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Ruidoso, New Mexico

(details inside)

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THIS MONTH"S ARTICLE:

Heat Stress Responses in Cattle

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Presented at the California Nutrition Conference

May 5-7, 2004
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New Mexico State University Extension Dairy Website: http://www/nmsu.edu/~dairy

The following videos are available for checkout from New Mexico State University. To obtain a video call Kathy Bustos, (505) 646-3326 or kbustos@nmsu.edu and the video will be sent in the mail, pending availability. There is only one copy of each video available, so we request that videos be returned within two weeks. Note that four of the videos contain an English and Spanish version.

1. The Milking School. Utah State University. Spanish and English. 30 minutes
2. Fitting and Showing Your Dairy Animal....A Winning Experience. Department of Dairy Science, University of Wisconsin. 20 minutes
3. Proper Milking Procedure. University of Florida. Spanish and English. 12 minutes
4. Milking Machine Maintenance. University of Florida. Spanish and English. 16 minutes
5. The Basics of Vacuum and Milking Systems. DHIA Services, 1991. 53 minutes
6. Understanding Dairy Cattle Behavior to Improve Handling and Production. Livestock Conservation Institute, 1992
7. Managing Milking/Ordenar Lecheria. Spanish and English. 1999. 33 minutes
8. Get Milk! Joining A Dairy Crew. University of New Hampshire, 1999. 45 minutes

Need to Calculate Production Costs?

University of Wisconsin dairy farm management specialist, Gary Frank, has developed a Excel spreadsheet to calculate variable cost of production and total cost of production. To access the spreadsheet, go to http://www.wisc.edu/dairy-profit, click on Decision Making Tools, then go to costcwt.xls.

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Heat Stress Responses in Cattle

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INTRODUCTION

Hot summer environmental factors such as, high ambient temperature, solar radiation, and high humidity are detrimental to dairy cattle, and result in decreased milk production and reproductive performance (Sharma et al. 1988, Cavestany et al. 1985). Heat stress results when a dairy cow's heat load is greater than her capacity to dissipate the heat causing body temperature to rise (Nickerson, 1987). Under these conditions, the primary route of heat loss is evaporative or insensible heat loss from sweating and respiration (Berman et al., 1985).

Indicators of heat stress in cattle include elevated rectal temperature (RT) and an increase in respiration rate (RR) (Lemerle and Goddard, 1986). The temperature-humidity index (THI) is commonly used for measurement of environmental heat load in dairy cattle (McDowell, 1972). However, THI is usually only available for ambient environment and does not provide information on the conditions immediately around the animals especially under cooling structures.

The inability of a cow to dissipate heat hinders feed consumption (Bernabucci et al., 1999), production (Johnson and Ragsdale, 1959; Brown et al., 1977), reproduction (Gwazdauskas et al., 1975; Collier et al, 1982a), and immune function. The use of shades and evaporative cooling is a cost effective method to reduce heat stress and increase milk yield (Collier et al., 1981; Igono et al., 1987, and Armstrong, 1994). To maximize economic gain from animal cooling, it is important to determine the thermal status of the cow under a given cooling structure. The method used to assess the thermal status should be rapid and economical. Previous studies have demonstrated that use of infrared thermography guns provide rapid and reliable information on skin surface temperature in cattle. (Collier et al 2003). Keeping skin surface temperatures below 35 ^{0}C minimizes milk production losses in a semi-arid environment (Collier et al 2003)

Acclimation is the response developed by the animal to an individual stressor within the environment (Fregley, 1996). The stressor may be severe enough that unless physiological changes are initiated decreased productivity or death are potential detrimental effects to the animal. Acclimatization is another term used to describe the process by which an animal adapts to several stressors within its natural environment (Willmer et al., 2000). Acclimation and acclimatization, often terms used interchangeably, are responses or changes in the animal's phenotype and include adjustments made to cold, heat, or toxic compound within its food source. This is contrary to evolutionary adaptation or natural selection where changes allow for preferential selection of an animal's phenotype and is based on a genetic component passed to the next generation. The altered phenotype of acclimated animals will return to normal if environmental stressors are removed. This is not the case in animals which are genetically adapted to their environment. Acclimation is still poorly understood and requires more research to determine the magnitude of its impact on animals.

ADAPTATIONS TO HEAT STRESS

Thermogenesis, the generation of body heat, is a major component of body temperature acclimation within mammals. Under natural conditions, there are two major components of heat production essential for maintaining body temperature homeostasis; heat generated from normal body processes (a by-product of metabolism) and the additional heat required for maintaining body temperature (Figure 1; Silva, 2003). As illustrated in figure 1, if ambient temperature decreases below the thermoneutral zone, heat production must increase to maintain body core temperature within the narrow temperature range necessary for normal physiological function. On the contrary, when body temperature begins to increase (due to increased heat acquired from elevated ambient temperature), the body mobilizes dissipitory mechanisms to reduce body temperature to its set point range.

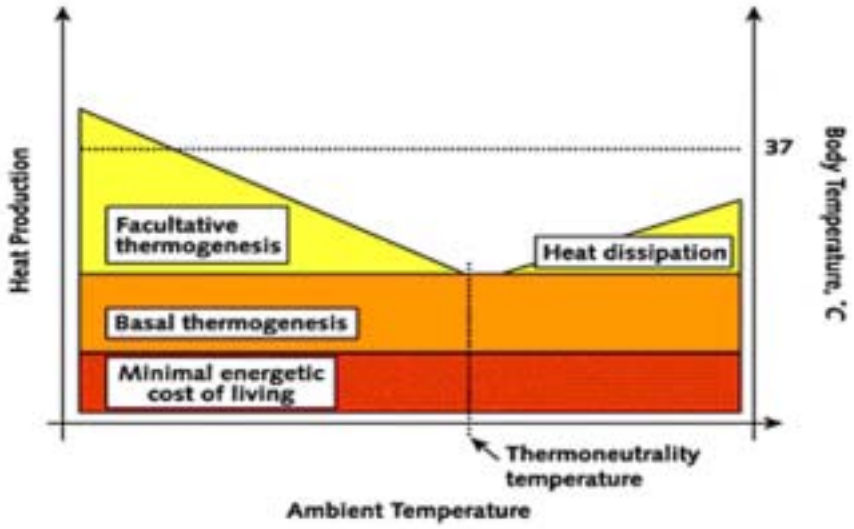

Figure 1- Effect of ambient temperature on thermogenesis in domestic animals (Adapted from Silva, 2003).

Thermotolerance is the result of short-term cellular changes caused by nonlethal, extreme heat exposure which permit an animal to survive heat stress (Moseley, 1997). For example, production of heat shock proteins (HSP) allow cells to survive heat exposure by chaperoning normal cellular proteins and preventing their degradation when exposed to denaturing stressors (i.e. ultraviolet radiation (Barbe et al., 1988). Differences exist between acclimation and thermotolerance because acclimation takes a period of days or weeks before animals develop the mechanisms to endure stress exposure. The ability of the acclimated animal to remain competent during heat stress occurs for some time after removal from the stressful environment. From this comparison, acclimation is a seasonal process that persists longer than thermotolerance but neither are the result of genetic change, so it is clear that acclimation and thermotolerance are part of the same phenomenon of phenotypic adaptation to thermal stress.

Heat acclimation requires metabolic and physiological changes within the body and this aspect is a homeorhetic process. As defined by Bauman and Curie (1980), homeorhesis is the “orchestrated changes for the priorities of a physiological state, i.e. coordination of metabolism in various tissue to support a physiological state”. Acclimation to heat stress involves the physiological changes required for animals to withstand a thermal stress.

Hormonal Control

Understanding the hormonal control and alterations within the heat acclimated animal are important for understanding the physiological mechanisms and responses to heat stress. The thyroid hormones, thyroxine (T_4) and triiodothyroxine (T_3), provide a major mechanism important for acclimation and have received the most research attention. As a general observation, it is well known that heat acclimation decreases endogenous levels of thyroid hormones and that mammals adapted to warmer climates

follow this pattern (Johnson and VanJonack,1976; Horowitz, 2001). Prolactin (PRL), which is indicative of heat stress (Collier et al. 1982a), and growth hormone (bST) may play important roles in acclimation through improved insensible heat loss and regulation of sweat gland function (Manulu et al 1991, Beede et al.1986).

Prolactin is a protein ligand that is a member of the lactogenic complex of hormones. PRL is important for mammogenesis (Buttle et al., 1979), lactogeneis (Collier et al., 1977; Akers et al., 1981), and to varying degrees across species, for galactopoiesis (Wilde and Hurley, 1996). Additional non-lactogenic roles for PRL, include altering sweat gland function during acclimation. Bromocryptine, a blocker of PRL secretion, treatment during heat acclimation in humans affects sweat gland function by preventing increases in sweat gland discharge and decreases Na^+ output from the gland (Kaufman et al., 1988). PRL concentrations within the blood can be manipulated by many factors including increased ambient temperature (Mueller et al., 1974; Wetteman and Tucker, 1979) and day length (Leining et al., 1979), but the mechanisms differ between these two factors. Increasing ambient temperature suppresses the negative feedback of prolactin on dopaminergic neurons within the infundibulum/pituitary stalk (Tucker et al., 1991) while function of the dopaminergic neurons is not altered with a longer photoperiod (Zinn et al., 1991).

Another important hormone within the galactopoetic complex of signals is bST. Dairy cows treated with exogenous (Bauman and Vernon, 1993) or endogenous (Dahl et al., 1991) bST increase their milk production. BST-treated cows repartition nutrients in favor of milk production and if cows are in negative energy or protein balance, at the expense of body tissues (Tyrrell et al., 1988). Heat production also increases in bST-supplemented cows (Tyrrell et al., 1988). If increased bST enhanced conversion of T_4 to T_3 thereby increasing metabolic rate it could cause negative effects in heat acclimated animals (Wolthers et al., 1996). However, little is known concerning growth hormone secretion during heat stress. Somatotroph cells within the pituitary, did not alter GH secretion between hot (30°C) or neutral (20°) conditions in 3 wk gilts (Matteri and Becker, 1994).

In hot environments the most common methods cows use to dissipate additional heat is through increasing respiration rate and/or sweating rate (McDowell et al., 1976). Lactating Holstein cows treated with bST during the summer demonstrated a 19 and 25% increased heat produced under thermoneutral and heat stressed conditions,(Manalu et al., 1991). However, heat dissipation in bSt treated cows was increased 36 and 24% under thermoneutral and heat stressed conditions; (Manalu et al., 1991). In the same experiments replicated in the winter, heat production increased 18 and 10% while heat dissipation increased only 8 and 0.3% compared to controls under thermoneutral and cold stress conditions (Manalu et al., 1991). In summer-acclimated cattle, their ability to carry an increased thermal load was improved by bST while winter acclimated cows improved their ability to withstand cold stress when treated with bST(Manalu et al., 1991). The treatment of growth hormone deficient human patients with human GH restored IGF-I concentrations to physiological normality and increased sweating rates (Hasan et al., 2001). Using immunohistochemistry, the motor neurons in the sweat gland increased expression of acetylcholinesterase and vasoactive intestinal polypeptide (Hasan et al., 2001), which are important for activation of the sweat gland via the nerves. GH treatment in these two cases increased sweating rates demonstrating GH potential to be beneficial in aiding in heat dissipation of heat-acclimated animals.

The hypothalamic-pituitary-adrenal axis including corticotropin releasing hormone (CRH), adrenocorticotropic hormone or corticotropin (ACTH), cortisol, and aldosterone also are altered by thermal stress and are involved in acclimatory responses to thermal stress (Collier et al, 1982a, Beede et al. 1986). CRH stimulates somatostatin release from the hypothalamus, which can inhibit GH and TSH secretion from the pituitary (Riedel et al., 1998), and down regulate the thermogenic effects of both GH and thyroid hormones. In dairy cattle, the glucocorticoids decrease during acclimation at 35°C

(Alvarez and Johnson, 1973) and are lower in thermal acclimated animals compared to controls (Collier et al. 1982a, Beede et al., 1986). Thus, the hypothalamic-pituitary-adrenal axis appears to play a role in heat-acclimation by decreased secretion of its products. The catecholamines, epinephrine (EPI) and norepinephrine (NE), appear not to be affected by acclimation (Alvarez and Johnson, 1973), but this could be due to increased basal levels required for increased sweating rate. Ghrelin infusion(1nmol/rat) into the third ventricle, but not GH (0.5 nmol/rat) increased food intake and decreased sympathetic nervous activity in brown adipose tissue (BAT) resulting in decreased energy expenditure and thermogenesis (Yasuda et al.,2003). Prolactin (Gualillo et al., 1999) and TSH (Menendez et al., 2003) can stimulate release of leptin from white adipose tissue indicating that leptin could be modulated in response to increases and decreases in ambient temperature, although no definitive research exists to provide evidence for this relationship. Melatonin could be important for heat acclimation through its hypothermia inducing capacity (Rozenboim et al., 1998). It attenuates the response to heat stress by decreasing core body temperature and increasing heat dissipation (Harlow, 1987). It would be unlikely melatonin plays an extremely important role in heat dissipation since increasing temperatures are usually correlated to increasing day length, which would suppress melatonin production. These hormones could be important in modulating heat acclimation in mammals although the thyroid hormones, prolactin, and growth appear to have substantial effects.

WHOLE BODY INTEGRATION

There is evidence for a biphasic pattern of heat acclimation divided into periods based on time. Short-term heat acclimation (STHA)is the phase where changes begin to take place within cellular signaling pathways (Horowitz et al., 1996). These changes create disturbances in cellular homeostasis and begin to reprogram the cell to combat the deleterious effects of heat stress (Horowitz, 2001). Full expression of the STHA is obtained when the drop in T_3 and T_4 begin to exceed 30-40% (Horowitz, 2001). When thyroid hormones concentrations decline, the regulation of gene transcription is altered transforming myosin from type V1 to a slower, more efficient form, type V3 (Horowitz et al., 1986). In lactating dairy cattle, STHA is characterized by an initial overcompensation to heat exposure resulting in a decrease in milk production (Johnson and Vanjonack, 1976). These STHA adaptations allow animals to compensate for increased heat stress before more permanent adaptations can be made.

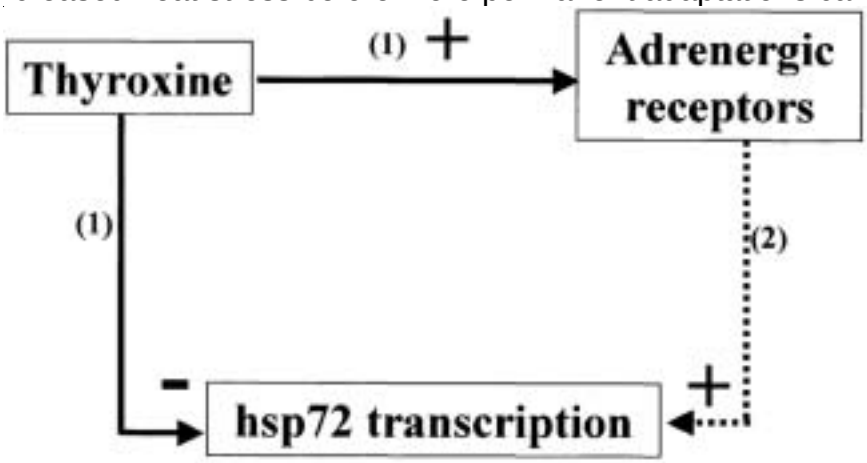

Figure 2-Adapted from Maloyan and Horowitz (2002). The proposed model for the regulation of HSP 72 expression by thyroxine and adrenergic receptors.

When all of the changes occurring during STHA are complete and the heat-acclimated phenotype is expressed, long-term heat acclimation (LTHA) has occurred (Horowitz, 2001). In LTHA animals, HSP 72 is expressed at 200 fold compared to non-acclimated controls (Maloyan and Horowitz, 2002). During 30 d acclimation (34°C), rats grew at a slower rate than controls while rats treated with thyroxine or â-adrenergic receptor antagonist did not deviate from controls (Maloyan and Horowitz, 2002). Maximal response to heat stress (41°C) occurred with heat acclimated rats treated with â-adrenergic receptor

antagonist (Maloyan and Horowitz, 2002). From these experiments, a proposed model for HSP response to acclimation has been created (Figure 2) where T_3 and T_4 exert a negative feedback on HSP production at the cellular level. When â-adrenergic receptors are activated under conditions of elevated temperature, HSP protein production increases allowing for HSP synthesis to be controlled by the decrease in thyroid hormones and activation of â-adrenergic receptors. The positive effects that T_3 and T_4 create on adrenergic receptors has yet to be explained in the model context, but it could allow for increased adrenergic receptors at the initiation of heat acclimation in order to provide more signaling cascades to activate HSP production while T_3 and T_4 are still decreasing (Maloyan and Horowitz, 2002). The muscarinic signal transduction pathway is important for the activation of the evaporative cooling system in the rat(Horowitz et al., 1996). An expanded list of the changes that occur in response to LTHA are presented in Table 1. Most of these examples deal with decreased heat production within the body and increased ability of the animal to dissipate the heat obtained from the environment. In lactating cows, when LTHA has occurred, the low producing cow's milk production will reach a level comparable to what she should milk while not under heat stress, but in the higher producing cattle it could still be below the milk production possible in a thermoneutral environment (Johnson and Vanjonack, 1976). The adaptations during this phase of acclimation allow the animal to persist in an environment without negative effects of acute heat stress. The bST examples demonstrates that LTHA can be improved by increasing heat dissipation capability of animals.

Table 1-A partial list of the adaptations made in target tissue to heat acclimation and stress.

Species	Cell type or tissue	Response	Reference
Cow	Sweat gland	Increased sweating rate	Manalu et al., 1991
Rat	Hypothalamus	Altered heat dissipation threshold	Horowitz, 2001
Rat	Cardiac	Increased HSP synthesis	Maloyan and Horowitz, 1999
Rat, cow	Thyroid	Depress thyroid function	Horowitz, 2001 Johnson et al., 1991
Rat	Cardiac	Reduced heart rate	Horowitz, 2001
Mouse	Brain-Medulla	Improved synaptic transmission	Kelty et al., 2002
Rat	Vascular system	Increased vascular reverse	Horowitz et al., 1986
Rat	Muscle	Switch from myosin isozyme V1 to V3	Horowitz et al., 1986

For whole body metabolism of carbohydrates, there appears to be no difference between acclimated animals and unacclimated controls. There were no differences in gluconeogenic rates from liver slices isolated from both heat acclimated (acclimated at 35°C and 35% RH) and non-acclimated hamsters (controls) producing glucose at approximately 4%/g of wet weight/h (Chayoth, 1976). Gluconeogenesis was enhanced in heat acclimated hamsters given T_3 compared to controls injected with T_3 most likely demonstrating a thyroid hormone but not an acclimation effect (Chayoth, 1976). These researchers went on to determine the effects of cyclic AMP on gluconeogenesis and demonstrated no cyclic AMP effects (Chayoth, 1976). With no response to cyclic AMP, a product that would occur in cells by adrenergeric receptor activation by NE or EPI, suggests no preferential partitioning of carbohydrates in the heat acclimated animal. Data from the rabbits with a disabled dorsal fornix in the hypothalamic-limbic system displayed altered pyruvate metabolism (Seto et al., 1985). In these rabbits, the response to heat exposure did not differ from d 1 to d 10 suggesting that the dorsal fornix may play an important role in regulating metabolism of pyruvate during heat exposure (Seto et al., 1985). There appear to be no differences in the heat acclimated animal in regards to carbohydrate metabolism.

An alteration of lipid metabolism occurs in the acclimated animal. These animals display a decreased activation of pathways associated with ù-6 fatty acids in favor ù-3 fatty acid production. In rats, heat acclimation at 34°C (30-40% RH) for 30 days decreased cardiac content of 16:0, 18:1, and 22:5 while 20:4 and 22:6 increased (32 and 96%, respectively; Shmeeda et al., 2002). The authors

used the ratio of docosahexaenoic acid (22:6) to ä-arachidonic acid (20:4) to determine changes in those pathways as they are major products of the ù-3 and ù-6 fatty acid synthesis pathways. The ù-3/ù-6 ratio increased from controls to the 2 d acclimated rats with the highest levels observed in the 30 d acclimated rats (0.84, 1.12, 1.25, for the control, 2 d, 30 d, respectively). The authors note that ù-3 production could be underestimated, because 20:5 (arachidonic acid), another product of the ù-3 pathway, could be diverted to the production of prostaglandins (Shmeeda et al., 2002). Also, inhibition of prostaglandin synthesis by a potent nonsteroidal anti-inflammatory, diclofenac, decreased circulating PRL levels from 183.9 ìIU/ml to 58.0 ìIU/ml (Joris et al., 1988) suggesting that prostaglandin synthesis is necessary for PRL secretion and thus inhibiting PRL synthesis would prevent release. These changes in plasma membrane lipids could play important roles in changing the affinities or G protein-coupled receptors and K^+/Na^+ ATPase pump that have been observed during heat acclimation by changing the fatty acid composition of the membrane (Horowitz et al., 1996; Kaspler and Horowitz, 1997).

Controls over lipolysis and FFA oxidation appear to change in response to heat acclimation. With BAT slices isolated from rats held at control lab temperatures, acclimated to heat, and acclimated to cold; basal lipolysis was similar between heat acclimated and thermoneutral condition, but lipolysis increased from cold-acclimated tissue was increased (3.2, 3.6, 9.7 ìeq/g/h, repectively; Rabi et al., 1977). Increases in lipolysis could be important in cold-acclimated animals for nonshivering thermogenesis to increase body temperature. Upon stimulation with EPI, heat-acclimated rats released less FFA (5.6, 8.3, 9.6 ìeq/g/h, respectively) and less glycerol (1.9, 2.9, 3.6 ìmol/g/h, respectively) than cold-acclimated and control hamsters (Rabi et al., 1977). This decrease of FFA release could provide an advantage for the heat-acclimated animal preventing FFA oxidation and decreased production of heat in response to sympathetic stimulation. The obese rat model provides an interesting look at the effects of heat-acclimation in a model that is unresponsive to factors affecting lipolysis. In a study comparing lean Wistar and obese Zucker rats (fa/fa), basal metabolic rate (BMR) was increased (kg/lean mass) in the heat acclimated obese group and NE increased metabolic rate in both lean and obese rats (Al-Arabi and Andrews, 2003). An effect of TRH on BMR did not occur during the first week in either the obese or lean rats of chronic TRH treatment, but it decreased BMR in lean rats (Al-Arabi and Andrews, 2003). TRH and NE chronically administered together for 2 wk potentiated the increase in BMR in both groups (Al-Arabi and Andrews, 2003). NE treatment increased food intake in both lean and obese rats (Al-Arabi and Andrews, 2003). TRH treatment significantly decreased accumulated food intake in lean rats compared to control where there was a numerical decrease in accumulated food intake in obese rats, although it was not significant (Al-Arabi and Andrews, 2003). Together, accumulated food intake did not differ from controls in both lean and obese rats with TRH and NE treatment (Al-Arabi and Andrews, 2003).

In rats, heat-acclimation was achieved by exposing lactating dams to heat (38°C, 40-50% RH) for 50 m in the morning. Resting rectal temperature was increased in rats acclimated to heat compared to lactating controls (Knecht et al., 1980). In heat acclimated dams, maternal weight was decreased compared to controls only at d 11 of lactation (326 vs. 298 g, respectively) while average pup weight was decreased in heat acclimated dams compared to controls at d 11 (17.1 vs. 21.9 g, respectively), d 16 (24.4 vs. 29.7 g, respectively), and d 21 (33.7 vs. 39.8 g, respectively; Knecht et al., 1980). No effect was noted on pup survival between both groups. Survival time when exposed to ambient temperatures of 39.5°C numerically increased for heat acclimated dams (250.5 vs. 159.0 m for controls), lethal temperature was decreased compared to controls (44.9 vs. 45.1°C, respectively; Knecht et al., 1980). Evaporative heat loss did not differ between groups (38.2 [heat acclimated] vs. 41.2 g/(kg*h) [controls]; Knecht et al., 1980). Maximal evaporative heat loss occurred at 41.5°C (rectal temperature) for both heat acclimated and control dams although it should be noted that although there were no statistical differences evaporative heat loss remained elevated at temperature greater than 41.5°C (Knecht et al., 1980).

CONCLUSION

Acclimation to heat stress is a homeorhetic process which involves changes in hormonal signals which affect responsiveness of target tissues to environmental stimuli. Improving our understanding of this process will lead to improved genetic selection of heat stress resistant genotypes.

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HIGH COW REPORT
APRIL, 2004

MILK

Arizona Owner	Barn#	Age	Milk	New Mexico Owner	Barn #	Age	Milk
* Withrow Dairy	1082	4-00	51,420	S.A.S. Dairy	4530	4-11	40,607
* Stotz Dairy	16824	3-00	40,080	* Providence Dairy	4674	3-10	38,490
* Triple G Dairy, Inc.	2340	4-02	37,450	* Goff Dairy	18936	4-03	37,550
* Stotz Dairy	14824	4-05	35,600	* Hide Away Dairy	4230	5-06	37,540
* Triple G Dairy, Inc.	1911	5-09	35,390	* Hide Away Dairy	4155	5-06	36,840
* Withrow Dairy	1363	3-11	34,890	* Providence Dairy	8322	---	36,820
* Mike Plyman Dairy	104	1-06	34,760	* Goff Dairy	16756	4-03	36,580
* Stotz Dairy	16361	3-05	34,500	S.A.S. Dairy	4453	5-00	36,341
* Treger Holsteins, Inc.	702	5-07	34,310	S.A.S. Dairy	82	5-03	36,251
* Stotz Dairy	11870	6-08	34,290	* Providence Dairy	8494	4-00	36,220

FAT

* Stotz Dairy	15904	3-07	1441	Pareo Dairy	9084	6-07	1506
* Withrow Dairy	1082	4-00	1423	* Hide Away Dairy	2900	6-06	1413
* Mike Pylman	6765	3-05	1420	Pareo Dairy	91	5-09	1355
* Triple G Dairy, Inc.	2340	4-02	1400	Pareo Dairy	1358	6-06	1339
* Mike Pylman Dairy	5547	5-02	1364	Pareo Dairy	8243	5-00	1314
* Stotz Dairy	14824	4-05	1337	Pareo Dairy	8195	6-08	1313
* Triple G Dairy, Inc.	1911	5-09	1334	Pareo Dairy	573	6-08	1299
* Stotz Dairy	16824	3-00	1280	Pareo Dairy	509	5-03	1291
* Stotz Dairy	19586	5-11	1269	Pareo Dairy	8355	5-02	1266
* Ponderovey Jerseys	21818	4-01	1262	Caballo Dairy	3982	4-09	1261

PROTEIN

* Withrow Dairy	1082	4-00	1203	* Hide Away Dairy	4230	5-06	1137
* Triple G Dairy, Inc.	2340	4-02	1136	S.A.S. Dairy	4530	4-11	1124
* Stotz Dairy	16824	3-00	1083	*Providence Dairy	8529	---	1120
* Stotz Dairy	14824	4-05	1077	* Hide Away Dairy	4155	5-06	1097
* Triple G Dairy, Inc.	1911	5-09	1070	* Goff Dairy	16756	4-03	1094
* Stotz Dairy	16361	3-05	1049	* Milagro Dairy	9030	4-03	1087
* Stotz Dairy	15904	3-07	1043	Breedyk Dairy	60	6-06	1087
* Mike Pylman Dairy	5167	5-08	1036	Pareo Dairy	8243	5-00	1074
* Mike Pylman Dairy	104	1-06	1017	Breedyk Dairy	63	5-06	1062
* Stotz Dairy	16270	3-05	997	* Hide Away Dairy	4806	4-03	1060

*all or part of lactation is 3X or 4X milking

ARIZONA – TOP 50% FOR F.C.M. [b]
APRIL 2004

OWNERS NAME	Number of Cows	MILK	FAT	3.5 FCM	D.I.M.
Stotz Dairy West	2087	27,593	993	28,035	233
University of Arizona Holsteins	165	27,621	971	27,690	240
Red River Dairy	4490	26,403	951	26,839	197
Triple G Dairy, Inc.	4112	25,231	938	26,121	197
Treger Holsteins, Inc.	627	25,930	887	25,597	225
Mike Pylman Dairy	3750	25,427	900	25,590	176
Danzeisen Dairy, LLC	1362	24,593	896	25,164	228
Yettem Dairy	2691	21,067	972	24,872	198
Stotz Dairy East	1133	24,155	861	24,407	240
Arizona Dairy Company	5742	23,933	843	24,020	170
Del Rio Holsteins	834	22,618	803	22,802	184
Zimmerman Dairy	1128	22,503	795	22,623	185
Dairyland Milk Company	2803	23,150	769	22,481	153
Paul Rovey Dairy	395	22,070	797	22,468	128
D C Dairy, LLC	1032	22,121	792	22,409	150
Hillcrest Dairy	2312	22,946	763	22,295	213
Butler Dairy	591	22,598	762	22,129	207
Saddle Mountain Dairy	2684	23,262	743	22,108	147
Withrow Dairy	5001	22,743	738	21,802	169
Shamrock Farm	7986	21,733	741	21,414	147
Goldman Dairy	2001	21,541	743	21,364	152
RG Dairy, LLC	1219	21,161	739	21,134	160
Lunts Dairy	556	20,573	739	20,880	198
Dutch View Dairy	1564	20,701	721	20,644	227
Parker Dairy	4188	20,293	722	20,483	230
Jerry Ethington Dairy	561	20,268	722	20,473	207
Caballero Farms, LLP	1854	20,160	721	20,410	196

NEW MEXICO – TOP 50% ACTUAL MILK
APRIL 2004

OWNERS NAME	Number of Cows	MILK	FAT	3.5 FCM	D.I.M.
Pareo Dairy #1	1411	26,277	935	26,769	217
Providence Dairy	2815	26,877	872	25,762	200
Tallmon Dairy	476	26,067	848	25,023	215
Pareo Dairy # 2	2979	24,181	889	24,872	192
Hide-Away Dairy	2076	26,126	837	24,870	196
Ken Miller Dairy	395	24,757	863	24,699	215
Do-Rene Dairy	2237	24,466	842	24,233	196
New Direction Dairy # 2	1847	23,147	862	23,987	229
New Direction Dairy # 1	37	22,925	851	23,713	193
Wormont Holsteins	1390	22,721	830	23,284	190
Butterfield Dairy	1680	22,521	816	22,970	182
Price's Roswell Farm	2813	22,792	806	22,925	195
Goff Dairy # 1	4140	22,306	813	22,829	209
Vaz Dairy	1817	23,263	781	22,724	209
Hafliger Dairy	1886	21,915	803	22,497	172
Milagro Dairy	3380	22,337	790	22,469	198
S.A.S. Dairy	2000	22,879	764	22,282	190

[b] all or part of lactation is 3X or 4X milking

average milk and fat figure may be different from monthly herd summary; figures used are last day/month

ARIZONA AND NEW MEXICO HERD IMPROVEMENT SUMMARY
FOR OFFICIAL HERDS TESTED APRIL, 2004

		ARIZONA	NEW MEXICO
1.	Number of herds	53	31
2.	Total cows in herd	81,935	62,199
3.	Average herd size	1546	2006
4.	Percent days in milk	93	87
5.	Average days in milk	213	199
6.	Average milk – all cows per day	65	62
7.	Average percent fat – all cows	3.5	3.5
8.	Total cows in milk	77,693	54,368
9.	Average daily milk for milking cows	70.2	71.0
10.	Average days in milk – 1st breeding	83	71
11.	Average days open	158	147.7
12.	Average calving interval	13.9	13.8
13.	Percent somatic cell – linear 0-4	93	80.9
14.	Percent somatic cell – linear 5-6	4	12.4
15.	Percent somatic cell – linear 7 & above	3	5.6
16.	Average previous days dry	63	62
17.	Percent cows leaving herd	34	30.4
		STATE AVERAGE	
	MILK	21,685	
	Percent butterfat	3.49	
	Percent Protein	2.91	
	Pounds fat	776	
	Pounds protein	637	

ARIZONA COOPERATIVE EXTENSION
U.S. DEPARTMENT OF AGRICULTURE
The University of Arizona
Tucson, Arizona 85721

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Upcoming Events:
Arizona Dairy Production Conference
November 4, 2004
Sheraton Phoenix Airport Hotel
Tempe, Arizona