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Short communication: Associations of udder edema with health, milk yield, and reproduction in dairy cows in early lactation

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ABSTRACT

Udder edema (UE) is a common condition of cows around calving, but its effects are not well characterized. The objectives of this study were to determine the associations of UE with the incidence of health disorders and with milk yield and reproduction in dairy cows in early lactation. On 3 commercial farms, UE was scored weekly on 1,346 cows, on a scale of 0 to 3, from 1 wk before calving to 3 wk after calving. Among cows with complete UE scores, 30% never had edema, 12% had edema only prepartum, 11% had it only postpartum, and 48% had edema prepartum and in at least 1 wk postpartum. Udder edema was associated with a greater incidence of clinical mastitis before 30 d in milk (5 vs. 2%). Subclinical ketosis (blood β-hydroxybutyrate ≥1.2 mmol/L) was more prevalent at wk 2 (11 vs. 6%) postpartum among cows with UE. No association was observed of UE with other diseases or culling in early lactation. In a subset of 912 cows with complete UE and 3 test-days of milk yield data, differences were observed in yield at test d 1 among UE categories. Cows with UE only prepartum produced less milk (39.9 kg/d) than cows with UE postpartum only (42.4 kg/d) and cows with UE both prepartum and postpartum (41.6 kg/d), none of which differed from cows without UE (40.9 kg/d). Udder edema was not associated with the prevalence of anovulation, or the time to or probability of pregnancy at first insemination, yet to 300 d in milk, cows that had UE postpartum had a shorter time from calving to pregnancy than cows without UE. The associations of UE with health and productivity are mixed, and the mechanisms underlying UE and its effects merit further investigation.
Key words: udder edema, health, transition

Short Communication

Udder edema (**UE**) is a common yet little-investigated metabolic disorder (Melendez et al., 2006; Kojouri et al., 2015). Udder edema is the accumulation of lymphatic fluid in the interstitial space of the mammary gland and surrounding tissues (Tucker et al., 1992; Kojouri et al., 2015). The causes of UE are not clear, but it can occur when circulating lipid and lipoprotein concentrations decrease due to impairment in liver function with low DMI (Kojouri et al., 2015). Kojouri et al. (2015) found that serum concentrations of total proteins, triglycerides, cholesterol, and lipoproteins were lower in cows with UE. Other problems related to UE include difficulty with milking machine attachment, risk of teat and udder injuries, mastitis, and reduction in milk production (Melendez et al., 2006; Bacic et al., 2007).

Cows, particularly entering the first lactation, generally have some UE in late pregnancy and at parturition, but extensive edema has been shown to affect milk production and health of the udder (Malven et al., 1983). Cows with longer gestation length showed increased severity of edema (Malven et al., 1983). Malven et al. (1983) also reported an association of higher plasma concentrations of estradiol-17α and estrone with increased severity of edema. Melendez et al. (2006) reported that milk yield at the first DHIA test day was 3.6 kg lower in cows with UE. Van Dorp et al. (1998) identified a positive genetic correlation between milk yield and UE.

The objective of this study was to measure the association of UE with the incidence of health disorders in the transition period, milk yield in early lactation, and reproductive performance. We hypothesized that UE would be associated with greater incidence of health disorders and lesser milk yield in early lactation.

The data for this observational study were collected over 1 yr on 3 commercial freestall dairy farms in Ontario with 195 to 450 milking cows, concurrent with a randomized controlled trial of a dietary supplement

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of B vitamins, reported elsewhere (Morrison, 2017). All farms used automated activity monitors for estrus detection as the primary means of reproductive management, supplemented by synchronization for timed AI. The treatment in the controlled trial had no effect on the prevalence, severity, or duration of UE, and did not modify the associations of UE with the outcomes reported here. The exposure of interest was the presence of UE (defined below as a score $\geq$2). Herds were purposively selected based on milking more than 150 Holstein cows, being enrolled in DHIA milk recording, and maintaining accurate disease records. Each farm was visited weekly from November 2015 to December 2016. The University of Guelph Animal Care Committee reviewed and approved the study protocols that were accepted and followed by the herds enrolled.

Udder edema scores were assigned weekly at 1 wk before expected calving and in each of the first 3 wk after calving. The cows were assigned a score of 0 to 3 using a scheme developed for this study (Figure 1). Scores were assigned based on visual assessment and palpation of the udder. The scoring system was developed from those used by Dentine and McDaniel (1983), Nestor et al. (1988), and Tucker et al. (1992). Those ranged from 5- to 10-point scoring systems, which makes discrimination between scores difficult to assess properly. The scoring system developed for this study was a 4-point scale from 0 for no edema to 3 for severe edema. Interrater agreement was assessed with 3 independent scorers at the time of the scoring development. Cohen's κ coefficient was calculated and substantial agreement of $\kappa = 0.76$ was found between raters 1 and 2, and raters 2 and 3, with almost perfect agreement of $\kappa = 0.88$ between raters 1 and 3.

Blood samples were collected from the coccygeal vessels into evacuated tubes without anticoagulant (Vacutainer, Becton Dickinson and Company, Franklin Lakes, NJ) between 4 and 10 d before the expected calving date and between 1 and 7 DIM to measure serum nonesterified fatty acids (**NEFA**). Serum NEFA was measured with a Cobas 6000 c501 (Roche, Basel, Switzerland) biochemistry analyzer using the Randox NEFA kit at the Animal Health Laboratory, University of Guelph. Blood samples were taken once each week for the first 3 wk postpartum to measure blood BHB with a validated point-of-care meter (Precision Xtra, Abbott Laboratories, Mississauga, Ontario, Canada). Blood samples to measure serum progesterone were collected at 6 and 8 wk postpartum from the coccygeal vessels. Progesterone was measured by the investigators using a validated (Broes and LeBlanc, 2014) ELISA kit (Ovucheck Plasma, Biovet, St. Hyacinthe, Quebec, Canada). Cows with serum progesterone <1 ng/mL in both samples were classified as anovular. Body condition was scored on a 5-point scale (Edmonson et al., 1989) 3 wk before and 3 wk after calving. Cows were examined at wk 5 postpartum for purulent vaginal discharge using a Metricheck device (Simcrotech, Hamilton, New Zealand). Cows with muco-purulent or purulent discharge were classified as having purulent vaginal discharge. Cows sold for dairy, domestic, or export purposes were not counted as culls within the first 30 DIM.

A total of 1,346 cows were enrolled in the study. The sample size was based on the underlying randomized trial. Data on disease occurrences, culling, and reproductive performance were extracted from each farm's computerized records (DairyComp 305, Valley Ag Software, Tulare, CA). Data for milk yield (kg/d), milk fat (%), milk protein (%), and SCC were recorded from DHIA records for the first 3 test-day samples (CanWest DHI, Guelph, ON, Canada).

All statistical analyses were completed in SAS (version 9.4, SAS Institute Inc., Cary, NC). Linear regression models (MIXED procedure in SAS) were used to evaluate all continuous outcomes (milk production data; BHB and NEFA concentrations) for associations with UE. Where relevant for blood BHB concentrations and milk yield, repeated measures were accounted for with an autoregressive type 1 covariance structure, selected based on providing the lowest Akaike's information criterion for the final model. In the milk yield models, covariates (DIM, parity, milk protein %, milkfat %, and SCC) were controlled for and removed from the model if they were not significant ($P > 0.05$). Residuals for the models were graphically examined and variables with nonnormal distributions had the outcome log-transformed for analysis. For categorical outcomes (clinical disease, ketosis, culling <30 DIM, and pregnancy at first AI), UE as an independent variable was evaluated using logistic regression models (MIXED or GLIMMIX procedure in SAS). Univariable analysis of the association of UE with categorical outcomes was done with chi-squared statistics before building models. Categorical outcomes from continuous data [BHB $\geq$1.2 mmol/L, NEFA $\geq$0.4 (the week before calving) and $\geq$0.7 or $\geq$1.0 mmol/L (in wk 1 after calving), and ovulation status] were evaluated using logistic regression models (GLIMMIX procedure in SAS).

Preliminary screening was done to determine the appropriate cut point of UE score to classify UE. Cut points at scores 1 and 2 were compared, and based on associations with the outcomes listed, a cut point of 2 was selected.

Each model was initially run with treatment (from the underlying clinical trial), parity (first, second, or third or greater), farm, and all possible interactions with UE status as fixed effects and then was reduced