Prolactin gene expression in bovine mammary epithelial cells after a challenge with coagulase-negative staphylococci

Kristine Piccart[1]*, Mario Van Poucke[2], Fernando N. Souza[3], Sofie Piepers[1], Joren Verbeke[1], Luc Peelman[2] and Sarne De Vliegher[1].

[1] M-team and Mastitis and Milk Quality Research Unit, Department of Reproduction, Obstetrics and Herd Health, Faculty of Veterinary Medicine, Ghent University, Salisburylaan 133, Merelbeke 9820, Belgium.

[2] Animal Genetics Laboratory, Department of Nutrition, Genetics, and Ethology, Faculty of Veterinary Medicine, Ghent University, Heidestraat 19, Merelbeke 9820, Belgium.

[3] Department of Clinical Science, Faculty of Veterinary Medicine and Animal Science, University of São Paulo, São Paulo 05508-270, Brazil.

* For correspondence; e-mail: Kristine.Piccart@UGent.be

Heading

PRL expression in MEC infected with CNS

Summary

This Research Paper addresses the hypothesis that the expression of the prolactin (PRL) gene increases in bovine mammary epithelial cells (MEC) following an infection with coagulase-negative staphylococci (CNS). Various studies have demonstrated in the past that dairy cattle with an intramammary infection caused by CNS have a higher milk yield than non-infected dairy cows. It has been suggested that this could be the result of an increased synthesis of the autocrine lactation hormone prolactin (PRL), since PRL is also involved in the inflammatory response of MEC during mastitis. Bovine MEC (more specifically MAC-T cells) were therefore inoculated using three well-defined CNS strains from varying habitats: one strain of *Staphylococcus fleurettii* originating from sawdust, and two different strains of *Staphylococcus chromogenes* (one isolated from a heifer's teat apex [*Staphylococcus chromogenes* TA], the other from a persistent intramammary infection [*S. chromogenes* IM]). Although *PRL* was expressed in all samples, the expression was not higher in CNS-challenged cells compared to the unchallenged control cells. The elevated PRL level previously observed in milk from cows with CNS infection might rather be the result of a disruption of the blood-milk barrier, although this needs further substantiation.