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Effect of the concentration of circulating prolactin on dairy cows' responsiveness to domperidone injection

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ABSTRACT

The objective of this study was to determine whether the responsiveness of the mammary gland to prolactin (PRL) is affected by the concentration of the hormone. After 1 pre-experimental week (d −7 to −1), 18 Holstein cows in mid to late lactation were injected intramuscularly twice daily with either 0.5 mg of quinagolide (QN) or 2 mL of water (control) for 2 wk (d 1 to 14; treatment period). After the treatment period, all cows received daily subcutaneous injections of 300 mg of domperidone (DOMP) for 3 wk (d 15 to 35; DOMP period). The cows were monitored for an additional 2 wk as a posttreatment period (d 36 to 49). Blood and milk samples were collected 3 times per week. Additionally, blood samples were collected during the a.m. milking on d −4, 14, and 35. Milk production was not affected by QN during the treatment period but was increased during the DOMP and posttreatment periods in the QN cows. With respect to milk composition, the treatments affected only the protein content, which was greater in the QN cows during the treatment period. Blood PRL concentration declined during QN injections and was lower in the QN cows than in the control cows between d 5 and 14. The basal concentration of PRL was increased by DOMP injections during the DOMP and posttreatment periods but was not affected by previous QN injections. Prolactin concentration in milk was not affected by the QN treatments but was increased by DOMP injections during the DOMP and posttreatment periods. Milking-induced PRL release was decreased by QN on d 14. On d 35, milking did not induce a significant release of PRL above the baseline for both treatments. In conclusion, the results of this experiment support the contention that the mammary gland's responsiveness to PRL is modulated by the previous level of the hormone.

Key words: quinagolide, prolactin, lactation, mammary gland

INTRODUCTION

Although the galactopoietic role of prolactin (**PRL**) in ruminants has been controversial for several decades (Knight, 1993), recent studies have shown that the inhibition of PRL with quinagolide (**QN**) decreases milk production in dairy cows (Lacasse et al., 2011; Ollier et al., 2013, 2014). In addition, exogenous PRL tends to increase milk production (Wall et al., 2006) and is able to partially counteract the inhibitory effect of QN on milk production (Lollivier et al., 2015). Furthermore, the administration of the dopamine antagonist domperidone (**DOMP**) increases basal PRL concentration and increases milk production in dairy cows (Lacasse and Ollier, 2015). Therefore, there is now good evidence that PRL is galactopoietic in dairy cows.

Basal PRL concentration, which is affected by the environment, changes throughout the year without similar changes in milk production (Koprowski and Tucker, 1973), suggesting that the mammary gland's sensitivity to PRL is adaptable. Accordingly, a short-day photoperiod during the dry period was found to reduce circulating PRL but increase subsequent milk production (Auchtung et al., 2005; Lacasse et al., 2014). Conversely, heat stress increases PRL concentration, and cooling cows during the dry period is followed by enhanced milk production (Tao and Dahl, 2013). One mechanism that could increase the mammary gland's responsiveness to PRL is an increase in the number of PRL receptors. McKinnon et al. (1988) observed that increasing the milking frequency increased the PRL-binding capacity of the mammary gland, and Knight (1993) found that a unilateral increase in milking frequency increased the milk response to PRL admin-

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istration in goats. It was also observed that the gene expression of long and short isoforms of PRL receptors was higher in mammary glands that were milked more frequently (Bernier-Dodier et al., 2010; Thompson et al., 2015). Taken together, the results of all the experiments cited here suggest that the mammary gland can adapt its sensitivity to PRL in ruminants.

Even though studies have shown the galactopoietic role of PRL, little is known about the factors modulating the responsiveness of the mammary gland to the hormone. Therefore, the objective of this experiment was to evaluate whether the milk response to the PRL secretagogue DOMP is influenced by previous PRL inhibition by QN.

MATERIALS AND METHODS

Animals and Experimental Design

Holstein cows (n = 18; 229 ± 13 DIM; 729 ± 13 kg of BW) from the herd at Agriculture and Agri-Food Canada's Sherbrooke Research and Development Centre (Sherbrooke, QC, Canada) were used for this study and were cared for in compliance with the rules and guidelines of the Canadian Council on Animal Care (1993). The cows showed no clinical signs of mastitis and were assigned to treatments according to their milk yield, SCC, and parity. Of the 18 cows, 9 received twice-daily (at 0700 and 1900 h) intramuscular injections of 0.5 mg of QN (Ferring, Wallisellen, Switzerland) for 2 wk (d 1 to 14; QN treatment), and the other 9 received injections of the solvent (water) as the control (**CTL**) treatment. After the treatment period, all the cows received daily (at 1030 h) subcutaneous injections of 300 mg of DOMP (Glentham Life Sciences Ltd., Corsham, UK) for 3 wk (d 15 to 35; DOMP period). The DOMP was injected as an oil suspension in 10 mL of canola oil. The cows were monitored for an additional 2 wk as the posttreatment period (d 36 to 49). Each cow's feed intake was recorded daily, and each cow's BW was determined at the start and end of the experiment. The cows were kept in a tiestall barn and were milked twice daily, at 0800 and 2000 h, throughout the experiment.

Caudal blood samples were collected at 1030 h 3 times per week from d −5 to 47 using uncoated vacutainers (Becton, Dickinson and Co., Rutherford, NJ). On d −4 (before the start of the treatments), d 14 (at the end of the treatments), and d 35 (at the end of the DOMP period), samples were collected from 4 cows per treatment in tubes without additives before, during, and after the a.m. milking (−20, −10, 0, 3, 5, 7, 10, 15, 20, 25, 30, 40, and 60 min relative to the start of milking) from a Silastic catheter (1.02 mm i.d., 2.16 mm o.d.; Dow Corning Corp., Midland, MI) inserted into the jugular vein. The blood samples were left for approximately 2 h at room temperature for clotting before centrifugation (1,900 × *g*, 4°C, 15 min). Then, the serum was stored at −20°C until determination of PRL concentrations.

Milk samples were collected at the a.m. milking 3 times per week from d −5 to 47. Milk lactose, protein, and fat concentrations and SCC were determined in a commercial laboratory (Valacta Inc., Ste-Anne-de-Bellevue, QC, Canada). In addition, aliquots of milk samples were defatted by centrifugation (1,900 × *g*, 15 min, 4°C) and then kept at −20°C for further analyses of PRL.

Hormone Analysis

The concentration of PRL in the serum and skim milk was measured by RIA as previously described by Bernier-Dodier et al. (2011). Bovine PRL, rabbit antiserum specific for bovine PRL, and goat anti-rabbit gamma globulin were purchased from the National Hormone and Peptide Program (Harbor-UCLA Medical Center, Torrance, CA). The intra- and interassay coefficients of variation were 5.89 and 4.08%, respectively.

Statistical Analysis

Data were analyzed by ANOVA using the MIXED procedure of the SAS software package (SAS Institute Inc., Cary, NC). Time was used as a repeated effect, and animal was used as the subject. Except for the milking-induced PRL release, data were analyzed separately for the pretreatment period (d <1), the treatment period (d 1 to 14), the DOMP period (d 15 to 35), and the posttreatment period (d >35). Means of the data from the pretreatment period were used as covariates. To compare the PRL concentrations during the different periods, the mean concentration was calculated for each period and analyzed by ANOVA using the MIXED procedure, with period used as a repeated effect and animal used as the subject. The amount of PRL released into the blood during milking was calculated by determining the area under the curve between 0 and 30 min relative to the start of milking, and basal PRL concentration was calculated by averaging the concentration obtained between −20 and 0 min. Data for milking-induced PRL were also analyzed by ANOVA using the MIXED procedure, with period used as a repeated effect and animal used as the subject. The proportion of steady-state PRL secreted via milk, which equals the amount of PRL secreted via milk in 24 h multiplied by 100 and divided by the amount of PRL present in the circulation, was calculated accord-

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