MULTISTATE PROJECT
S-299

"ENHANCING PRODUCTION AND REPRODUCTIVE PERFORMANCE OF HEAT-STRESSED DAIRY CATTLE"

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ENHANCING PRODUCTION AND REPRODUCTIVE PERFORMANCE OF HEAT-STRESSED DAIRY CATTLE

1. PROJECT NUMBER:

2. TITLE: Enhancing production and reproductive performance of heat-stressed dairy cattle.

3. DURATION: October 1, 2000 through September 30, 2005

4. STATEMENT OF THE PROBLEM:

Dairy cattle throughout the Southeast United States and in many subtropical and tropical regions are subject to high ambient temperature and/or high relative humidity for extended periods. In the Southeast US, ambient temperature and relative humidity frequently exceed the critical temperature-humidity index (**THI**) necessary for heat stress 4 to 6 months of each year. Reduced feed intake and elevated body temperatures associated with high THI results in reduced milk yield and reproductive performance, including increased incidence of early embryonic mortality. Genetic selection for milk yield has reduced the dairy cows' ability to regulate body temperature during heat stress depressing fertility even further. Environmental modifications that reduce the negative effects of heat stress have been developed, but a recent analysis of DHI records shows that additional research is needed. Dairy herds in the Southeast US have lower milk yields, greater days open, and higher somatic cell counts than herds in the northern United States. A thorough understanding of dietary, genetic, and environmental modifications to minimize heat stress is necessary to develop practices to reduce the negative effects of heat stress. A more complete understanding of the effects on the cellular and physiological mechanisms by which cows regulate body temperature will allow the development of new strategies for improving reproduction and embryonic survival during heat stress.

5. JUSTIFICATION:

Dairy cattle in the Southern Region are annually exposed to prolonged periods of high humidity and heat which reduce feed intake, milk production and reproductive performance, including an increased incidence of early embryonic mortality. Development and testing of effective management strategies to mitigate these negative effects of heat stress require a coordinated research effort that includes fundamental and applied research in nutrition and reproduction. This can be best accomplished through a regional research project that includes shared resources and scientific expertise. There exist RRF projects in the Southern region focused in whole or in part on enhancing production and reproductive performance of swine (S-145), poultry (S-233), and beef cattle (S-299), but no similar regional effort focused on dairy cattle. There are two related regional projects in the Western region: W-112, Reproductive Performance in Domestic Ruminants; and W-173, Stress Factors of Farm Animals and Their Effects on Performance. One of the expected outcomes of W-173 is to develop more cost effective techniques for reducing heat stress. Neither of these projects addresses nutrition and reproduction interactions in heat-stressed dairy cattle. The intent of the current proposal is to complement, not duplicate, objectives of these two existing projects. Techniques that have been shown to be effective in reducing heat stress will be incorporated into the project and expanded to examine the potential of nutritional modification and hormonal therapy on subsequent production and reproduction. This project proposal fits within the region's priorities as described in the Southern Strategic Research Plan. The emphasis is on interdisciplinary research to enhance efficiency and sustainability of an animal system in our region's unique climate.

6. RELATED CURRENT AND PREVIOUS WORK:

Introduction

High ambient temperature, relative humidity, and radiant energy compromise the ability of the lactating dairy cow to dissipate heat, and coupled with metabolic heat makes it difficult to maintain thermal balance. Elevated body temperatures initiate compensatory and adaptive mechanisms to reestablish homeothermy and homeostasis. Stott (1981) stated that "these readjustments to maintain homeostasis by homoeostatic mechanisms are referred to as adaptations and may be favorable or unfavorable to the economic interests of humans. Nevertheless, they are essential for survival of the animal."

The impact of heat stress on livestock is broad in geographic terms. Dairy cattle across the Southeastern United States and in many subtropical and tropical regions are subject to high ambient temperature and/or high relative humidity for extended periods. In the Southeast, high ambient temperature and relative humidity exceeding the temperature-humidity index (**THI**) associated with heat stress persist for 4 to 6 mo of each year. Several trends will make heat stress an even greater dilemma in the future. Heat stress itself may increase in magnitude if continued global warming occurs (Hulme, 1997). Secondly, the world's population is growing faster in tropical and subtropical regions of the world than in temperate regions (Roush, 1994) and it is reasonable to assume that a greater proportion of the world's food animals will live in hot, humid climates than is the case at present. Finally, changes in the genetics and physiology of food animals for increased production are making these animals less able to regulate body temperature, i.e., less adapted to warm environments. This is especially true for dairy cattle. Selection for milk yield reduces thermoregulatory ability in the face of heat stress (Berman et al., 1985) and magnifies the seasonal depression in fertility caused by heat stress (Al-Katanani et al., 1998). Also, use of bovine somatotropin can increase body temperatures during heat stress (West et al., 1991; Elvinger et al., 1992; Cole and Hansen, 1993). Moreover, adaptation of dairy cattle is reduced when native, genetically-adapted cattle in tropical or semitropical regions are replaced by higher producing, non-adapted breeds.

Beede and Collier (1986) identified three management strategies to minimize the effects of heat stress: physical modification of the environment, genetic development of heat tolerant breeds, and improved nutritional management practices. Nutritional modifications that minimize hyperthermia during heat stress have been developed, but the response to dietary changes may be improved by environmental modifications. Nutrient excesses also contribute to reduced efficiency of energy utilization, potentially adding to stress levels. Therefore, a thorough understanding of dietary modifications on ruminal fermentation and cellular metabolism is necessary to minimize heat stress.

Overall reproductive performance of a herd of dairy cattle can be estimated as the herd pregnancy rate (Hansen and Aréchiga, 1999), which is the product of estrus detection rate (the proportion of cows in estrus that are detected as in estrus) and pregnancy rate per insemination (the proportion of inseminated cows that become pregnant and maintain pregnancy to term). Heat stress is a major limitation to reproductive function of dairy cows because it can have major deleterious effects on both components that determine herd pregnancy rate. Historically, the major approach to reducing effects of heat stress has been to alter the environment so as to reduce the magnitude of forces driving the cow into hyperthermia. Further research in this area is critical because existing housing systems have proven inadequate for eliminating seasonal depression in fertility (Hansen and Aréchiga, 1999). In addition, it may be possible to develop new strategies for improving reproduction during heat stress by regulating the physiology of the cow or the cell biology of the tissues involved in reproduction so as to prevent or counteract actions of heat stress that would otherwise lead to infertility. These approaches will depend upon greater knowledge of the cellular and physiological mechanisms by which heat stress leads to decreased estrous activity and embryonic survival as well as a better understanding of the physiological processes by which cows regulate body temperature.

Metabolic Effects of Heat Stress Which Impact Nutrient Intake

Johnson et al. (1967) termed cows that exhibited sharp increases in body temperature upon exposure to high ambient temperatures as heat intolerant, while those with lesser increases were termed heat tolerant. When ambient temperatures were increased from 18°C to 29°C, the heat intolerant cows exhibited a 1.4°C increase in body temperature and a 4-kg decline in daily milk yield, compared to a .7°C increase and 2 kg decline for heat tolerant cows. Such responses suggest metabolic or physiologic differences among cattle that could be exploited if the mechanisms were identified.

Hormonal changes which occur in response to heat stress may play an integral role in the decline in productivity. Plasma growth hormone concentration and growth hormone secretion rate declined with hot temperatures (35°C) [Mitra et al., 1972]. Growth hormone content in milk of low, medium, and high production groups declined when THI exceeded 70, possibly reflecting suppressed production of growth hormone so that metabolic heat production is reduced (Igono et al, 1988). The thyroid hormones triiodothyronine and thyroxine declined in response to heat stress (Johnson et al., 1988; Magdub et al., 1982), probably in an attempt to reduce metabolic heat production in the cow. However, reduced metabolic rate compromised productive capability as well. Greater plasma content of epinephrine and norepinephrine with high ambient temperatures indicates a stress response (Alvarez and Johnson, 1973), and passage rate in the digestive tract is reduced. Slower passage rate leads to greater gut fill and limited intake, but improved digestibility because of greater residence time in the gut. Mean total tract retention time for cattle was 36.6 and 43.2 h when the ambient temperatures were 18 and 32°C (Warren et al. 1974). Digestibility of DM, ADF, and NDF was 6.7, 11, and 8.1% greater for cattle in the hot environment, partially offsetting the reduced nutrient intake that occurred with reduced DM consumption.

Influence of Heat Stress on the Physiological Responses of the Cow

Reactions of cattle and other homeotherms to moderate climatic changes generally are compensatory and are directed at maintaining or restoring thermal balance (Kibler and Brody, 1953). The numerous physiologic mechanisms for coping with heat stress have been reviewed (Blackshaw and Blackshaw, 1994). These include sweating, more rapid respiratory rate, and greater vasodilation with increased blood flow to the skin surface. Reduced rate of metabolism, decreased DM and nutrient intake, and altered water metabolism all occur in response to heat stress. Unfortunately, responses to heat stress often have negative effects on the physiology of the cow.

Feed DMI starts to decline and maintenance expenditures increase when environmental temperatures exceed 25°C (NRC, 1981). However, THI may describe more precisely the effects of the environment upon the cow's ability to dissipate heat. Milk yield and TDN intake declined slightly when the THI exceeded 72 and declined sharply when an index of 76 was exceeded (Johnson et al., 1963). Milk yield declined when body temperature exceeded 38.9°C, and for each 0.55°C increase in rectal temperature, milk yield and intake of TDN declined 1.8 and 1.4 kg, respectively (Johnson et al., 1963). By moderating the increase in rectal temperature one can expect to improve nutrient intake and subsequent milk yield.

Genetic differences exist for heat tolerance of cattle. Bos indicus breeds are more heat tolerant than Bos taurus breeds because of a greater sweating capacity and lower metabolic rate (Blackshaw and Blackshaw, 1994). Milk yield decline for dairy cows exposed to temperatures of 24 or 34°C and to low (38 to 46%) or high (76 to 80%) relative humidities, was greatest for Holstein, intermediate for Jersey, and least for Brown Swiss cows (Johnson and Vanjonack, 1976). Holstein cows are larger and have less surface area relative to internal body mass and would be expected to dissipate heat less effectively than Jerseys. Brown Swiss are generally acknowledged as being more heat tolerant than Holsteins, but generally produce less milk than Holsteins. Milk temperatures for Holstein and Guernsey cows increased as production increased from low, medium, and high levels during hot weather (Igono et al, 1985), an indication that high yielding cows are more sensitive to hot conditions. Heat production was about 14%

greater for cows subjected to hot (35°C) versus cool (20°C) conditions and was reflected in elevated rectal temperatures (Robinson et al, 1986).

Environmental conditions such as temperature and humidity are interrelated and the combined effects must be considered when determining effects on intake and milk yield. The mean daily temperature had the greatest effect on milk yield and rectal temperature when compared with minimum and maximum temperatures (Kaguga and Sarpong, 1991). Holter et al. (1996) reported that the minimum daily THI was more closely correlated with DMI than maximum THI in Jersey cows. Reductions in DMI commenced when minimum THI exceeded 56 to 57 and continued until THI reached 72. Corresponding maximum THI ranged from 71 to 85; DMI declined 4.4 kg/d, or 22%. Similarly, correlation of DMI with minimum and maximum THI for Holsteins was -.63 and -.62, and maximum THI had a .95 correlation with minimum THI (Holter et al, 1997). During heat stress DMI was reduced 22% for multiparous cows and 6% for primiparous cows, probably reflecting smaller body size for the primiparous cows as well as lower DMI and less metabolic heat production.

Effects of weather conditions on intake and milk yield are most likely mediated through changes in body temperature. Across early, middle, and late stages of lactation, 9% of the variation for milk yield, 13% for milk fat, 5% for feed intake, and 65% for rectal temperature were attributable to weather conditions (Maust et al., 1972). However, weather conditions during the 2 to 3 d preceding the day of temperature measurement were most closely associated with milk yield and composition and comparisons using a single day of temperature measurement could be misleading. In addition, DMI and milk yield for cows in mid-lactation were reduced most by hot weather conditions (Maust et al., 1972), possibly because early lactation cows rely on body stores for a portion of the nutrients for production while late lactation cows consume fewer total nutrients. Araki et al. (1984) reported that lactating cows were more sensitive to the effects of heat than were dry cows, consistent with the greater metabolic heat production for the former.

Effects of Cooling on Intake and Milk Yield

Environmental modification is necessary to maintain productivity by the lactating dairy cow. Maintenance of DMI is critical to production and minimizing the increase in body temperature during hot conditions will translate directly into greater feed consumption. The most obvious environmental modification is the use of shade. Shading in a hot, humid climate reduced rectal temperature by 2 to 4.1%, respiratory rate by 29 to 60%, improved DMI by 6.8 to 23.2%, and improved milk yield by 9.4 to 22.7% compared with unshaded cows (Mallonee et al., 1985; Schneider et al., 1986; Schneider et al., 1984). Cows shaded during the dry period yielded 4.5 and 13.6% more milk at 100 and 305 d postpartum and delivered calves weighing 3.1 kg more compared with cows receiving no shade while dry, even though cows were handled similarly postpartum (Collier et al., 1982a). This suggests that cows may have had greater energy reserves entering the lactation. Additional benefits from cooling cattle with fans and sprinklers can be derived in hot or hot and humid climates. Reports as early as the 1940's (Seath and Miller, 1948) demonstrated that, used independently, fans and sprinklers reduced rectal temperature and respiratory rate, but when used together the effects were synergistic. Sprinklers used in combination with fans also reduced diurnal variation of body temperature. Much of the response to cooling is a direct result of increased DMI. Bucklin et al. (1991) summarized studies from Florida, Kentucky, and Missouri and reported improvements in DMI of 7.1, 9.2, and 7.1% and milk yields of 11.6, 15.8, and 8.6%, for the three states, respectively. Rectal temperatures for the Kentucky and Missouri cows were reduced by 0.5 and 0.4°C. High producing cows in early lactation were most sensitive to heat stress, and milk yield declined significantly when rectal temperatures exceeded 39°C for more than 16 h (Igono and Johnson, 1990). Because genetic potential for milk yield has improved and the predominant dairy breed has become Holstein, the impact of heat stress on production has increased. Physiologic improvements with cooling included greater circulating growth hormone concentrations and lower prolactin concentrations (Igono et al., 1987). Accompanying these physiologic changes were a 2.3 and 2.0 kg/d increase in DMI and milk yield. Similar to shade research with dry cows (Collier et al., 1982b), cooling with fans and

sprinklers during the dry period increased mean 150 d milk yield by 3.5 kg/d, and the effect was greater with increasing age (Wolfenson et al., 1988). Milk yield was 0.9, 2.5, and 7.3 kg per day greater in lactation 2, 3, and 4+ for cows which were cooled during the dry period. It is apparent that with increasing production, body size and maturity, cows become more sensitive to the effects of environmental stress. Supplemental cooling of dry cows increases milk yield during the first 120 d of lactation and the increase was greater as daily temperatures increased (Armstrong, 1994). Effective nutritional programs need to be implemented with some environmental modification if high production is to be maintained.

Nutrition Modifications to Reduce Heat Stress

The last three weeks of the dry period and the first three weeks of lactation (termed the transition period) is a time of extreme stress for the lactating dairy cow. Normal intake during the dry period is in the range of 1.8 to 2% of body weight, but declines by up to 25 to 30% during the final week prepartum (Bertics et al., 1992). Current nutritional guidelines (NRC, 1989) do not consider this intake decline. Reasons for reduced prepartum DMI include hormonal fluctuations, repositioning of the fetus, and increasing nonesterified fatty acid (NEFA) content in the blood. In a research study where 89 Holstein cows were monitored prepartum through 30 days postpartum, feed intake during the final 27 days prepartum declined 33% for normal cows, but declined 50% for cows which exhibited one or more early postpartum disorders (Zamet et al., 1979). Fifty-one percent of the cows experienced one or more early postpartum disorders. Low feed intake prepartum is highly related to the incidence of metabolic disorders at and following parturation. Hormonal changes, especially decreased blood progesterone and increased blood estrogen concentrations (Grummer, 1995) are believed to be a major factor contributing to reduced dry matter intake (DMI) at calving. Unfortunately, nutrient demands for the placenta and fetal calf are at their highest level (Bell, 1995), while DMI is declining.

Cows at transition are already stressed due to a number of metabolic and physical factors. Heat stress further depresses intake and reduces the probability of a successful transition. Often the dry cow has the least shading and usually no supplemental cooling. However it is becoming increasingly apparent that cooling with fans, sprinklers, or misters both pre- and postpartum may return large benefits in the form of healthier cows with less condition loss due to the greater feed intake possible with supplemental cooling.

Extensive research in the areas of heat abatement, nutritional management, and the interaction of these factors has been conducted with the lactating dairy cow. To compensate for lower DM intake nutrient density is frequently increased to maintain milk yield, but the results have not always been favorable. Feeding protein, especially rumen degradable protein, in excess of NRC (1981) recommendations has been shown to decreases DM intake and milk yield in heat stressed cows (Huber et al., 1994). The decrease in milk yield is greater than the reduction in energy intake because additional energy is required to convert the excess protein into urea for excretion. Improving protein quality supports higher levels of milk yield in cows under heat stress (Huber et al., 1994). Cows fed diets containing higher concentrations of lysine (from a combination of soybean, fish, and blood meals) produced 11% more milk under heat stress conditions (Chen et al. 1993). The results of including supplemental fats which have a low heat increment have been variable. Milk yield increased (Chan et al., 1992; Huber et al., 1993) or remained the same (Chan et al., 1993) suggesting that the response to supplemental fat is less for heat stress cows than unstressed cows. Addition of fungal cultures has been reported to increase milk yield by approximately 4% and decrease rectal temperatures and respiratory rates (Huber, 1994), but the results have not been consistent.

There is a dearth of heat stress research investigating these areas for the transition cow. The research is difficult to conduct, takes large cow numbers, and is subject to extreme variation between animal units. It is necessary to conduct the research in several locations to generate sufficient numbers of cows and environments to draw sound conclusions. Research in the areas of cooling benefits, enhanced dietary

nutrient density and quality, physiologic responses to dietary changes (including metabolic heat production and acid-base chemistry), and the subsequent effects upon intake, milk yield, and reproductive performance is critical to addressing this high risk, and high cost, time for the dairy cow under heat stress that is typical in the Southeast and Midsouth.

Influence of Heat Stress on the Expression and Detection of Estrus

Heat stress reduces the length (Monty and Wolff, 1974; Abilay et al., 1975) and intensity (Gangwar et al., 1965) of estrus. In Virginia, Nebel et al. (1997) reported that Holsteins in estrus during summer had 4.5 mounts per estrus versus 8.6 for those in winter. Changes in estrous activity caused by heat stress reduce the likelihood that estrus will be detected by dairy personnel. The percentage of undetected estrous periods on a commercial dairy in Florida were estimated at 76-82% during June to September versus 44-65% during October to May (Thatcher and Collier, 1986).

The causes of reduced expression during heat stress have not been completely defined. Some effects of heat stress may involve ACTH. Heat stress can cause increased cortisol secretion (Roman-Ponce et al., 1981; Wise et al., 1988a; Elvinger et al., 1992) and ACTH has been reported to block estradiol-induced sexual behavior (Hein and Allrich, 1992). In certain experiments, though, heat-stress induced elevations in circulating cortisol concentrations were transitory (Christison and Johnson, 1972; Miller and Alliston, 1974; Elvinger et al., 1992). In others, there was no increase in cortisol concentrations associated with heat stress (Wise et al., 1988b, West et al., 1991) or heat stress depressed cortisol concentrations (Abilay et al., 1975). Accordingly, effects of heat stress on estrus behavior likely includes actions independent of the pituitary-adrenal axis. Some reports indicate that heat stress causes a reduction in peripheral concentrations of estradiol-17β at estrus (Gwazdauskas et al., 1981; Wilson et al., 1998a) although this effect has not always been observed (Rosenberg et al., 1982). It is possible that the major reason that heat stress reduces the expression of estrus is because of the physical lethargy produced by heat stress. Reduced physical activity is itself probably an adaptive response that limits heat production.

Heat-Stress Induced Embryonic Mortality

Elimination of problems of estrous detection caused by heat stress is not sufficient to restore herd pregnancy rates to a level seen in cool weather because of the severe consequences of heat stress for embryonic development. Depression in pregnancy rate per insemination in warm or hot periods of the year has been well-documented in many regions (Stott et al., 1962; Monty and Wolff, 1974; Rosenberg et al., 1977; Cavestany et al., 1985; King et al., 1988; Du Preez et al., 1991; Ryan et al., 1993). There are two types of evidence that this depression is caused primarily by heat stress and not other environmental variables. Specifically, experimental application of heat stress can reduce fertility and embryonic survival (Dunlap and Vincent, 1971; Putney et al., 1988a, 1989b; Ealy et al., 1993) and cooling cows during the summer can improve pregnancy rate per insemination (Stott et al., 1972; Thatcher et al., 1974; Roman-Ponce et al., 1977; Wolfenson et al., 1988).

The mechanism by which heat stress causes decreased fertility is probably multifactorial and may vary depending upon the magnitude of heat stress. When rectal temperatures in the hot season were low (39.0°C) because of environmental cooling, most early embryonic mortality associated with the hot season occurred between day 6 and 14 of pregnancy (Ryan et al., 1993). In contrast, Putney et al. (1988a) observed that experimental application of a heat stress that caused rectal temperatures to rise to 41.1°C caused a large reduction in embryonic development at an earlier time (day 7 after estrus). Since the process by which heat stress leads to embryonic loss may be different following mild versus severe heat stress, it follows that the optimal strategy for improving fertility during heat stress may depend upon climatic conditions. Elucidation of the physiological and biochemical pathways through which heat stress causes disruption of embryonic survival are therefore important for development of techniques to intercept those pathways and improve fertility.

Causes of Embryonic Loss Due to Heat Stress

The peri-ovulatory period and first few days of early pregnancy are very susceptible to disruption by heat stress. Exposure of superovulated heifers to heat stress for 10 h beginning at the onset of estrus had no effect on fertilization rate but reduced the proportion of normal embryos recovered on day 7 after estrus (Putney et al., 1989). The experiment was designed to avoid effects of heat stress on spermatozoa; heifers were not inseminated until body temperatures had returned to normal. Similarly, heat stress at day 1 (Ealy et al., 1993) or day 1-3 postbreeding (Dunlap and Vincent, 1971) reduced embryonic survival. Heat stress of superovulated cows at day 3, 5 or 7 after estrus did not affect embryonic development or survival at day 8 (Ealy et al., 1993). This result indicates, that as for other species (Dutt, 1964; Tompkins et al., 1967; Wolfenson and Blum, 1988), effects of heat stress on embryonic survival decrease as embryos proceed through development.

Disruption of early embryonic development results from actions on the embryo itself or on the oviductal or uterine environment in which the embryo resides. Alliston and Ulberg (1961) used a reciprocal embryo transfer scheme in ewes to show that both the embryo and reproductive tract are compromised by heat stress, with greater effects being exerted on the embryo itself. Exposure of cultured bovine embryos to heat shock (a term used herein to mean exposure of cultured cells to any elevation in temperature above normal body temperature) can compromise subsequent development (Ealy et al., 1995; Edwards and Hansen, 1997; Sugiyama, 1998). For the experiments of Sugiyama (1998), the temperatures that embryos were exposed to varied in a diurnal manner to match variations of rectal temperatures of dairy cows in the summer in Queensland. Thus, the elevated temperature that an embryo is exposed to when its dam becomes hyperthermic could lead directly to embryonic death. In addition, heat shock of maturing oocytes in culture can lead to reduced protein synthesis, fertilization rate and subsequent developmental competence (Lenz et al., 1983; Edwards and Hansen, 1996; Sugiyama, 1998). However, development to the blastocyst stage was less damaged by heat shock when applied to oocytes than when applied to 2-cell embryos (Edwards and Hansen, 1997).

The fact that the deleterious effects of maternal heat stress decline as pregnancy proceeds (Ealy et al., 1993) may reflect acquisition of thermal resistance by the preimplantation embryo as it progresses from the zygote to blastocyst stage. Heat shock caused a greater reduction in the proportion of cultured 2-cell embryos that developed to the blastocyst stage than heat shock of 4-8 cell embryos; morulae were unaffected by heat shock (Edwards and Hansen, 1997). Thus, susceptibility of embryos to heat shock in vitro appears to parallel the situation in vivo. Caution must be exercised when interpreting these data because stage differences in resistance of mouse embryos to heat shock was not apparent for all developmental endpoints (Aréchiga and Hansen, 1998). In particular, 2-cell embryos were more affected by heat shock than 4-cell embryos or morulae when the endpoint was development to blastocyst but not when the endpoint was development to the hatched blastocyst stage.

If, as apparent, bovine embryos become more resistant to heat shock as development proceeds, the mechanism responsible is not understood. Once, it was hypothesized that early embryos (<8-16 cell stage) would be more susceptible to heat shock because these embryos are transcriptionally quiescent and unable to produce protective molecules such as heat shock protein 70 (**HSP70**) in response to heat shock. It is now known that heat shock can induce synthesis of HSP70 as early as the 2-cell stage (Edwards and Hansen, 1996). Heat-induced HSP70 synthesis is the result of new transcription (Chandolia et al., 1999).

Though embryos are most sensitive to elevated temperature early in development, heat stress at more advanced times can reduce development. For example, heat stress compromised embryonic development when applied from days 8-16 of pregnancy (Biggers et al., 1987). In addition, Ryan et al. (1993) observed differences in embryonic loss of superovulated cows between cool and hot periods of year at day 13-14 of pregnancy but not at day 6-7. Furthermore, exposure of bovine embryos flushed from

superovulated heifers at day 6 or 7 of pregnancy to heat shock in culture reduced cell number (Sugiyama, 1998).

Uterine and oviductal tissues may be compromised during heat stress for several reasons. First, heat stress leads to a redistribution of blood flow from the visceral organs to the periphery; the resultant decreased perfusion of nutrients and hormones could compromise endometrial and oviductal function. Roman-Ponce et al. (1978) observed that the increase in uterine blood flow caused by injection of estradiol-17β was reduced in cows not exposed to shade in summer as compared to those receiving shade. Secretion of the hormones regulating reproductive tract function may also be altered by heat stress. Recent experiments suggest that heat stress can cause an increase in peripheral concentrations of estradiol-17β between days 1-4 of the estrous cycle (Wolfenson et al., 1995) and a reduction from days 4-8 (Wolfenson et al., 1995) and 11-21 of the cycle (Wilson et al., 1998ab). These results must be compared with earlier reports of no effect of heat stress (Roman-Ponce et al., 1981; Wise et al., 1988ab). Similarly, heat stress has been reported to increase (Abilay et al., 1975; Roman-Ponce et al., 1981; Trout et al., 1998), decrease (Rosenberg et al., 1982; Younas et al., 1993) or have no effect (Wise et al., 1988a; Wolfenson et al., 1995; Wilson et al., 1998ab) on peripheral concentrations of progesterone during the luteal phase of the estrous cycle.

Some of the variation in hormonal responses to heat stress probably reflects the fact that ovarian steroid concentrations are dependent not only on rate of secretion from ovarian tissue but also on rate of vascular perfusion of the ovary, possible adrenal release (at least for progesterone), metabolism in the liver and other organs, and the degree of hemodilution or hemoconcentration. The extent to which heat stress affects these other physiological characteristics could lead to variable changes in steroid hormone concentrations in peripheral blood. For example, heat stress can cause either dilution, concentration, or no effect on blood plasma volume (Richards, 1985; McGuire et al., 1989; Johnson et al., 1991; Elvinger et al., 1992) and the nature of effect of heat stress on blood volume will affect steroid hormone concentrations in blood.

Actions of steroid hormones on reproductive tract tissue could conceivably be reduced during heat stress as a result of increased synthesis of heat shock proteins. Heat shock can lead to increased synthesis of HSP70 and heat shock protein 90 in endometrium (Malayer et al., 1988; Malayer and Hansen, 1990). Both these proteins are part of the complex of proteins associated with the progesterone and estrogen receptor (Johnson et al., 1996; Nair et al., 1996; Sabbah et al., 1996). Increased synthesis of heat shock proteins might alter assembly, transport or binding activities of steroid receptors. Indeed, heat shock blocked estradiol induced transcription of the vitellogenin gene in Xenopus hepatocytes (Wolffe et al., 1984).

Direct action of elevated maternal temperature on the function of reproductive tract tissues remains a possibility although exposure of cultured endometrial explants to 43°C caused relatively small changes in protein and DNA synthesis (Putney et al., 1988b; Malayer et al., 1988; Malayer and Hansen, 1990). However, heat shocks of 42 and 43°C increased output of prostaglandins by cultured endometrium collected at day 17 of the estrous cycle (Putney et al., 1988b; Malayer et al., 1990) and exposure of cultured day 17 conceptuses to 43°C reduced secretion of interferon-τ (Putney et al., 1988b). Furthermore, heat stress on day 17 of pregnancy increased uterine production of prostaglandin F2α in response to oxytocin (Wolfenson et al., 1993).

One area of active investigation is possible effect of heat stress on ovarian follicular function. The follicle destined to ovulate emerges as an antral follicle about 40 d before ovulation (Lussier et al., 1987). Therefore, heat stress during the period of follicular growth has the potential to compromise the oocyte,

either because of direct actions of elevated temperature on the oocyte or because of alterations in follicular function that would compromise oocyte quality.

Heat stress can alter follicular dynamics by reducing follicular dominance. Badinga et al. (1993) observed that heat stress beginning on the day of ovulation reduced the diameter and volume of the dominant follicle on day 8 of the estrous cycle. Heat stress from day 3-5 of the estrous cycle increased androstenedione and reduced estradiol-17β concentrations in follicular fluid of the dominant follicle collected at day 7 (Wolfenson et al., 1997). In another experiment (Wolfenson et al., 1995), heat stress beginning at day 1 of the estrous cycle caused an increase in number of follicles > 10 mm in diameter, earlier emergence of the dominant follicle of the second follicular wave and tended to reduce plasma concentrations of inhibin.

Initiation of heat stress at day 11 of the estrous cycle caused more estrous cycles characterized by three follicular waves versus two follicular waves, reduced estradiol-17β concentrations in blood and caused estrous cycle extension (Wilson et al., 1998ab). The increase in estrous cycle length was ascribed to reduced estrogenic support for the uterine luteolytic mechanism. In another study, however, there was no extension of estrous cycle length caused by initiation of heat stress at day 11 (Trout et al., 1998).

Changes in follicular function in response to heat stress could be the result of endocrine changes in LH secretion (Wise et al., 1988a; Gilad et al., 1993) or changes in metabolic hormones that affect ovarian function. Heat stress tended to reduce concentrations of somatotropin (Igono et al., 1987; McGuire et al., 1991) but did not affect IGF-1 concentrations (McGuire et al., 1991). Elevated body temperature may also directly affect follicular function. Culture of thecal cells at 40.5°C reduced androstenedione production from cultured thecal cells but generally had no effect on estradiol-17β production from cultured granulosa cells (Wolfenson et al., 1997). Likewise, heat shock reduced LH-stimulated progesterone secretion from rat luteal cells in a process that could be partially blocked by antisense oligonucleotide to HSP70 (Khanna et al., 1995).

It is not known whether effects of heat stress on follicular development are sufficient to alter subsequent fertility and, if so, to what extent altered follicular development contributes to the summer decline in fertility. Recently, an analysis of Dairy Herd Improvement Association data from South Georgia and North Florida revealed results consistent with the possibility for fertility-altering consequences of heat stress before breeding (Al-Katanani et al., 1998). A subset of data was created that included only cows exposed to relatively cool temperatures (average dry bulb temperatures of < 25°C) from day 9 before breeding until day 1 before breeding. For this group of cows, those experiencing average air temperatures > 20°C on day -10 before breeding had lower 90-day non-return rates than those cows experiencing air temperatures < 20°C. While these results suggest that pre-ovulatory effects of heat stress may be important for subsequent fertility, the definitive experiment in which heat stress is experimentally applied at various times before breeding remains to be performed. In sheep, such an experiment revealed that heat stress on day 12 of the estrous cycle before breeding reduced fertilization rate and lambing rate (Dutt, 1964).

Influence of Heat Stress on Male Fertility

Many mammals, including the bovine, have evolved so that male gametogeneisis is unable to occur at body core temperatures. Artificially raising the temperature of the testis in the bull for as little as one hour causes a disruption in spermatogenesis (Austin et al., 1961). The stage of spermatogenesis that is most susceptible to elevated temperature is the primary spermatocyte although damage to B spermatogonia can occur in bulls and prolonged exposure to heat can damage dividing spermatocytes and spermatids (Thatcher and Collier, 1982). Scrotal mammals have evolved an intricate anatomical and physiological system for local thermoregulation of the testis that involves placement of the testis outside

the body cavity, exchange of heat between the body core and testis through countercurrent mechanisms in the pampiniform plexus and regulation of heat loss to the surrounding air via muscular control of the placement of the testis relative to the body and the surface area of the scrotum and via sweat. As a result, testicular temperature is about 4 C below core body temperature. Spermatogenesis is disrupted when either the thermoregulatory system that maintains testicular temperature is disrupted (Austin et al., 1961) or there is an elevation in body temperature of the blood reaching the testis caused by fever or heat stress (Johnston et al., 1963; Skinner and Louw, 1966; Meyerhoeffer et al., 1985).

Alterations in sperm production caused by heat stress commonly include decreased sperm numbers, decreased sperm motility and increased numbers of abnormal sperm (Meyerhoeffer et al., 1985). There is a delay of about two weeks between heat stress and the onset of the first alterations in sperm output (Johnston et al., 1963; Meyerhoeffer et al., 1985) because the primary spermatocyte is the most susceptible germ cell. The process of spermatogenesis takes approximately 54 days in the bull (Setchell et al, 1994) and effects of heat stress on sperm output persist for 8 weeks after the end of heat stress (Meyerhoeffer et al., 1985).

7. OBJECTIVES:

7.1 To further elucidate direct and indirect effects of chronic heat stress in a hot, humid climate on nutrient intake and metabolism, endocrine status, and reproductive and lactational performance of dairy cattle.

7.2 To develop physiological, nutritional and environmental strategies to enhance milk production, reproductive performance, profitability and heat tolerance of dairy cattle in a hot, humid climate.

8. PROCEDURES:

8.1 Objective 1.To further elucidate direct and indirect effects of chronic heat stress in a hot, humid climate on nutrient intake and metabolism, endocrine status, and reproductive and lactational performance of dairy cattle.

8.1.A Effect of environmental stressors on body temperature, nutrient intake, and performance.

The effects of the ambient environment (including temperature, relative humidity, radiant energy, and air movement) and heat abatement methods on body temperature and subsequent intake, milk yield and composition and body weight changes will be determined in the high producing dairy during cool and hot weather (**GA, LA, MO, NC**). Each location will use similar management practices, heat abatement techniques and research protocols in the replicated trial. Data from each location will be compiled (**GA**) and analyzed. The effects of environmental conditions on body temperature will be compared to determine at what point heat stress occurs, which environmental variables or combinations of variables have the greatest impact on cow performance and if there are delayed (or lag) effects of environmental conditions. Lactation performance will be evaluated using daily milk yield, milk composition analysis, and changes in body weight and condition. Reproductive performance will be evaluated by days to first breeding, services per conception, and pregnancy rate. Data for economic analysis will be collected using standardized protocols and coordinated by NC.

The effects of environmental stressors on nutrient metabolism during cool and hot weather will be studied in a series of trials (**FL, GA, MO**). Trials will examine the effects of dietary fiber content, protein amount and form, lipid content, and dietary cation-anion level. Researchers will collaborate to implement shared research protocols and diets to address specific nutrients. Data from replicated trials will be compiled and analyzed by researchers at MO who will coordinate these trials. Ruminally and duodenally cannulated cows will be used to measure changes in ruminal fermentation and nutrient digestibility. Selected metabolites in blood related to nutrient metabolism and stress will be monitored. Intake, milk yield, and milk composition will be evaluated in additional intact cows.

8.1.B Effects of climate pre- and postpartum on intake and performance.

Supplemental cooling has been shown to reduce the negative effects of heat stress on intake, performance and health of lactating cows, but few data are available to determine the effect of heat abatement techniques in the transition cow. The negative effects of heat stress and the potential of supplemental cooling on intake and subsequent performance of the transition dairy cow will be determined in replicated studies (**GA, MO, NC**) coordinated by GA. A standardized research protocol will be used at each location to allow data to be compiled for analysis. Cows will be provided shade plus no supplemental cooling or supplemental cooling provided by fans and sprinklers or high pressure misters at each location. Determination of body temperature and climatic variables will be made together with daily feed intake, weekly body weight, and periodic body condition score during pre- and postpartum periods. At calving, all cows will be handled similarly. Nutrient intake, milk yield and composition, body weight and condition score, and concentrations of critical metabolites (NEFA, BUN, and minerals) will be measured and incidence of metabolic disorders will be recorded. Effects of climatic variables on prepartum intake, as well as the effects of heat abatement procedures will be determined. Additional data on the effects of environment on the transition cow will be collected for cows calving in spring, summer and fall (GA, LA, NC, FL). Nutrient intake, body weight and condition score, body temperature, and blood metabolites (insulin, NEFA, beta-hydroxybutyrate, BUN, and progesterone) will be determined prepartum along with nutrient intake, milk yield and composition, health data, select endocrine concentrations, and reproductive performance postpartum using a shared research protocol. Data from each location will be compiled for analysis coordinated by GA.

8.1.C Effect of environmental stressors on reproductive performance.

Many dairy producers have adopted some form of environmental modification for their lactating cows. Therefore, the effects of environmental stressors on reproductive performance will be evaluated in concert with environmental modifications as described in Section 8.2.C. Additional information will be collected as described in other sections related to effects of nutritional modification.

Testicular function of Holstein and Senepol (a heat tolerant breed used as a positive control) will be evaluated on the basis of sperm production and endocrine response (**USVI, NC, TN**). Bulls will be provided either supplemental cooling 70 days before the breeding season or maintained in their normal environment without cooling. Environmental data will be obtained from the USVI weather station and from black bulb thermometers located in the sun and under supplemental cooling facilities at each farm. Semen will be collected by electroejaculation and evaluated for motility, viability, morphology and concentration at 28-d intervals (Godfrey et al., 1990) To evaluate LH and testosterone secretion, bulls will be given a GnRH challenge at 28-d intervals. Pregnancy rates of cows bred to each bull will be determined using rectal palpation and ultrasound. Researchers at USVI will coordinate this project and be responsible for data and sample collection. Researchers at NC and TN will collaborate in sample and data analysis.

8.1.D Effect of genetic selection on heat tolerance or intolerance.

Potential genetic factors related to heat stress tolerance will be studied (**GA, MO**). The relationship between test day yield and black globe and temperature-humidity index (BGTHI, THI) will be established for several climatological regions in the South and Midwest using records from the Dairy Records Management Service. Subsequently, genetic parameters of heat stress for production and reproduction traits will be calculated for each of the regions separately. Predicted transmitting abilities (PTA) of heat stress will be calculated for active sires available from commercial studs. Sires will be ranked based on best profitability under different levels of heat stress. Researchers will collaborate to implement a shared research protocol.

Effects of coat color on fertility of dairy cows and bulls will be evaluated (**USVI**). Digital photos will be taken of each animal and percentage of black and white hair coat will be determined using image analysis software or a manual method (Godfrey and Hansen, 1996). Analysis will be conducted to determine if there is a relationship between coat color and fertility - heat stress - scrotal thermography. Data will be collected for at least three years on three commercial dairies near St Croix. Environmental data will be obtained from the USVI weather station and from black bulb thermometers located at each farm.

8.1.E Effect of heat stress and production level on endocrine profiles.
Endocrine profiles (growth hormone, IGF-1) will be evaluated (**AL**) in both high and low producing cows at various stages of lactation during periods of heat stress. Alterations in the secretion pattern of GH, the temporal relationship of plasma GH spikes to plasma and hepatic IGF-1 concentrations, and hepatic IGF-1 mRNA content will be measured in cows both during thermoneutral and heat-stress periods as a means of evaluating the effects of heat stress on energy partitioning.

8.2 Objective 2. To develop physiological, nutritional and environmental strategies to enhance milk production, reproductive performance, profitability and heat tolerance of dairy cattle in a hot, humid climate.

8.2.A Effect of nutritional strategies to enhance intake and efficiency of nutrient utilization and improve reproduction in hot, humid climates.

Reduced DMI and altered physiologic state of dairy cows affect nutrient requirements and necessitate altering the density of dietary nutrients during heat stress. During hot weather, dietary crude protein content is usually increased; however, excessive dietary protein must be converted to urea by the liver and excreted in the urine at an energetic cost to the cow. Models which consider the biological state of the cow provide extensive guidelines regarding protein requirements, but additional research is needed to define protein and amino acid requirements during heat stress and determine the energetic benefits to providing diets with optimum protein quantity and quality. Replicated studies will be conducted during cool and hot weather using diets that vary in protein content and quality (**AL, GA, LA, SC, NC**). Preliminary studies using cows with ruminal and duodenal cannulae will be used to compared diets for delivery of microbial protein and amino acids to the small intestine (**GA**). Production studies using a shared research protocol will be used to compare protein treatment effects on DMI, milk yield, efficiency of milk yield, and digestibility of dietary nutrients. Days to first service, services per conception, and pregnancy rate will be monitored to evaluate reproductive efficiency. Research will be coordinated by NC and data from each location will be compiled for analysis.

Dietary cation-anion difference (DCAD) has been shown to directly impact DMI of cows and support greater use of both potassium and sodium. Studies will be conducted to examine the use of very high

DCAD diets, using combinations of K and Na to vary DCAD (**GA, LA, NC**). Researchers will use a standard experimental design for replicated trials. Response variables include DMI, milk yield, change in body weight and condition score, efficiency of production, days to first service, services per conception, and pregnancy rate. Data from each location will be compiled by GA and results used to determine the effect of DCAD under heat stress conditions.

The potential for improving lactational and reproductive performance of dairy cows by supplementation of antioxidants such as vitamin E and selenium will be examined (**FL**). Approximately 300 cows calving from March through June will be treated with three injections of either 500 mg vitamin E and 50 mg Se or a placebo at 21 days prepartum and at 30 and 80 days postpartum. Blood will be collected from a select number (10/treatment) of cows for analysis of vitamin E and Se content. The incidence of retained placentas and uterine infections developing after freshening will be recorded. Cows will be bred at estrus after the voluntary waiting period (90 days in milk) and pregnancy confirmed by rectal palpation at 45 days after breeding.

Dietary ingredients (enzymes, direct fed microbials, yeast cultures, etc.) designed to improve dry matter digestibility have shown some potential for improving lactational performance. Studies will be conducted to determine if these additives will improve performance during periods of heat stress (**AL, LA**). Effects of these ingredients will be determined by measuring DMI, milk yield, change in body condition score and weight, efficiency of production and nutrient digestibility. Research will be coordinated by LA. When appropriate, shared protocols will be used to provide replication of treatments and data compiled for analysis.

8.2.B Effect of hormonal supplements to enhance reproductive performance in hot, humid climates.

The potential for increasing serum progesterone concentrations to reduce embryo mortality will be tested by treatments designed to increase serum progesterone in the cycle before and after breeding (**NC, TN, MS**). Cows at each location in the replicated experiment will be treated with GnRH on day 5 after a synchronized ovulation (Ovsynch program) and then bred after a PGF induced estrus or a second Ovsynch treatment. Pregnancy determinations will be made by ultrasonography on days 25-30 post-insemination and repeated at 45-50 days. The GnRH treatment should increase serum progesterone. Higher progesterone concentrations in the cycle before breeding have been reported to increase fertility in one study with beef cows. Use of two Ovsynch procedures will help determine if cows synchronized by Ovsynch and treated but not bred can be resynchronized with success using the Ovsynch program to avoid estrus detection. Also serum progesterone concentrations can be increased through the use of progesterone using a controlled intravaginal drug release (CIDR) delivery system. The CIDR's will be inserted beginning day 3 after insemination to determine if this will enhance pregnancy rates (**NC**). Milk production will be monitored during studies to determine any effect of treatments. Data from each location will be compiled by NC for analysis.

Effects of heat stress and hormonal supplementation on embryonic development will be determined in studies outlined below. Results of each trial will be used to identify subsequent areas of research and potential means of improving embryonic survival.

Embryos resulting from in vitro fertilization will be exposed to heat shock before freezing (**FL**) to determine whether heat shock before freezing improves subsequent pregnancy rate when embryos are transferred under commercial conditions. Each week, all open cows in a large commercial dairy that have completed the voluntary waiting period for breeding will be subjected to an estrous synchronization scheme (day 0, gonadotropin releasing hormone, day 7 prostaglandin-F2α). Cows detected in estrus will be randomly assigned to one of three groups. One group (negative control) will be bred via AI. Another group will receive at day 8 after estrus an embryo that has been frozen according to standard conditions.

A third group will receive an embryo that had been exposed to 41°C for 90 min followed by 38.5°C for 3 h before freezing. Embryos will be produced at Wisconsin and air-shipped to Florida. It is planned to include 100-150 cows in each group. Pregnancy rate will be determined by rectal palpation at day 45 of pregnancy.

The potential of using IVF-derived embryos identified as having survived freezing and being capable of establishing a pregnancy based on oxygen consumption will be tested (**FL**). Cow selection and hormonal treatment will be as described above. Cows will be randomly selected to receive an embryo frozen and thawed under normal conditions or an embryo selected for transfer based on oxygen consumption. To select embryos based on oxygen consumption, a group of embryos three times larger than required for that day will be thawed and oxygen consumption measured for 40 min after placing the embryos in individual sealed chambers of the multichannel respirometer developed by Overstrom (1996). Those embryos ranking in the top third for oxygen consumption will be transferred into recipients. It is planned to include 100-150 cows in each group. Pregnancy rate will be determined by rectal palpation at day 45 of pregnancy.

Differences in blastocysts produced by IVF compared with those recovered from superovulated cows will be evaluated (**FL, NC, TN**) and coordinated by FL. Apoptosis will be determined by the TUNEL assay, heat shock protein 70 mRNA by RT-PCR and antioxidant systems determined by Western blotting (superoxide dismutase, glutathione peroxidase) or by direct enzymatic measurement (glutathione). If differences are observed in endpoints between embryos derived by IVF versus those from superovulation, the potential of culture conditions to modify the endpoints will be used to test whether culture conditions that most closely mimic the in vivo situation with respect to level of apoptosis, etc. allow for increased embryo survival to freezing as determined by ability to hatch in culture following freezing and then by determining embryo survival after transfer to recipients.

8.2.C Effect of environmental modification strategies on lactational and reproductive performance in hot, humid climates.

Environmental modifications to dry lots, free-stall barns and milking parlors in a combined effort will be investigated to examine how such modifications may impact the production performance of dairy cattle (**MS, NC**). Shared research protocols will be used so data can be compiled for analysis by NC. Environmental modifications will include either high pressure misters or sprinklers and fans. Seasonal influences of chronic heat stress on the reproductive performance of dairy cattle will be assessed using production data from station herd and DHIA records relative to station location. Such historical data will provide the benchmarks from which to examine the impact of future environmental modifications at these facilities on reproductive function. As environmental modifications are installed and investigated, multiple procedures will be employed to examine reproductive processes and lactational performance of the dairy cow and first-calf heifer. It is proposed to examine the effects of cooling on cows during the transition phase beginning prepartum to determine the effect on subsequent breedings when temperatures may be reduced in order to assess possible lingering effects on reproduction. (**NC**)

Blood samples will be collected at varying intervals to monitor changes in the reproductive cycle and competency of ovarian function in heat stress and cooled scenarios. Hormonal assays for progesterone, estradiol, luteinizing hormone and other hormonal indicators will be utilized for assessing luteal and follicular function. To monitor ovarian activity directly, transrectal ultrasonography will be employed to visually appraise ovarian follicular dynamics and measure the size of ovarian structures (CLs or follicles) under differing environmental, seasonal or facility modification strategy. Breeding trials using both traditional estrus detection methods (visual, Kmar, etc.) and methods for the synchronization of estrus

(Ovsynch, PGF2α, etc.) will be conducted to examine the influence of chronic heat stress and cooling on synchronization protocol success, first service conception rates, the number of services per conception and pregnancy rates. These parameters will be evaluated using transrectal ultrasonography and/or palpation for pregnancy, and visual inspection of the breeding herd for return to estrus. Hormonal and ultrasonographic analyses will also be employed in tandem to monitor the incidence of early or late embryonic/fetal loss, and cases of ovarian dysfunction (ovulation failure, follicular and/or luteal cysts). Milk yield, composition, and quality will be quantified for individual or groups of cows under various environmental facilities modifications. Incidence of mastitis and other metabolic diseases will be recorded. The economic impact of introduced environmental modifications on milk production and reproductive performance will be assessed using standardized protocols. Data collection and economic analysis will be coordinated by NC.

Comparisons of the method of breeding during heat stress and effects of cooling on pregnancy rates will also be conducted through artificial insemination and embryo transfer as breeding methodologies. Investigations will also be conducted in the laboratory in vitro to examine the thermal sensitive and thermal resistant phases of early embryo development (**FL, MS, TN**). Research will be coordinated by FL using shared protocols. This will be accomplished through the cellular and molecular monitoring of the heat shock protein response in bovine embryos exposed to a thermal insult. Nuclear and cytoplasmic maturation of the maturing oocyte in response to elevated temperature will also be evaluated. Data from each location will be compiled for analysis.

9. EXPECTED OUTCOMES:

Results of this project will provide additional insight into the biological changes induced by heat stress which result in decreased lactational and reproductive performance for lactating dairy cows. With a better understanding of the physiologic and endocrine changes that occur in response to heat stress, improved management practices can be developed to reduce the negative effects of heat stress. One particular focus of this project is to assess the potential for improving production and reproduction in the subsequent lactation through incorporation of effective heat stress abatement techniques in combination with nutritional modifications during the dry period. This, combined with hormone therapy, would greatly improve both lactation and reproductive performance. Identification of genetic lines that are more tolerant of heat stress offers the potential to reduce the trend toward lower reproductive performance associated with selection for improved milk production. Dietary modifications or supplements that would either reduce heat production through improved digestion or production of metabolites that interfere with reproduction would be easy to implement.

Implementation of these practices at the farm level would improve overall efficiency and profitability. Management practices that improve overall efficiency would also reduce the number of animals sold due to poor reproduction or production, reducing the number of costly replacements required to maintain herd size. Improved lactation performance during late summer and fall would reduce the amount of milk imported into the Southeast US which is of particular interest to the many milk processors. Long term, this could reduce prices consumers pay as well as reduce some of the problems associated with shipping milk into the market.

Results of this project will be communicated to the end user through a variety of media including scientific journal articles, popular press articles, presentations at regional and state producer conferences, and presentations at regional, state, and local field days. Results of collaborative research projects will be reported in jointly authored manuscripts and submitted for publication in refereed journals selected by the authors. A regional symposium with a published proceedings will be scheduled near the end of the project to present results of the project and provide recommendations for reducing heat stress to enhance production and reproduction. Joint extension bulletins and popular press articles will be prepared to

communicate the results of this project. A web site will be established for the project. The site will include a list of project members, annual reports, and publications resulting from the project. As the project progresses, significant outcomes and recommendations will be posted.

10. ORGANIZATION:

The organization and supervision of this regional project will be by the Regional Technical Committee. This committee will consist of an Administrative Advisor appointed by the Southern Association of Agricultural Experiment Station Directors (SAAESD), a Technical Advisor appointed by USDA-CSREES, and one or more representatives from each cooperating Agricultural Experiment Station. Participating SAES with more than one representative will designate one representative as the voting member.

Members of the Regional Technical Committee will elect a Chair, a Chair-elect and a Secretary from the membership to serve as officers for the Regional Technical Committee. The Executive Committee shall consist of the current officers. A new Secretary shall be elected each year at the annual meeting by the voting members of the Regional Technical Committee. The previous Secretary shall become the Chair-elect for one year before ascending to the office of Chair. The Chair, in consultation with the Administrative Advisor, will notify the Regional Committee Members of the time and place of the annual meeting, prepare the agenda and preside at the meeting. The Chair-elect will be responsible for preparing the annual report. The secretary will record and prepare the minutes of the annual meeting for distribution to the membership, to SAAESD and to CSREES. If any member of the Executive Committee resigns, the remaining members shall, with the advice and consent of the Administrative Advisor, appoint a member of the Regional Technical Committee to fill the vacancy.

11. SIGNATURES:

Regional Project Title: Enhancing production and reproduction performance of heat-stressed dairy cattle.

David G. Morrison	8/1/00
Administrative Advisor	Date
Chair, Regional Association of Directors	Date
Administrator Cooperative State Research Service	Date

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ATTACHMENT 1: PROJECT LEADERS

LOCATIONS	PRINCIPAL OR CO-INVESTIGATORS	AREA OF SPECIALIZATION
Alabama	K. A. Cummins*	Nutritional endocrinology
	B. R. Moss	Ruminant nutrition
Florida	P. J. Hansen*	Reproductive physiology
	C. R. Staples	Ruminant nutrition
Georgia	J. K. Bernard*	Dairy cattle nutrition and management
	I. Misztal	Breeding and genetics
	J. W. West	Ruminant nutrition
Louisiana	J. D. Ward*	Ruminant nutrition
Mississippi	S. T. Willard*	Reproductive physiology
Missouri	M. C. Lucy	Reproductive physiology
	J. N. Spain*	Dairy cattle nutrition and management
	D. E. Spiers	Environmental physiology
North Carolina	G. A. Benson	Agricultural economist
	B. A. Hopkins	Ruminant nutrition
	S. P. Washburn	Reproductive physiology
	C. S. Whisnant*	Reproductive physiology
	L. W. Whitlow	Ruminant nutrition
South Carolina	J. A. Bertrand*	Ruminant nutrition
Tennessee	J. L. Edwards*	Reproductive biology/Embryology
	F. N. Schrick	Reproductive physiology
Virgin Islands	R. W. Godfrey*	Reproductive physiology

*Official Station Representative on Technical Committee

ATTACHMENT 2: RESOURCES

	Objective		Average Annual Input*		
Participant	1	2	**SY**	**PY**	**TY**
Alabama			**0.2**	**0.4**	**0.4**
K.A. Cummins	X		0.1	0.1	
B.R. Moss	X		0.1	0.3	0.4
Florida			**0.3**	**0.2**	
P.J. Hansen	X	X	0.3	0.2	
C.R. Staples	X				
Georgia			**0.7**	**1.1**	**0.2**
J.K. Bernard	X	X	0.2	0.2	0.2
I. Misztal	X		0.2	0.5	
J.W. West	X	X	0.3	0.4	0.4
Louisiana			**0.3**	**0.2**	**0.5**
J.D. Ward	X	X	0.3	0.2	0.5
Mississippi			**0.2**	**0.2**	**0.1**
S.T. Willard		X	0.2	0.2	0.1
Missouri			**0.25**	**0.35**	**0.1**
J.N. Spain	X	X	0.15	0.25	
D.E. Spiers	X		0.1	0.1	0.1
North Carolina			**0.4**	**0.2**	**0.4**
G.A. Benson	X	X	0.1		
B.A. Hopkins	X	X	0.1		0.1
S.P. Washburn		X	0.1	0.2	0.2
C.S. Whisnant	X	X			
L.W. Whitlow	X	X	0.1		0.1
South Carolina			**0.1**	**0.1**	
J.A. Bertrand		X	0.1	0.1	
Tennessee			**0.2**	**0.1**	
J.L. Edwards		X	0.1	0.1	
F.N. Schrick	X	X	0.1		
Virgin Islands			**0.2**	**0.2**	
R.W. Godfrey	X		0.2	0.2	

*Total average annual input for each location is indicated in bold.