J. Dairy Sci. 99:1–6
http://dx.doi.org/10.3168/jds.2015-9990

Effects of late-gestation heat stress on immunity and performance of calves[1]

G. E. Dahl,*[2] S. Tao,† and A. P. A. Monteiro†
*Department of Animal Sciences, University of Florida, Gainesville 32611
†Department of Animal and Dairy Science, University of Georgia, Tifton 31793

ABSTRACT

Lactating cows that experience heat stress will have reduced dry matter intake and milk yield and shift metabolism, which ultimately reduces the efficiency of milk production. Dry cows that are heat stressed similarly experience lower intake, reduced mammary growth, and compromised immune function that ultimately results in a poorer transition into lactation and lower milk yield in the next lactation. A recent focus in our laboratory is on the effects of late gestation, in utero heat stress on calf survival and performance. We have completed a series of studies to examine preweaning growth and health, and later reproductive and productive responses, in an attempt to quantify acute and persistent effects of in utero heat strain. Late gestation heat stress results in calves with lower body weight at birth, shorter stature at weaning, and failure to achieve the same weight or height at 12 mo of age observed in calves from dams that are cooled when dry. A portion of the reduced growth may result from the lower immune status observed in calves heat stressed in utero, which begins with poorer apparent efficiency of immunoglobulin absorption and extends to lower survival rates through puberty. Heat-stressed calves, however, have permanent shifts in metabolism that are consistent with greater peripheral accumulation of energy and less lean growth relative to those from cooled dams. Comparing reproductive performance in calves heat stressed versus those cooled in utero, we observe that the cooled heifers require fewer services to attain pregnancy and become pregnant at an earlier age. Tracking the milk production in calves that were heat stressed in utero versus those cooled in late gestation revealed a significant reduction of yield in the first lactation, approximately 5 kg/d through 35 wk of lactation, despite similar body weight and condition score at calving. These observations indicate that a relatively brief period of heat stress in late gestation dramatically alters the health, growth, and ultimate performance of dairy calves. Thus, it is critical to effectively manage heat stress of dry cows to avoid negative effects on the calf.

Key words: calf, in utero heat stress, growth, health

INTRODUCTION

Heat stress causes significant negative responses during all phases of the life and production cycle of dairy cattle. Indeed, when lactating cows experience heat stress, they reduce intake, alter metabolic priorities, and eventually suffer a reduction in milk yield (Collier et al., 2006; Baumgard and Rhoads, 2013). Calves also suffer under heat stress, with dramatic reductions in growth apparent before and after weaning (Broucek et al., 2009). Of interest, Broucek et al. (2009) observed lower growth without reductions in feed intake, suggesting a reduction in feed efficiency of heat-stressed calves relative to those under moderate environmental conditions. During the postweaning growth phase, heat stress reduces ADG of calves (O'Brien et al., 2010). In contrast to mature cows, this growth decrement can be explained by reduced DMI alone rather than the interaction of lower DMI and metabolic shifts (Rhoads et al., 2010). It is clear, however, that heat stress reduces productivity of growing and lactating cows.

In addition to the negative effect on production, heat stress may also depress immune function in cows, although evidence of direct effects is scarce. Elvinger et al. (1992) reported a reduction in circulating leukocyte migration into the mammary gland under heat stress relative to thermoneutral conditions, and the effect was independent of yield as bST treatment did not alter the response to heat stress despite greater milk production. Indirect evidence of negative effects of heat stress on immune status in cows is apparent from examination of seasonal effects on peripheral blood mononuclear cell (**PBMC**) proliferation, where DNA synthesis was consistently decreased in cows calving in the summer

Received June 19, 2015.
Accepted September 28, 2015.
[1]Presented as part of the Animal Health: Understanding and Reducing the Impact of Various Stressors on Immune Responses and Health of Cattle Symposium at the ADSA-ASAS Joint Annual Meeting, Orlando, Florida, July 2015.
[2]Corresponding author: gdahl@ufl.edu

relative to spring (Lacetera et al., 2005); however, IgM secretion from those same PBMC was decreased in spring compared with summer, suggesting an inconsistency of immune response to heat stress. Relative to thermoneutrality, calves under chronic heat stress have decreased IgG production and increased mortality (Kelley et al., 1982), further support for the concept that hyperthermia reduces immune status in old and young cattle. But less is known regarding the effect of in utero heat stress on calf health and performance.

Beyond the negative outcomes in lactating and growing dairy cattle, heat stress during the dry period significantly affects the success of the transition and reduces production efficiency in the next lactation (Tao and Dahl, 2013). Specifically, heat-stressed dry cows have reduced mammary growth, poorer immune status in the transition, and lower subsequent milk yield relative to cows that are cooled for the entire dry period. Further, previous studies and our recent work suggest that the negative effects of heat stress during late pregnancy result in substantial adverse effects on the developing calf before and after birth. Ongoing studies in our laboratory have revealed a series of shifts in immune, metabolic, and performance outcomes that result from a brief period of heat stress in late gestation. These changes in metabolism and health appear to have lifelong effects on the calf, and reduce the productive potential of that calf later in life.

HEAT STRESS EFFECTS ON CALF DEVELOPMENT

It is important to begin with a description of how we manage cows during the dry period to gain perspective on the relatively brief duration and limited level of heat stress that can result in substantial reductions in performance. In all of our controlled studies, multiparous cows are used; that is, they have calved at least once previously and are drying off after a typical lactation. The treatment we impose is simple; cows are milked for a final time, dry cow antibiotic therapy is given along with teat sealant, and the cows shift to a lower energy diet than they were consuming during lactation. Cows are housed in a typical freestall barn with sand beds, and the treatment consists of turning the fans and feedline soakers off in half of the barn, which results in half of the cows having only shade (i.e., heat stressed) relative to the other cows that are actively cooled with fans and soakers under shade (i.e., cooled). Relative to cooled cows, heat-stressed cows typically show a consistent 0.3 to 0.4°C rise in average rectal temperature and an 1.5- to 2-fold increase in respiration rate. These responses appear within a day of treatment and persist for the entire dry period. Thus, the calves developing in the uteri of heat-stressed dams are exposed to increased maternal core temperature and respiration rate during the final 6 to 7 wk of gestation. Despite the relatively modest level and duration of heat stress, significant negative outcomes result in the calf.

In confirmation of earlier work with cows maintained on pasture with shade or without during the dry period (Collier et al., 1982), actively cooling dry cows had profound positive effects on calf birth weight relative to those that were heat stressed (Tao et al., 2012). Thus, even under improved housing conditions in a free-stall environment, in utero heat stress results in a smaller calf at birth relative to cows that are cooled. Of interest, that birth weight difference was maintained at weaning despite the fact that weight gain after birth did not differ between calves heat stressed in utero and those born to cooled dams (Tao et al., 2012). Gestation length is also reduced with heat stress during the dry period, typically by 4 to 5 d, which accounts for a portion of the smaller calf size (Tao and Dahl, 2013). Lighter birth weight likely results primarily from placental insufficiency during late gestation, as heat stress decreases bovine placental weight (Collier et al., 1982) and function, as indicated by reduced placental hormone production including estrone sulfate, placental lactogen, and pregnancy-associated glycoprotein (Collier et al., 1982; Bell et al., 1989; Thompson et al., 2013). But lower maternal intake is also a factor in the lower birth weight of heat-stressed calves. Further evidence that metabolic adaptation to heat stress differs in the dry period versus lactation is found in a recent study by Lamp et al. (2015), where the effects of heat stress were separated from the effects of reduced DMI using a pair-feeding model. Specifically, late gestation cows under heat stress retain the ability to effectively mobilize adipose stores similar to the response of pair-fed normothermic cows (Lamp et al., 2015). Late gestation heat-stressed cows also mobilize protein reserves, all in an effort to support the demand of the developing fetus. Therefore, calves from heat-stressed dams are challenged before birth and must make physiological accommodation in response to higher heat loads, less effective placental support, and reduced maternal nutrient intake.

The mechanism of heat-stress-induced reductions in gestation length has not been determined, but may be related to altered uterine prostaglandin synthesis and subsequent luteolysis. Late gestation heat stress increased postpartum $PGF_{2\alpha}$ concentrations relative to cows that received heat stress abatement, although prepartum $PGF_{2\alpha}$ was not assessed (Lewis et al., 1984). Early pregnancy heat stress increases $PGF_{2\alpha}$ synthesis in cultured uterine endometrium, an effect that may contribute to heat-stress-associated early embryonic losses in vivo (Putney et al., 1988). Because of the