USE OF CIRCULATING METABOLITES, MINERALS, AND HORMONES AS POTENTIAL MARKERS FOR SUBSEQUENT DEVELOPMENT OF MASTITIS

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TAKE HOME MESSAGES

- Our objective was to identify specific blood markers as risk factors for development of mastitis during early lactation.

- Cows that developed clinical mastitis during early lactation had higher circulating concentrations of non-esterified fatty acids (NEFA) and aspartate aminotransferase (ASAT; an indicator of liver tissue damage) in the weeks prior to development of mastitis when compared to healthy cows that did not develop mastitis at any time during early lactation.

- No markers were identified as potential risk factors for subclinical mastitis during early lactation.

- Based on the growing information available from this study and published literature, blood concentrations of NEFA may be the most promising marker for risk of CM during early lactation.

INTRODUCTION

The transition from late gestation through early lactation is the most metabolically challenging time period in the life cycle of a dairy cow. During this period, cows are at high risk for development of diseases such as ketosis and mastitis. Mastitis is the most costly of all diseases in the dairy industry, exceeding $2 billion dollars annually in the United States alone. Understanding the causes of mastitis will improve disease prevention and animal well-being, which may enable cows to reach their maximum genetic potential for milk yield and increase profitability of dairy farms. The severity of negative energy balance (NEB) during the transition period, which is characterized by increased concentrations of NEFA and beta-hydroxybutyric acid (BHBA) and decreased glucose in blood, may contribute to suppression of immune system function during the transition period. The objective of this experiment was to identify specific blood markers from several candidate energy metabolites, minerals, and hormones, as risk factors for the development of naturally occurring mastitis during early lactation, using a subset of cows from a larger experiment.
MATERIALS AND METHODS

Data for this study originated from a larger experiment that totaled 634 lactations within 317 cows ranging from parity 1 through 4 at the farm Ammibøl Skovgaard, Denmark. Data was collected over a 5-year period from January 1996 through October 2001. Weekly blood samples were collected via puncture of the jugular vein throughout each cow’s productive life. Samples were analyzed for hormones including insulin, growth hormone, and triiodothyronine, metabolites such as NEFA, BHBA, glucose, urea nitrogen, cholesterol, and albumin, minerals including calcium, magnesium, and phosphorous, and enzymes such as ASAT. Cows were milked twice daily. At each milking, milk yield was measured and fat, protein, lactose, and SCC (1000 cells/mL) were analyzed. Energy balance was calculated. Cows were classified as healthy (H), having subclinical mastitis (SM), or developing clinical mastitis (CM) during the first 90 days in milk (DIM). The actual time of mastitis was defined as the DIM where increases in SCC were first observed and was designated as time of mastitis = 0. Before statistical analysis, the dataset was adjusted for any variations in parameters other than udder health status. Therefore, the natural increases in NEFA and BHBA and decline in glucose normally observed in cows during the periparturient period were eliminated. The individual cow variation was used to evaluate the use of individual cow differences in metabolites, hormones, energy intake, energy balance, and minerals as risk factors for the development of mastitis in early lactation. Estimated between-cow differences for all parameters were analyzed 2 weeks before the time of mastitis using the MIXED procedure of SAS to identify potential markers for mastitis.

RESULTS AND CONCLUSIONS

For all parameters analyzed, SM did not differ from H or CM cows. Therefore, no clear markers were identified for the incidence of SM during early lactation. The CM cows had higher NEFA and a tendency towards higher BHBA than H cows 2 weeks before mastitis was diagnosed (Figure 1A). In addition, ASAT was higher 2 weeks before mastitis in CM than H cows during the first 90 DIM (Figure 1B). Between-cow differences in hormones (insulin, T3, and GH), minerals (calcium, phosphorous, and magnesium), energy intake, energy balance, and milk yield were not significantly different among CM, SM, and H cows 2 wk before time of mastitis. The growing information available on the relationship between metabolism and risk of mastitis indicates that NEFA have the most consistent and least conflicting positive association with development of mastitis; therefore, NEFA may be the most useful as a potential marker for risk of mastitis during early lactation. Our results indicate that ASAT may be a better indicator for risk of CM than for SM during early lactation; however, the low specificity of ASAT to any particular disease may make it a less useful marker for risk of mastitis during early lactation.
Figure 1. Estimated between-cow differences in circulating non-esterified fatty acid (NEFA; A) and aspartate aminotransferase (ASAT; B) concentrations between cows that developed clinical mastitis (CM), sub-clinical mastitis (SM), and did not develop mastitis (healthy; H) relative to time of mastitis (week = 0) during early lactation. *Concentrations differed significantly between CM and H cows.