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Heifers infected with coagulase-negative staphylococci in early lactation have fewer cases of clinical mastitis and higher milk production in their first lactation than noninfected heifers

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ABSTRACT

Intramammary infections (IMI) in recently calved dairy heifers are more common than was formerly believed but their relevance for future performance has been studied only rarely. In the present study, the association between the IMI status of fresh heifers and their subsequent udder health, milk production, and culling in first lactation was explored. Quarter milk samples were collected between 1 and 4 d in milk (DIM) and between 5 and 8 DIM from 191 dairy heifers in 20 dairy herds for bacteriological culturing and somatic cell count (SCC) analysis. Monthly milk recording data including composite milk SCC and test-day milk yield (MY) were obtained for the first 285 DIM or until culling. Farmer-recorded clinical mastitis cases were available. Data were analyzed using mixed models and survival analysis. Approximately 80% of the fresh heifers (79.8%) had at least one culture-positive quarter. Coagulase-negative staphylococci (CNS) were the most frequently isolated pathogens (72%), followed by esculin-positive streptococci (4.6%) and *Staphylococcus aureus* (3.5%). Overall geometric mean SCC at quarter level decreased between the first and second samplings from 348,000 to 116,000 cells/mL. Heifers infected with CNS had an intermediate average test-day SCC (84,000 cells/mL) during the first 285 DIM compared with noninfected heifers (53,000 cells/mL) and heifers infected with major pathogens (195,000 cells/mL). Heifers infected with major pathogens had a much lower average daily MY (18.3 kg) during first lactation compared with noninfected animals (21.3 kg). That CNS-infected heifers out-produced their noninfected counterparts could be at least partially explained by their significantly lower incidence of clinical mastitis (incidence risk 3.6 vs. 21.0%) during first lactation compared with noninfected heifers. We conclude that although CNS cause the majority of IMI in heifers around calving, they should not be a reason for serious concern.
Key words: coagulase-negative staphylococci, early lactation, heifer, intramammary infection

INTRODUCTION

During the last 20 yr, several studies have reported a high prevalence of IMI in dairy heifers (reviewed in Fox, 2009). In Flanders, Belgium, 35% of heifers in early lactation were suspected to suffer from subclinical mastitis as evidenced by a composite milk SCC >150,000 cells/mL between 5 and 14 DIM (De Vliegher et al., 2001). Furthermore, the early lactation period of dairy heifers is characterized by a high incidence rate of clinical mastitis (**CM**) (Barkema et al., 1998; Nyman et al., 2007; Olde Riekerink et al., 2008). More than 30% of the CM cases in heifers occur in the first 2 wk of lactation, which is substantially higher than in multiparous cows (Barkema et al., 1998).

Subclinical mastitis in heifers in early lactation has been associated with financial losses incurred by impaired milk production, an increased risk of subclinical and clinical mastitis, and premature removal in first lactation (Huijps et al., 2009). Heifers with a SCC of 50,000 cells/mL at 10 DIM produced 119 and 155 kg more milk during the first 305 d of the lactation compared with heifers with SCC of 500,000 and 1,000,000 cells/mL, respectively (De Vliegher et al., 2005b). However, the data indicated that the impact was related to the pathogen involved in elevating the early SCC (De Vliegher et al., 2004, 2005a, b).

Because of the high prevalence of heifer mastitis and its potentially compromising effects for future productivity, prepartum treatment of heifers with antibiotics has been considered as a measure to reduce the prevalence of IMI at calving, and was found to be effective in some (e.g., Sampimon et al., 2009) but not all studies (e.g., Borm et al., 2006). Still, the most commonly recovered pathogens from milk from fresh heifers belong to the CNS group (reviewed in Fox, 2009). Although their harmless nature has recently been disputed (Ta-

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ponen et al., 2006), CNS as a group are still regarded as minor pathogens causing mainly transient infections (Schukken et al., 2009). In the latter study, cows and heifers infected with CNS had a slightly higher milk production at test-day than did noninfected cows and heifers. Despite the high prevalence of CNS infections in fresh dairy heifers, little information is available about the relevance of such infections for future productivity, with the exception of 2 studies (Kirk et al., 1996; Compton et al., 2007).

The higher SCC in mammary secretions collected before calving and shortly thereafter from CNS-infected quarters compared with those from noninfected quarters implies that the infections at least provoke an inflammatory response in the heifer's udder (Trinidad et al., 1990; Hallberg et al., 1995; Barkema et al., 1999). On the other hand, the geometric mean SCC of quarters infected with CNS decreases in the first 3 d after calving (Barkema et al., 1999). Compared with CNS, *Staphylococcus aureus* and environmental pathogens are less frequently isolated from fresh heifer milk, but are expected to cause more long-lasting damage (Trinidad et al., 1990; Hallberg et al., 1995; Barkema et al., 1999) as they are more likely to persist into lactation (Roberson et al., 1994). In this regard, one could question whether the long-term effects of CNS IMI are detrimental enough to warrant treating all heifers before calving and whether the recommendation of this practice should not be limited to herds with a high prevalence of either subclinical mastitis caused by *Staphylococcus aureus* (Barkema et al., 2006) or environmental streptococci or clinical mastitis.

The main objective of the present study was to explore the effect of pathogen-specific IMI in early lactating heifers on udder health (SCC and clinical mastitis), milk production, and culling hazard during first lactation; specifically, we wanted to determine the relevance of IMI caused by CNS for heifer performance during first lactation.

MATERIALS AND METHODS

Herds, Heifers, and Sampling

In total, a convenience sample of 191 dairy heifers from 20 dairy herds (average of 9 heifers per herd, range 4 to 10) participating in the DHI program in Flanders (CRV, Oosterzele, Belgium) were included in the study. The herds were randomly selected from a database of the Flemish Cattle Breeding Association (Oosterzele, Belgium) including all dairy herds (n = 241) within a radius of 30 km of the Faculty of Veterinary Medicine (Merelbeke, Belgium) using the Excel RAND function (Excel 2007, Microsoft Corp., Redmond, WA). Herds treating heifers with antibiotics before calving were not included. However, none of the selected herds needed exclusion for this reason.

Quarter milk samples were aseptically collected by a veterinarian (the first author) at 1 to 4 DIM (sampling 1, **S1**) and at 5 and 8 DIM (sampling 2, **S2**) for bacteriological culture (5 mL) and determination of quarter milk SCC (**qSCC**; 30 mL) between March 2006 and December 2007. An interval of at least 3 d was maintained between the samplings. All milk samples were collected before morning milking after disinfection of the teats and after the first streams of milk were discarded. Milk samples were immediately stored at 4°C and then transported under cooled conditions to the laboratory (Animal Health Service, Torhout, Belgium).

Outcome Variables

Composite milk SCC and milk yield (**MY**; kg of milk/d) at test-day until 285 DIM were used per heifer on a 4- to 6-wk basis as part of the DHI program.

Clinical mastitis cases were detected by the farmer based on clinical signs [any abnormal aspect of the milk (flakes, clots, and a watery or other unusual appearance) with or without other visible abnormalities of the udder (e.g., redness, swelling)] during the first 285 DIM or until culling. The date of occurrence and the identification number of the heifer and the affected quarter(s) were recorded. Heifers with CM were treated by the farmer according to the usual protocol for that herd. The incidence rate of CM was calculated and expressed as the number of quarter cases per 10,000 heifer-days at risk from calving to 285 DIM. The interval between cases of CM in the same quarter had to be $\geq$14 d for a case to be included in the analysis (Barkema et al., 1998). Clinical mastitis cases in a different quarter within the same heifer were considered as a new case, independently of the time interval. Heifer-days at risk were calculated as the total number of days that a heifer was present in the herd, starting from parturition to 285 DIM, minus 14 d after each case of CM.

Date of culling and 1 of the 6 classifications (low milk production, reproductive disorders, udder disorders, foot-leg problems, death, and nonspecific reasons) for culling of all heifers was recorded by the farmer and was available for further analyses.

Laboratory Analyses

Bacteriological culture was done as described previously (Piepers et al., 2007). Briefly, 0.01 mL of milk was plated on a blood-esculin agar (Oxoid, Erembodegem, Belgium; 1 plate per cow) and on MacConkey's agar (Oxoid; 1 plate per cow). All plates were incubated