# Reported Use of Non-Steroidal Anti-Inflammatory Drugs in Food Animal Practice

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A cross-sectional study was designed to determine 1) the extent to which nonsteroidal anti-inflammatory drugs (NSAIDs) are used in the treatment of food animals, 2) demographic and practice factors that may influence their use, and 3) if withdrawal times for milk and slaughter are recommended. A survey questionaire was mailed to a stratified random sample of 2000 veterinarians whose practices were at least 50% food animal. A multiple regression model was used for data analysis. Seventy percent of practitoners contacted responded. Of those, approximately 95% reported using NSAIDs, with 58, and 24 and 18% of respondents reporting use more than once a week, once a week, and 1 to 2 times per month, respectively. Use of flunixin meglumine was reported more frequently than the use of other NSAIDs such as aspirin, phenylbutazone and dipyrone. Eighty percent of respondents that used NSAIDs, did so in combination with antibiotic therapy. Withdrawal times for milk and meat were based on guidelines for the antibiotic. When using NSAIDs as a sole therapy, recommendations for withdrawal times for milk and meat were wide ranging.

# N-Acetyl-B-D-Glucosaminidase as a Predictor of Milk Loss and Recovery Following Clinical Mastitis

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### Introduction

N-acetyl-B-D-glucosaminidase (NAGase) is a lysosomal enzyme found in milk and other body fluids. Milk NAGase level has been reported to be increased in clinical and nonclinical mastitis, primarily due to leakage from damaged secretory epithelial cells (3,4,6,10,12). The value of NAGase as a prognostic indicator of severity of clinical mastitis has been suggested (6,10), but never evaluated.

Clinical mastitis is becoming more important as management improvements have resulted in greater control of nonclinical pathogens (2,11). Economic losses due to clinical mastitis include decreased milk production, discarded milk, loss of animals due to culling or death, therapeutic costs, veterinary fees, and labor (1,5). Clinical mastitis cases vary greatly in lost milk production, duration of treatment, duration of clinical signs, and risk of culling from the herd. At the onset of signs, it is difficult to predict which cases will be most severe. Accurate early prognosis for mastitis has economic value. This study was conducted to assess the ability of a single milk NAGase level (obtained at clinical onset) to predict severity of mastitis.

### Materials and Methods

Milk samples were collected at clinical onset from 508 episodes of mastitis on a 1700 cow Michigan dairy farm. Milking personnel detected mastitis by presence of a hot, hard, or swollen quarter and/or abnormal flakes, color or consistency of the milk. Milk from all four quarters of clinical cows was collected for NAGase and WMT determination (9,13). Milk from clinical quarters was aseptically collected for microbiological culture (7). Daily milk production, all disease events, and treatments were recorded for all cows in the herd using a computerized record keeping system.

NAGase concentration was determined using a commercially available test kit (8,9). NAGase activity was expressed as uMoles/min/L (uM). NAGase results are reported as mean +/- standard error of the mean in units of uM. NAGase was measured for each clinical quarter and for one reference quarter from the same cow (the quarter with the lowest WMT value).

### Results

Etiologic agents isolated included *Staphylococcus aureus* (13.8%), *Staphylococcus spp.* (8.6%), Diphtheroids (2.8%), non agalactiae *Streptococcus* (7.0%), coliforms (*Escherichia coli, Klebsiella* spp., *Citrobacter* spp., and

*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.

### References

1. Blosser, T.H. Economic losses from and the national research program on mastitis in the United States. J. Dairy Sci. 62:119-127, 1979. 2. Eberhardt, R.J., Natzke, R.P., Newbould, F.H.S., Nonnecke, B., and Thompson, P. Coliform mastitis- a review. J. Dairy Sci. 6:21-22, 1979. 3. Fox, L.K. and Schultz, L.H. Effect of infection status on quarter milk production and composition following omitted milking. J. Dairy Sci. 68:418-423, 1985. 4. Fox, L.K., Shook, G.E., and Schultz, L.H. Factors related to milk loss in quarters with low somatic cell counts. J. Dairy Sci. 68:2100-2107, 1987. 5. Jasper, D.E., McDonald, J.S., Mochrie, R.D., Philpot, W.N., Farnsworth, R.J., and Spencer, S.B. Bovine mastitis reasearch needs, funding, and sources of support. Proc. 21st National Mastitis Council, pp. 184-193, 1982. 6. Kitchen, B.J., Middleton, G. and Salmon, M. Bovine milk N-acetyl-beta-D-glucosaminidase and its significance in the detection of abnormal udder secretions. J. Dairy Res. 45:15-20, 1978. 7. Laboratory and field handbook on bovine mastitis. National Mastitis Council, 1987. 8. Linko-Lopponen, S. and Makinen, M. A microtiter plate assay for N-acetyl-beta-D-glucosaminidase using a flourogenic substrate. Analyt. Biochem. 148:50-53, 1985. 9. Milk NAGase Test Instruction Manual. Flow Laboratories Inc. McLean, Va., 1987. 10. Obara, Y. and Komatsu, M. Relationship between N-acetyl-beta-D-glucosaminidase activity and cell count, lactose, chloride, or lactoferrin in cow milk. J. Dairy Sci. 67:1043-1046, 1984. 11. Rothbauer, D.L., Buchner, E.C., Wells, S.J. Effect of vaccination with E. coli on the incidence of acute mastitis. Proc. 21st Annual conference American Association of Bovine Practitioners, 112-115, 1988. 12. Schaar, J. and Funke, H. Effect of subclinical mastitis on milk plasminogen and plasmin compared with that on sodium, antitrypsin and N-acetyl-beta-D-glucosaminidase. J. Dairy Res. 53:515-528, 1986. 13. Wisconsin Mastitis Test Kit. Z.D. Roundy, Orem, Utah.

# 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