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Acute brief heat stress in late gestation alters neonatal calf innate immune functions¹

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ABSTRACT

Heat stress, as one of the environmental stressors affecting the dairy industry, compromises the cow milk production, immune function, and reproductive system. However, few studies have looked at how prenatal heat stress (HS) affects the offspring. The objective of this study was to evaluate the effect of HS during late gestation on calf immunity. Calves were born to cows exposed to evaporative cooling (CT) or HS (cyclic 23–35°C) for 1 wk at 3 wk before calving. Both bull and heifer calves (CT, $n = 10$; HS, $n = 10$) were housed in similar environmental temperatures after birth. Both CT and HS calves received 3.78 L of pooled colostrum within 12 h after birth and were fed the same diet throughout the study. In addition to tumor necrosis factor α , IL-1 β , IL-1 receptor antagonist (IL-1RA), and toll-like receptor (TLR)2, and TLR4 mRNA expression, the expression of CD14⁺ and CD18⁺ cells, and DEC205⁺ dendritic cells were determined in whole blood samples at d 0, 3, 7, 14, 21, and 28. The neutrophil to lymphocyte ratio, differential cell counts, and the hematocrit were also determined. During late gestation, the HS cows had greater respiration rates, rectal temperatures, and tended to spend more time standing compared with the CT cows. The HS calves had less expression of tumor necrosis factor- α and TLR2 and greater levels of IL-1 β , IL-1RA, and TLR4 compared with CT calves. The HS calves also had a greater percentage of CD18⁺ cells compared with the CT calves. Additionally, a greater percentage of neutrophils and lesser percentage of lymphocytes were in the HS calves compared with the CT calves. The results indicate that biomarkers of calves' immunity are affected in the first several weeks after birth by HS in the dam during late gestation.

Key words: dairy cattle, calf, heat stress, innate immunity

INTRODUCTION

Heat stress (HS) is a major environmental concern in the dairy industry. In dairy cattle, HS occurs when ambient temperatures are above 25°C (Armstrong, 1994). Heat stress causes greater rectal temperatures (RT) and elevated respiration rates (RR; Ominski et al., 2002; do Amaral et al., 2011). Dairy cattle also spend less time lying when exposed to hot temperatures (Overton et al., 2002; Legrand et al., 2011). In addition to behavioral responses, HS in dairy cattle reduces feed intake (Adin et al., 2009), milk production (Collier et al., 2006), and reproductive performance (Hansen, 2009). Heat exposure before parturition caused decreased neutrophil phagocytosis and oxidative burst (do Amaral et al., 2011) after animals were returned to a thermoneutral environment following calving. Reduction of lymphocyte proliferation (Lacetera et al., 2006) and reduced IgG concentrations (do Amaral et al., 2011) occurred in cows under HS during the dry period and decreased (do Amaral et al., 2010) tumor necrosis factor α (TNF- α) responses of lymphocytes from HS cows. The immune impairment due to HS can cause increased susceptibility of dairy cattle to many diseases (Lacetera et al., 2006; do Amaral et al., 2009).

Maternal HS during late gestation also inhibits the immune response of the offspring, by modifying T and B cell function (Merlot et al., 2008), decreasing IgG concentration in the calf (Donovan et al., 1986; Tao et al., 2012), and compromising the proliferation rate of mononuclear cells (Tao et al., 2012). Moreover, sows under HS during the last 2 wk of gestation causes lower circulating IgG in piglets compared with piglets from sows under a thermoneutral environment (Machado-Neto et al., 1987). The immune system in the neonate is fully developed at birth but it is unprimed (Tizard, 1992). The fetus is protected primarily by the innate immune system, but the phagocytic activity of its immune cells is not fully developed until late in gestation

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(Barrington, 2001). B cells in the fetus only make up 1% of total lymphocytes compared with the 4% at 1 wk after birth and with the 10% in mature calves (Kampen et al., 2006). This results in the lack of any endogenous antibody response until 2 to 4 wk of age making the ingestion of colostrum extremely important in providing an immunologic defense to the calf during the first 2 to 4 wk of life (Chase et al., 2008). Adequate colostrum transfer has been recognized to have beneficial effects on the calf's immune response earlier in life (Furman-Fratczak et al., 2011), by the uptake of cytokines (Nguyen et al., 2007; Chase et al., 2008) and the absorption of immunoglobulins during the first 48 h (Sangild, 2003). Recent work has isolated the effects of colostrum from heat-stressed cows and the absorptive ability of the neonate from heat-stressed cows (Monteiro et al., 2014), determining that calves had reduced passive transfer regardless of colostrum source. Previous research of our laboratory (unpublished data), conducted during the summer with a short heat event during the last month of gestation, resulted in calves that were more prone to disease and grew slower even when born after the HS event, prompting the hypothesis of this research.

Currently, few data are available on the effects of maternal HS during late gestation on the immune function in dairy cattle. Therefore, the objective of the present study was to evaluate the effect of a brief maternal HS during the late gestation period on the postnatal immune function of dairy calves. Our hypothesis was that a brief late gestation heat stress would alter neonatal calf innate immune measures.

MATERIALS AND METHODS

Animals and Experimental Design

This study was conducted at the Purdue Animal Sciences Research and Education Dairy Unit from May to September 2014. The experimental protocol was approved by the Animal Care and Use Committee at Purdue University. Twenty multiparous Holstein cows were randomly assigned to 1 of 2 environmental treatments, control (CT) or HS, and moved into a metabolism room approximately 3 wk before calving. All cows were managed in a metabolism barn with tie stalls. A single treatment was in the room at one time, using 3 replications per treatment. Cows were moved into the metabolism room for an acclimation period of 7 d and then were on the treatment for the following 7 d. The metabolism barn for the CT cows was equipped with evaporative cooling equipment. For the HS cows, the temperature was increased to $35^{\circ}\text{C} \pm 6$ over 12 h each

Table 1. Guaranteed analysis of milk replacer and starter for calves

Nutrient	Milk replacer ¹	Calf starter ²
CP, minimum, %	22	20
Fat minimum, %	20	4.5
Crude fiber minimum, %	0.15	6.0
ADF minimum, %		8.0
Ca minimum, %	1.0	1.0
P minimum, %	0.7	0.3
Salt minimum, %		0.525
Selenium minimum, %		0.3
Vitamin A, IU/kg	44,000	19,800
Vitamin D ₃ , IU/kg	11,000	4,400
Vitamin E, IU/kg	220	110
Monensin, g/t		40 g/ton

¹Land O'Lakes, Amplifier Max MOS, Shoreview, MN.

²Momenta, Vita Plus Corporation, Madison, WI.

day and was reduced to $23^{\circ}\text{C} \pm 4$ over 12 h each night. Following the treatment conditions, cows were moved to box stalls under geothermal, thermal neutral ($22^{\circ}\text{C} \pm 2$), environmental conditions for approximately 1 wk before their expected calving date.

Both bull and heifer calves born to those cows were used in the current study (CT: bulls = 6, heifers = 4; HS: bulls = 4, heifers = 6). Both CT (n = 10) and HS (n = 10) calves received 3.78 L of pooled superior (defined as greater than 50 mg/mL of IgG) colostrum (from multiparous cows in geothermally controlled environments) within 12 h after birth and were fed the same diet throughout the study. Calves from both treatments were removed from their dam immediately after birth and housed in individual hutches exposed to ambient temperatures. Calves were fed commercial milk replacer of all milk components (Table 1) and offered dry feed (Table 1) from d 3. Water was replaced twice daily and always available.

Measurements and Sample Collection

The respiration rates (RR), rectal temperatures (RT), and posture of cows in both treatments were monitored hourly for 12 h during 7 d of HS or CT treatment from 0800 to 1900 h. Two consecutive RR were measured in breaths per minute using a stopwatch. The average of those 2 consecutive RR per cow was recorded hourly. The posture of the cow, and whether they were lying or standing at the moment before RR, were counted (an hourly instantaneous scan sample) and calculated as the percentage of observations. The RT were measured using a digital thermometer hourly and disinfected between individual cows with 70% isopropyl alcohol. The room temperature and relative humidity were recorded hourly by taking the average of

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