Effect of feeding heat-treated colostrum on risk for infection with *Mycobacterium avium* ssp. *paratuberculosis*, milk production, and longevity in Holstein dairy cows

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ABSTRACT

In summer 2007, a randomized controlled field trial was initiated on 6 large Midwest commercial dairy farms to investigate the effect of feeding heat-treated (HT) colostrum on transmission of Mycobacterium avium ssp. paratuberculosis (MAP) and on future milk production and longevity within the herd. On each farm, colostrum was collected daily from fresh cows, pooled, divided into 2 aliquots, and then 1 aliquot was heat-treated in a commercial batch pasteurizer at 60°C for 60 min. A sample from each batch of colostrum was collected for PCR testing (MAP-positive vs. MAP-negative). Newborn heifer calves were removed from the dam within 30 to 60 min of birth and systematically assigned to be fed 3.8 L of either fresh (FR; n = 434) or heattreated (HT; n = 490) colostrum within 2 h of birth. After reaching adulthood (>2 yr old), study animals were tested once annually for 3 yr (2010, 2011, 2012) for infection with MAP using serum ELISA and fecal culture. Lactation records describing milk production data and death or culling events were collected during the 3-yr testing period. Multivariable model logistic and linear regression was used to investigate the effect of feeding HT colostrum on risk for testing positive to MAP during the 3-yr testing period (positive/negative; logistic regression) and on first and second lactation milk yield (kg/cow; linear regression), respectively. Cox proportional hazards regression was used to investigate the effect of feeding HT colostrum on risk and time to removal from the herd. Fifteen percent of all study animals were fed PCR-positive colostrum. By the end of the 3-yr testing period, no difference was noted in the proportion of animals testing positive for MAP, with either serum ELISA or fecal culture, when comparing the HT group (10.5%) versus the FR group (8.1%). There was no effect of treatment on first- (HT = 11.797 kg);

Received February 8, 2015.

FR = 11,671 kg) or second-lactation (HT = 11,013 kg; FR = 11,235 kg) milk production. The proportion of cows leaving the herd by study conclusion was not different for animals originally fed HT (68.0%) versus FR (71.7%) colostrum. Although a previous study showed that feeding HT colostrum (60°C for 60 min) produces short-term benefits, including improved passive transfer of IgG and reduced morbidity in the preweaning period, the current study found no benefit of feeding HT colostrum on long-term outcomes including risk for transmission of $Mycobacterium\ avium\ ssp.\ paratuberculosis$, milk production in the first and second lactation, and longevity within the herd.

Key words: colostrum, heat treatment, Johne's disease, *Mycobacterium avium* ssp. *paratuberculosis*, pasteurize

INTRODUCTION

Johne's disease is a chronic intestinal infection of ruminants caused by *Mycobacterium avium* ssp. paratuberculosis (MAP). At an estimated \$100 loss per cow in Johne's-positive herds, the disease is one of the most economically important infectious diseases of US dairy cattle, with losses attributed to progressive weight loss, reduced milk production and premature culling (USDA, 1997; Ott et al., 1999). It was recently estimated that MAP is present in 91.1% of US dairy herds (Lombard et al., 2013).

Whereas new infections do occur in adult animals (Espejo et al., 2012), Johne's disease-control programs primarily emphasize the adoption of management practices designed to prevent transmission to newborn calves and youngstock (Kudahl et al., 2008). Fecal—oral transmission from contaminated environments is deemed the most important source of exposure. However, infective colostrum or milk represents an additional potential source of exposure to MAP. This could arise from fecal contamination of milk or colostrum from teat skin during harvest, or from direct shedding of the organism into the mammary system of infected dams (Pithua et al., 2011). Sweeney et al., (1992) reported that 27% of

Accepted April 19, 2015.

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subclinically infected cows had culture-positive supramammary lymph nodes and 12% had culture-positive milk. Streeter et al. (1995) reported that up to 22% of infected cows shed the organism in milk and colostrum. Whereas the importance of colostrum and milk in MAP transmission has not been well described, Johne's control programs generally include such management recommendations as avoiding the feeding of pooled colostrum, feeding colostrum from test-negative cows, use of colostrum-replacement products, and feeding commercial powdered milk replacers (Nielsen et al., 2008; Pithua et al., 2009, 2011).

Another strategy to mitigate this risk may be the pasteurization of milk and colostrum using commercially available on-farm pasteurization systems. The efficacy of pasteurization to destroy MAP in milk remains controversial: Most in-laboratory or on-farm inoculation studies simulating batch pasteurization (63°C for 30 min) or HTST pasteurization (72°C for 15 s; PMO, 2007) report that the process is completely effective in destroying this pathogen in milk (Stabel et al., 1996; Keswani and Frank, 1998; Grant et al., 1999; Stabel, 2001; Stabel et al., 2003). However, some other laboratory-based inoculation studies simulating HTST pasteurization have reported that small numbers of MAP colonies may remain viable if the organism is inoculated into milk at higher concentrations (Chiodini and Hermon-Taylor, 1993; Grant et al., 1996; Sung and Collins, 1998; Gao et al., 2002).

Developing a technique to pasteurize colostrum on farms has proven to be much more challenging. Early studies attempting to pasteurize colostrum using traditional Pasteurized Milk Ordinance (PMO, 2007) temperatures have yielded unacceptable results. Batch pasteurization of colostrum at $63^{\circ}\text{C} \times 30$ min resulted in a 24 to 59% reduction in colostral IgG concentration, depending on batch size and quality, and resulted in a significant reduction in serum IgG concentrations in calves (Godden et al., 2003). Unpublished trials by thesame investigator (S. Godden) found that HTST pasteurization (72°C × 15 s) resulted in a solid pudding-like product that was impossible to feed and plugged up or cooked onto equipment, resulting in significant cleaning challenges.

The aforementioned failures with traditional pasteurization temperatures led this team of researchers to develop a lower-temperature, longer-time approach to heat-treat (**HT**) colostrum, the goal being to reduce pathogen exposure while still preserving important colostral IgG and viscosity characteristics. Early laboratory inoculation studies reported that heating bovine colostrum at 60°C for 60 min did not damage colostral IgG but significantly reduced concentrations of viable MAP and eliminated other important pathogens, in-

cluding Mycoplasma bovis, Listeria monocytogenes, Escherichia coli, and Salmonella enteritidis (McMartin et al., 2006; Godden et al., 2006). Several subsequent field studies have demonstrated that colostrum may be successfully heat-treated on farm $(60^{\circ}\text{C} \times 60 \text{ min})$, in larger pooled batches or in individual 3.8-L aliquots, resulting in a significant reduction in total bacteria counts and total coliform counts while having no negative effect on colostral IgG (g/L), DM (%), true protein (%), crude fat (%), lactose (%), SNF (%), other solids (%), insulin (ng/mL), lactoferrin (mg/mL), and IGF (ng/mL; Johnson et al., 2007; Donahue et al., 2012; Godden et al., 2012b; Kryzer et al., 2015).

Multiple field studies have consistently reported that calves fed HT colostrum experience significantly improved efficiency of IgG absorption resulting in higher serum IgG concentrations as compared with calves fed fresh (FR) colostrum (Johnson et al., 2007; Godden et al., 2012b; Kryzer et al., 2015). Though the exact mechanism to explain this has not been confirmed, it is hypothesized that improved passive transfer occurs because of reduced bacterial interference with IgG absorption across the small intestine (James et al., 1981; Poulson et al., 2002; Peterson et al., 2008). Finally, in one multiherd randomized field trial involving 1,071 calves from 6 large commercial Midwest dairy herds, the hazard for being treated for scours or for being treated for any illness during the preweaning period was 1.32 (95% CI: 1.14, 1.53) and 1.25 (95% CI: 1.08, 1.44), respectively, for calves fed FR colostrum compared with calves fed HT colostrum (Godden et al., 2012b). A pathway analysis suggested that the health benefits associated with feeding HT colostrum may be attributed to improved passive transfer of IgG.

Based on the benefits to neonatal calves reported by these earlier studies, the US dairy industry has already begun the process of adopting the technique of HT colostrum on farms. The USDA-NAHMS 2007 Dairy study (USDA, 2008a) reported that 6.4% of large dairies (500 or more cows) and 0.8% of all dairy operations that hand-fed colostrum reported heat-treating colostrum before feeding. However, this is still a relatively new management tool and research is still needed to fully investigate if calves fed HT colostrum will also experience long-term health or performance benefits, including reduced risk for infection with MAP. In 2007, a multiherd randomized clinical trial was initiated using 1,071 newborn heifer and bull calves on 6 commercial dairy farms in Minnesota and Wisconsin; the effects of HT on colostrum characteristics and on calf health in the preweaning period have already been reported (Donahue et al., 2012; Godden et al., 2012a,b). The major objective of the current study was to follow heifer calves from this large field study into adulthood

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