Enterobacter spp.) (22.4%), *Pseudomonas* spp. (5.6%), *Serratia* spp., *Salmonella* spp., *Proteus* spp. and Yeast (2.8%), contaminated (11.2%), *Bacillus* (1.2%), mixed major pathogens (2 pathogens isolated from the same sample) (4.8%), and no growth (19.8%).

Mastitic quarter NAGase (NAGM) differed significantly among agents (P<.01, one-way ANOVA). NAGM was lowest for mixed major pathogens (5.6 +/- 1.2 uM) and highest for *Strep.* non-ag's (13.9 +/- 1.9 uM). NAGM mean for all 508 samples was 9.0 +/- 0.5 uM. The difference between NAGM and reference NAGase was 7.9 +/- 0.4 uM. Reference NAGase was not significantly different among different etiologic agents (one-way ANOVA).

Mean change in daily milk production following mastitis was -2.2 +/- 0.2 kg for 344 cases. Production loss varied among etiologic agents (P < .05, one-way ANOVA). Mixed major pathogens had the greatest loss of any agent group (-4.9 +/- 1.7 kg), while least milk loss was associated with *Serratia, Salmonella, Proteus,* and Yeast (-0.5 +/- 0.9 kg).

NAGase was statistically associated with the severity of mastitis. Increased milk NAGase was significantly (P<.001, one-way ANOVA) associated with: 1) increased duration of treatment, 2) increased duration of clinical signs, 3) decreased daily milk production, and 4) increased risk of being culled because of mastitis. NAGase was a superior predictor of severity of clinical mastitis to either WMT or microbiological culture. NAGase was combined with stage of lactation, parity, baseline milk production before mastitis onset, and season of onset in general linear models to predict the outcome of clinical cases as measured by the first 3 aforementioned variables. The statistical models explained vary little of the variability among cases in duration of therapy (R^2=.11), duration of clinical signs (R^2=.11), and milk production change (R^2=.09).

Discussion

NAGase was a better predictor of several measures of clinical severity of mastitis than other information currently available at onset, such as age, stage of lactation, season of onset, level of milk production, milk culture, and WMT. However, this association was too weak for NAGase to be of great value as a prognostic test for clinical mastitis. NAGase alone or combined with other variables could not consistently predict the sequelae of clinical cases. Most of the variability in outcome among clinical mastitis cases remained unexplained.

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Clinical Lameness in Dairy Cows in the Midwestern United States: A Preliminary Report

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Introduction

The economic cost of lameness in dairy cattle has recently been reported as $7.69 per cow at risk of lameness (per cow in the herd) per year.[1] This work, from herds with a 5% reported annual incidence of lameness, listed losses from culling, treatment and prevention costs, and death, but failed to include milk loss other than that from withheld milk due to treatment. An earlier report in herds with 25% annual lameness incidence, estimated the annual cost of lameness per cow at risk to be 11.75 £ (about $21).[2] In this study, a 2.4% reduction of the total lactation yield of about 12,000 lbs. of milk was estimated, in addition to culling and treatment costs. This value is therefore likely to be quite conservative in higher producing herds.

Reproductive losses, although not included in either

of the previous economic evaluations, increase the cost of lameness substantially.[3-5] An increase of 14 to 30 days in the interval from calving to conception has been shown in lame cows when compared to nonlame cows. These economic factors make bovine lameness a very important disease syndrome in the dairy industry.

Goals of this study were to determine the prevalence of clinical lameness in dairy cows in the midwestern United States and to determine cow level risk factors for clinical lameness.

Materials and Methods

The seventeen Minnesota and Wisconsin dairy herds selected into this study were visited twice, in the summer of 1989 and in the late winter of 1990. The mean number of milking cows at each visit was about 50.

The clinical lameness scoring system utilized for identification of clinical lameness consisted of individual observations of each milking cow at a walk by two observers at each farm. A lame cow was defined as one classified lame by at least one observer. The prevalence of clinical lameness was defined to be the number of lame cows divided by the total number of milking cows on these farms.

To determine cow level risk factors for clinical lameness, a case-control study design was used. Cases (cows with clinical lameness) were previously identified from the prevalence study. A control nonlame cow was selected from the same herd as each case matching on parity and stage of lactation.

Factors evaluated in each case and control animal for their association with clinical lameness included the following: bodyweight, body condition score, dorsal claw angles, and presence and character of limb lesions. Sole lesions were not evaluated.

Results

Using our scoring system, the prevalence of clinical lamness in these herds was 13.6% of milking cows in summer and 16.7% in late winter.

From univariate analysis of late winter data, higher bodyweight was positively associated with clinical lameness, while higher body condition score appeared to have a strong negative association with lameness. Lesions positively associated with clinical lameness included abnormal hoof overgrowth and rear limb superficial swelling in locations other than the tarsus.

The multivariate statistical technique of conditional logistic regression will be utilized to yield a model from which estimates of relative risk can be determined, after controlling for confounding and evaluating interactions among these variables.

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Mastitis in the Beef Cow and Its Effects on Calf Weight Gain

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Quarter samples for culture were collected from 120 beef cows in early, mid, and late lactation to assess prevalence of mastitis pathogens and effects of mastitis on calf weight gain. In early, mid and late lactation respectively, 25.8, 29.2, and 54.4% of cows and 13.1, 14.9, and 27.5% of quarters were infected.

Staphylococcus aureus was found in 2.9, 2.7, and 3.2% of quarters in early, mid and late lactation respectively. Staphylococcus hyicus was found in 2.5, 1.7, and 2.5%, and Corynebacterium bovis the most prevalent organism, in 4.0, 7.6, and 18.2% of quarters at the respective sampling times. Geometric mean somatic cell counts (cells/ml X 10^3) by quarter infection status were: *S. aureus,* 792: *S. hyicus,* 477; *C. bovis,* 102; and uninfected, 18. Adjusting 205 day weight gain of 224.1 kg for calves with *S. aureus*-infected dams was lower ($p < 0.5$) than the 233.7 kg for calves with uninfected dams. Effects of other infections on calf weight gain were not significant. California Mastitis Test did not effectively select infected quarters for dry cow therapy.

In order to evaluate the effect of treatment at fall weaning, cows were randomly divided between control and dry treatment, and final milk cultures collected at 2 to 4 weeks post calving the following spring. Prevalence of infection with any organism was reduced primarily due to elimination of infections present at weaning.

Currently a study is in progress to determine economic advisability of dry treating all cows at weaning on the basis of improved calf growth during the subsequent lactation. Concerns which remain to be addressed include; injectable products available for dry treatment, age groups requiring preferential treatment, frequency of treatment, and effect of treatment on cow longevity.