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Milk prolactin response and quarter milk yield after experimental infection with coagulase-negative staphylococci in dairy heifers

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ABSTRACT

Coagulase-negative staphylococci (CNS) are the most common bacteria involved in subclinical mastitis in dairy cows. Remarkably, CNS-infected dairy heifers produce more milk than uninfected heifers. Because the lactation hormone prolactin (PRL) is also involved in mammary gland immunity, we investigated the milk PRL response and the mammary quarter milk yield following experimental CNS challenge. Eight healthy Holstein-Friesian heifers in mid-lactation were experimentally infected using a split-udder design with 3 different CNS strains: one *Staphylococcus fleurettii* (from sawdust bedding) and 2 *Staphylococcus chromogenes* strains (one isolate from a teat apex, the other isolate from a chronic intramammary infection). Three mammary quarters per heifer were simultaneously inoculated with 1.0×10^6 cfu, whereas the remaining mammary quarter was infused with sterile phosphate-buffered saline, serving as a control. An existing radioimmunoassay was modified, validated, and used to measure PRL frozen-thawed milk at various time points until 78 h after challenge. The mean milk PRL level tended to be higher in the CNS-challenged mammary quarters compared with the control mammary quarters (7.56 and 6.85 ng/mL, respectively). The increase in PRL over time was significantly greater in the CNS-challenged mammary quarters than in the control mammary quarters. However, no difference was found in the PRL response when comparing each individual CNS strain with the control mammary quarters. The mean mammary quarter milk yield tended to be lower in the CNS-infected mammary quarters than in the control mammary quarters (1.73 and 1.98 kg per milking, respectively). The greatest milk loss occurred in the mammary quarters challenged with the intramammary strain of *S. chromogenes*. Future observational studies are needed to elucidate the relation between PRL, the milk yield, and the inflammatory condition, or infection status, of the mammary gland.
Key words: coagulase-negative staphylococci, dairy heifer, experimental mastitis, prolactin

INTRODUCTION

Bovine mastitis, an inflammation of the mammary gland, creates a huge economic burden on the global dairy industry (Bradley, 2002). Coagulase-negative staphylococci are the predominant group of bacteria involved in subclinical mastitis (Pyörälä and Taponen, 2009) and can cause clinical mastitis with mild symptoms (Taponen et al., 2006). Thus far, more than 10 species of CNS have been isolated from bovine milk (Piessens et al., 2011) with documented species-specific differences in putative virulence (Vanderhaeghen et al., 2014), ecology, and epidemiology (Vanderhaeghen et al., 2015). Contrary to what one might expect, various studies have observed a higher test-day milk yield in CNS-infected dairy heifers and multiparous cows compared with noninfected cows (Compton et al., 2007; Schukken et al., 2009; Piepers et al., 2010). Some studies have attributed a protective effect to pre-existing CNS IMI against IMI with more virulent mastitis pathogens (e.g., Piepers et al., 2010). A meta-analysis could not confirm this finding in observational studies, but nonetheless revealed a pronounced protective effect in challenge trials (Reyher et al., 2012). Still, the positive effect on milk yield could be an indirect result of the reduced incidence of clinical mastitis observed in CNS-infected animals (Piepers et al., 2010). High-producing dairy cows might also be more susceptible to CNS IMI than low-yielding animals (Compton et al., 2007). However, even after correcting for these factors, an unexplained difference in milk yield of 2.0 kg/d remained between CNS-infected and uninfected herd mates (Piepers et al., 2013), leaving the exact mechanism to be determined.

Prolactin (**PRL**) has been associated with over 300 different biological actions, including lactation and mammary gland development (Bole-Feysot et al.,

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1998). In dairy cattle, PRL is required to initiate (Akers et al., 1981) and maintain the milk production after parturition (Lacasse et al., 2012). The protein hormone also acts as a cytokine on molecular and functional levels (Goffin et al., 2002). The ubiquitous PRL receptor belongs to the class I cytokine receptor superfamily, which also includes the receptors of several interleukins and hematopoietic growth factors (Bazan, 1989, 1990). The hormone promotes the activity of macrophages (Edwards et al., 1987), inhibits the apoptosis of T-lymphocytes caused by glucocorticoids (Krishnan et al., 2003), and stimulates the production of tumor necrosis factor-α and IL-12 (Brand et al., 2004). Considering the immunomodulatory actions of PRL, several studies have focused on its potential involvement in bovine mastitis. The periparturient PRL peak coincides with the principal risk period for developing mastitis (Burton et al., 2001). The hormone induces the in vitro synthesis of several cytokines in bovine mammary epithelial cells through the activation of nuclear factor kappa B (Boutet et al., 2007). Although the circulating PRL level is not affected by acute, clinical mastitis (Hockett et al., 2000; Vanselow et al., 2006), a positive correlation was found between SCC and PRL concentration in milk of chronically infected mammary quarters (Boutet et al., 2007).

Because PRL is recognized as a pro-inflammatory cytokine, we hypothesize that milk PRL increases in response to an IMI with CNS. Furthermore, we hypothesize that the quarter milk yield (**QMY**) also increases after CNS IMI, assuming PRL stimulates the production of milk. To investigate this, an experimental challenge trial was set up using 8 clinically healthy, mid-lactating dairy heifers using 3 different CNS strains. An existing radioimmunoassay for fresh milk was modified and subsequently validated to measure bovine PRL in frozen-thawed milk samples. To assess the epithelial integrity of the blood-milk barrier, the sodium, potassium, and chloride levels were also determined in milk.

MATERIALS AND METHODS

The study is in compliance with the European Directive 2010/63/EU and was approved by the ethical committee of the Faculty of Veterinary Medicine, Ghent University (EC2012/73).

Animals

The study took place between December 2012 and May 2013 at the research dairy farm of Ghent University (Biocentrum Agri-Vet, Melle, Belgium). Eight clinically healthy Holstein-Friesian heifers in mid-lactation (78–278 DIM) were selected. Heifers with a known history of clinical mastitis or persistent high SCC (>150,000 cells/mL) were excluded from the trial. Milk samples were cultured according to NMC guidelines 48 and 24 h before inoculation to ensure all mammary quarters were free from IMI (NMC, 1999).

CNS Strains

All heifers were inoculated with 2 different wild strains of *Staphylococcus chromogenes* and 1 *Staphylococcus fleurettii* strain. The *S. fleurettii* isolate was recovered from sawdust bedding in a dairy barn (Piessens et al., 2011; Breyne et al., 2015). The first *S. chromogenes* strain originated from a cow suffering from a persistent IMI (hereafter referred to as *S. chromogenes* **IM**; Supré et al., 2011; Breyne et al., 2015), whereas the second *S. chromogenes* isolate was cultured from the teat apex of a heifer (hereafter referred to as *S. chromogenes* **TA**; De Vliegher et al., 2004; Breyne et al., 2015). The *S. chromogenes* TA strain has the ability to inhibit the growth of several major pathogens under laboratory conditions (De Vliegher et al., 2004), whereas the *S. chromogenes* IM strain does not. The 2 strains also elicit a different immune response in mice (Breyne et al., 2015), and the TA strain is unable to grow in anaerobic iron-depleted medium unlike the IM strain (unpublished data). An inoculum of 1.0×10^6 cfu of each strain was prepared to induce an experimental infection. The live number of cfu was determined by plating serial dilutions of the bacterial stock on tryptic soy agar.

Experimental Study Design

A split-udder design was used. The concept of the split-udder model is grounded on within-heifer comparisons to reduce individual variation (Sipka et al., 2014). Following the morning milking, 3 mammary quarters of each heifer were instantaneously inoculated with the 3 aforementioned CNS strains (one per mammary quarter) diluted in 5 mL of PBS using a sterile catheter (Vygon, Ecouen, France). The fourth mammary quarter, serving as a control, was infused in the same manner with 5 mL of sterile, pyrogen-free PBS. Milk samples for PRL analysis and microbiological culturing were collected from each mammary quarter at 0, 4, 6, 9, 12, 18, 24, 28, 32, 36, 48, 54, 60, 72, and 78 h postinoculation (**PI**). Milk samples for ion analysis were collected at 0, 24, and 48 h PI. The milk SCC was determined using a DeLaval Cell Counter (DeLaval, Tumba, Sweden). Bacteriological culturing was performed according to NMC guidelines (NMC, 1999). The milk samples for the PRL and ion analysis were stored at −20°C. The cows were milked twice a day with 12-h intervals, and QMY was registered using a mammary

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