New developments on the galactopoietic role of prolactin in dairy ruminants

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In most mammals, prolactin (PRL) is essential for maintaining lactation and its suppression strongly inhibits lactation. However, the involvement of PRL in the control of ruminant lactation is less clear since inconsistent effects on milk yield has been observed with short-term suppression of PRL by bromocriptine. Therefore, a series of experiments was conducted to assess the galactopoietic role of PRL in ruminant using quinagolide, which is a more potent PRL inhibitor that has fewer side effects than bromocriptine. In a first experiment, the effect of the long-term inhibition of PRL release was assessed in early lactating dairy cows. Five Holstein cows received daily i.m injections of 1 mg of quinagolide for 9 wk. Four control cows received the solvent (water) only. During the last wk of the treatments, one udder half was milked once a day (1X) and the other twice a day (2X). The daily injections of quinagolide reduced milking-induced PRL release and induced a faster decline in milk production, which was about 5.3 kg/d lower in the quinagolide-treated cows during the last 4 wk of treatment. Milk production was significantly correlated with the quantity of PRL released at milking. The reduction of milk yield by quinagolide was associated to a reduction in mammary cell activity indicated by reduced milk protein expressions and lower mammary cell survival and proliferation. During wk 9, the inhibition of milk production by quinagolide was maintained in the udder half that was milked 2X but not in the udder half milked 1X, suggesting that response to PRL is modulated at the level of the gland. In a second experiment, 9 cows in mid-lactation were assigned to a 3X3 latin square design where treatments were 5 days of 1) daily i.m. injection of 2 mg of quinagolide; 2) daily quinagolide injection and i.v injection of bPRL (2µg/kg of body weight) twice a day at milking time, or 3) daily injection of water as control. As for the first experiment, quinagolide reduced milk, protein and lactose yields. PRL injections had no effect on milk production but tended to increase milk protein and lactose yields compared to quinagolide alone. Although PRL injections at milking time were not sufficient to restore milk yield, they increased the viability of milk purified mammary epithelial cells and milk protein gene expression were intermediate between control and quinagolide treatment. In a third experiment, dairy goats received daily quinagolide injections (1 mg/injection) for 4 weeks. Surprisingly, quinagolide failed to inhibit PRL. Milk production of these goats was also not affected, thus reinforcing our view that the quinagolide effect on lactation is due to PRL release inhibition. Recently, we have started to investigate the use of PRL-inhibitor to reduce milk production before the drying-off and to accelerate mammary gland involution. Eight cows in late lactation received twice daily i.m. injection of quinagolide (2 mg per injection) from 4 days prior to drying-off to 3 days after; while 8 others received injections of the solvent. Inhibition of PRL-release decreased milk production by 25% within the first day of treatment. We also observed faster increases in somatic cells and BSA content in mammary secretions during early involution suggesting a hastening mammary gland involution. In conclusion, these data combined to those from others now provide a good body of evidence indicating that PRL is galactopoietic in dairy cows. However, the response to PRL appears to be modulated at the mammary gland level.