Regular herd vaccination, especially in dry cows, should increase the levels of specific antibody in calves receiving adequate amounts of colostrum. Vaccination of calves with vaccines after colostral immunity has declined or with intranasal vaccines in the face of passive immunity may produce a protective immune response in calves that prevents or limits the severity of DCP for certain infectious agents.

Environmental Factors

The calf's immediate environment impacts the calf in a number of ways. Ambient temperature is an important factor affecting dairy calf health.[51] Cold weather is especially important for young calves, which have little body insulation. Increased humidity or precipitation in the calf's environment worsens the calf's ability to maintain thermal neutrality. Warm weather can also be undesirable, as young calves are capable of greater perspiration per pound of body weight than adults, and warm weather may predispose young calves to dehydration.[51]

The bacterial content of air in cattle barns can be as high as 10^6 organisms/m^3.[20] Disease incidence can be affected by length of pathogen survival time as an aerosol and the concentration of the pathogen in the air space. Humidity is an important limiting factor affecting pathogen survival. The optimum zone for limiting survival time of bovine pathogens is 55% to 75% relative humidity.[7] Adequate fresh air flow into the calf's environment is important in limiting humidity and reducing the concentration of noxious gases and pathogens.[6] The flow of air should be from younger, more susceptible cattle to older, less susceptible cattle to limit moving pathogens from older cattle to younger cattle. Adequate fresh air flow and proper directional movement of air are important goals of ventilation.

Calf housing with overcrowding of calves or excessive stocking densities results in increased transmission of pathogens, especially if there is mixing of age groups. Overcrowding also puts additional stress

on the building ventilation through build-up of noxious gases and pathogens. Bates et al[6] has recommended standards for ventilation, including building location, fan capacity and location, intake location and design, temperature regulation, air space needed and airflow directions, and acceptable humidity levels. Individual calf hutches that are properly located provide the calf with adequate fresh air free of pathogens and noxious gases and overcome many of the problems found with the calf barns.[3] Calves moved out of hutches can then be put into small groups separated from older cattle using super hutches.[3] Like the calf hutch, the super hutch also serves to limit pathogen transmission and build-up of noxious gases in this susceptible group of calves.

Sivula et al[44] found that 80% of calf barns provided housing that failed to meet adequate standards of ventilation and housing[3, 6] regardless of whether they were housed individually or in groups.[44] In addition, calf housing where calves share the same air space as adults never met the adequate standards of ventilation and housing.[44] A much higher percentage of calf housing that used calf hutches met these adequate standards of ventilation and housing, and virtually 100% would have had adequate housing if the hutches had been positioned correctly.[44] Calves raised in inadequate housing have significantly poorer growth rate than do calves raised in housing that is considered adequate.[44] This emphasizes the importance of adequate housing, as calf barns, especially those using mechanical ventilation, rarely meet adequate standards of housing and ventilation.[6, 44] Studies examining the use of calf hutches have found that about 20% to 40% of all dairy producers raise their calves in hutches.[16, 44, 53, 60] The percentage of producers that use calf hutches continues to increase, as the benefits of their use are documented and published.

DESCRIPTIVE EPIDEMIOLOGY AND RISK FACTORS FOR RESPIRATORY DISEASE

Morbidity and Mortality

Waltner-Toews et al[60] determined from producer diagnosis that 15% of Ontario Holstein dairy calves were treated for pneumonia before weaning. Curtis et al[16] reported that Holstein calves in New York had a crude incidence risk of 7.4% for respiratory tract illness, as diagnosed by the farmer.[16] Sivula et al[44] found 7.6% of 845 Minnesota dairy calves were diagnosed by producers as having pneumonia.[44] Van Donkersgoed et al[54] found the risk of pneumonia in Saskatchewan dairy calves was 39%, as diagnosed by the farmer, and 29% when the pneumonia was veterinarian diagnosed. Virtala et al[56] found the risk of pneumonia was 11% in New York dairy calves when diagnosed by producers and 25.6% when diagnosed by a veterinarian.

The morbidity associated with DCP clearly varies greatly depending on the number of calf samples, selection biases in selecting study popula-

tions, and geographic bias, which may influence management practices. It is generally accepted that producers underdiagnose cases of DCP, and the findings of Virtala et al would confirm this. Van Donkersgoed et al, however, found that randomly selected producers actually diagnosed more cases of DCP than did the study veterinarians. In this study it was suggested that veterinary visits (bimonthly) may have missed cases diagnosed with daily observations.[54] Virtala et al conducted weekly visits, and veterinarians were able to diagnose more cases of DCP than producers selected by convenience sampling.[56] Sivula et al[44] conducted monthly visits to randomly selected farms, but was unable to detect calves with DCP undiagnosed by the producer.

Mortality rates reported for DCP varies from 1.8%[44, 54] to 4.2%.[56] Other studies looking at epidemiology of dairy calf diseases did not determine cause of death, and as a result, were not able to report mortality rates or case fatality rates.[16, 40] Case fatality rates reported for calves with DCP range from 2.2%[56] to 9.4%,[44] and will vary with the sensitivity of the initial detection method (veterinarian versus producer).[56]

Pneumonia accounts for a significant proportion of the mortality (proportionate mortality) in dairy calves raised on dairy farms. Pneumonia accounted for 24% of deaths in New York calves[56] and 30% in Minnesota calves.[44] In one study examining Ontario veal calves raised in veal barns, pneumonia accounted for 52% of mortality in 4863 calves on 6 farms.[39] Producer accuracy in diagnosing causes of mortality was examined by Sivula et al.[44] Producers were found to be moderately accurate, but often listed the cause of death as unknown.[44] If these unknown cases were removed from the analysis, producer accuracy improved greatly.[44] Said more simply, when producers recorded a cause of death, they were usually correct; however, in many cases the producer did not know why the calf died.[44] This emphasizes the importance of laboratory confirmation of mortality over producer diagnosis in any epidemiologic study of dairy calf diseases.

Risk Factors for Respiratory Diseases

A number of studies have examined herd level risk factors on overall calf mortality. Herd size has been shown to significantly increase calf mortality in dairy calves.[22, 32, 58] Lower mortality rates have been reported on farms where the producer or family member took care of the calves when compared to hired help.[22, 34] Seasonal effects, such as hot and dry conditions in summer and cold, wet, and windy winter weather, have been associated with increased calf mortality[29, 32, 46]; greater mortality was reported in the winter in northern states[46] and in the summer in southern states.[32]

Calf housing and its effect on mortality have also been examined. Oxender et al[34] found lower mortality rates when calves are raised away from the cows, but other studies have not found this to be true.[46] The

type of housing is also important when calves are housed separately from adults. Waltner-Toews et al[58] found lower mortality rates in calves housed in hutches as opposed to those housed in group pens, which agrees with a number of other studies suggesting outdoor hutches are superior to all other methods of calf raising.[28, 44]

Calf level risk factors that affect mortality include dystocia, assisted first-colostrum feeding, and birth outside the maternity pen.[58] Calf level risk factors that have been reported to increase the risk of DCP morbidity are birth from a first-calf heifer,[17] concurrent disease,[17, 59] and pail feeding of colostrum.[59] Housing the calves in hutches and prophylactic antibiotics at birth had a protective effect on calf morbidity associated with DCP.[59] Other calf level risk factors for DCP that were discussed earlier in this article were reported by Virtala et al[56] and Van Donkersgoed et al.[54] Those risk factors having a negative association included low serum immunoglobin,[54] low first-month titers to *P. haemolytica*,[54] cultures of *P. multocida* and *M. dispar* from a tracheal wash,[3] and rising titers to *M. dispar*.[56]

Large-scale epidemiologic investigation poses a significant challenge for researchers. Selecting cohorts of calves to study that are representative of the population as a whole, including large enough sample size in the study and collecting data in a manner that gives accurate representation, are but a few of the challenges. These types of studies are major undertakings that rarely provide conclusive answers to the large number of questions being asked. Over time, however, as more and more of these epidemiologic investigations are conducted with improved methodology, trends emerge which document what common sense and general practice have suggested to be important factors.

COSTS ASSOCIATED WITH DAIRY CALF PNEUMONIA

Economic Estimates of the Cost of Dairy Calf Pneumonia

Pneumonia in dairy calves can cause economic loss in a number of ways. In addition to obvious causes of economic loss, such as death loss and treatment costs, the long-term effects of poor growth rate and reduced lifetime milk production may be of even greater importance. Respiratory disease in Ohio dairy cattle has been reported to cost $10.53 per cow year.[33] Michigan producers estimated that respiratory disease in adult cattle had a cost of $3.95 per cow year, but DCP cost $14.71 per calf year.[25] In California DCP was reported to account for $0.75 per calf month.[43]

Dairy Calf Pneumonia and Its Effect on Growth Rate

Concern over the effect of subclinical, clinical, and chronic pneumonia on growth rate is important not only in dairy calves destined to

become replacement animals, but also for dairy calves raised for veal or beef. Other industries, such as the beef feed lot industry and pork industry, have recognized this potential. Disease monitoring in the swine industry has been done through slaughter checks since the 1960s. Evaluation of lungs at slaughter has been used as a diagnostic screen for enzootic swine pneumonia, with the finding that every 10% of pig's lungs affected with pathology equates to a depression in growth of 37.4 g.[24] Beef calves with lung lesions at slaughter have decreased mean daily gain (0.076 kg/day) compared to those without lesions.[62] Cross-bred beef and dairy calves also had reductions in mean daily gain as well as carcass weights if lung lesions were present at slaughter.[50] Similarly, veal calves with lung lesions at slaughter had carcass weights of 4.3 kg less than carcasses of calves that lacked lesions.[55]

Beef calves that are treated for respiratory disease have been shown to be associated with increased amount of pulmonary lesions when compared to untreated cohorts.[62] Feedlot calves that received treatment for respiratory disease did not perform as well as cohorts not receiving treatment when average daily gain was examined for both groups.[5] Taken together, these studies suggest that when respiratory disease is present, it will result in decreased growth rate of calves experiencing respiratory disease regardless of whether these calves have been treated for respiratory disease or not. Calves with poor growth rate can be expected to have a delayed age at first calving.

Age of First Calving

The average age at first calving for dairy heifers is approximately 28 months (ranging from 22 to greater than 30).[26] Total lifetime milk yield and gross income is maximized when age at first calving is 22 to 24 months.[12] A negative correlation has been demonstrated between age at first calving and productive lifespan.[42] Early calving heifers have a greater chance of survival in the herd after 3 years than heifers calving later than 24 months of age.[48] Calving interval and percentage of cows culled for breeding problems have also been shown to increase with either age or weight at first calving.[41] Finally, risk of dystocia and metritis are also minimized with an early age of first calving.[52]

Chase and Otterby[11] have determined the cost of rearing replacement heifers to range from $750 to $1300, and that a loss of $1 to $3 per day can occur for each day beyond the goal of 24 months of age at calving; these estimates include feed costs, overhead costs (loan interest, veterinary-related charges, facilities, utilities, labor), a one-time return from transition down to a 24-month age at first calving, and other costs (genetic material, decreased interest cost, opportunity costs).

Effect of Dairy Calf Pneumonia on Productive Lifespan

Concern exists that subclinical and clinical DCP may result in chronic pneumonia that can be exacerbated into clinical disease or serve

as foci of chronic disease. Chronic pneumonia may depress growth rate so that heifers never reach the lactating herd or it may impair milk production and reproductive performance in cows once they reach the lactating herd. Waltner-Toew et al reported that calves which were treated for pneumonia in the first 3 months of life were 2.45 times more likely to die before time of calving than those heifers which had not been treated for pneumonia.[58] Correa et al[15] reported that heifers not experiencing DCP were twice as likely to calve, and calved for the first time 6 months earlier than heifers that experienced respiratory disease as calves.[15] Herd cows that were treated for pneumonia either as adults or as calves have been shown to be at an increased risk of culling when compared to herd mates that had not been treated for pneumonia.[12] Curtis et al[15] confirmed this finding in 1989, when they reported that calves which had experienced DCP were at increased risk of culling once in the milking herd when compared to herd mates of the same age that had not experienced DCP.

ECONOMIC CONSIDERATIONS FOR CONTROL OF DCP

Replacement heifers represent a significant economic investment for the dairy producer. Goodger et al[21] has stated that the investment in replacement heifers is the second largest financial input into the dairy enterprise,[21] with feed costs representing the largest dollar input. As discussed previously, the cost of DCP can be considerable. Control programs combined with housing and management practices aimed at elimination of DCP could have a profound effect on the costs associated with heifer replacements. The most profound effect would be through a reduced age at first calving, as this would decrease the time period of expenditures made on the heifer prior to reaching the lactating herd. Death loss and the cost of treatment for clinical cases would be virtually eliminated, and heifers would enter the lactating herd free of chronic pneumonia, which limits lifetime productivity.

The decision that modern dairy farmers must now make is if they are capable and willing to raise the heifers in housing that will maximize their genetic potential. Dairy farmers that use facilities with inadequate housing and ventilation and fail to use herd vaccination will be faced with the reality that they must adopt the changes needed to be competitive or consider having their heifers raised by a contract heifer raiser. Contract heifer raisers typically take the calves from dairy farms from birth to weaning and raise the heifers until they are returned as bred heifers 1 month before calving. Contract heifer raising can provide a number of advantages, including increased time and labor for the dairy farmer to concentrate on the lactating herd and proper forage harvesting and storage. Space may be freed up for uses, such as dry cow facilities, maternity facilities, or adding cows to the lactating herd. The competitiveness of most contracts for heifer raising necessitates that optimum

housing and management practices be in place so that financial rewards can be realized. One concern of contract heifer raising arrangements is that the returning heifers may bring infectious agents, such as Johne's disease or bovine leukemia virus, back to the dairy farm. Hopefully, contract raisers will use control measures to prevent such disease and demonstrate to the dairy farmer that there is a real advantage to their services. In addition, for some farms, raising the heifers at a separate facility may significantly decrease the incidence of currently experienced conditions, like Johne's disease, because of age segregation. A final option for the producer not wanting to raise his own replacements would be to purchase heifers from other dairies or sales barns. This would increase the risk of purchasing cattle with disease and would not guarantee heifers that had been raised under ideal housing and management unless the seller was known to the buyer. Purchasing heifers would also not lend itself to genetic improvement of the herd.

In summary, DCP is a preventable disease that proper housing and management can virtually eliminate. The economic pressures of today's dairy industry necessitate that replacement heifers and bull calves be raised under conditions which limit this important disease.

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Address reprint requests to

Trevor R. Ames, DVM, MS
Department of Clinical and Population Sciences
College of Veterinary Medicine
University of Minnesota
1365 Gortner Avenue
St. Paul, MN 55108