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Genetic basis of heat tolarance in cattle

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Abstract
Now-a-days, heat stress is a major problem in cattle population by impairing cattle health and production ability which indirectly affects the economy of farmers. The cellular heat stress (HS) response is one component of the acute systemic response to HS. Gene networks within and across cells and tissues respond to environmental heat loads above the thermoneutral zone with both intracellular and extracellular signals that coordinate cellular and whole-animal metabolism. Activation of these systems appears to be initiated at skin surface temperatures exceeding 35 °C as animals begin to store heat and rapidly increase evaporative heat loss (EVHL) mechanisms. Genes regulating heat tolerance in cattle should be known for selection of better animals in future.

Keywords: Cattle, heat tolerance, genes, thermoregulation

Introduction
The thermal environment plays a major role during early lactation of cow, negatively affecting milk production of dairy cows. More attention has been paid to the thermoregulatory mechanism especially in animals of high genetic merit. The impact of heat stress can be relieved by modification of the environment (nutrition, cooling) or by genetic selection of animals. Identification of such animals can be based on measurements of their immediate response (rectal, skin, milk temperature, respiration rate) to the exposure to heat stress conditions.
Heat stress is a condition caused by an animal's inability to dissipate body heat effectively to maintain normal body temperature, a vital process known as thermoregulation.
Alternatively, a decline of production due to heat stress can be used as an indicator of heat tolerance. The animal with a minimal decline of milk production per degree of increase of a climatic variable is identified as heat tolerant.

Overview of Heat Stress
Heat stress results from a negative balance between the net amount of energy flowing from the animal to its surrounding environment, and the amount of heat energy produced and absorbed by the animal. Essentially, cattle that are producing and absorbing more heat from the environment than they can dissipate will experience heat stress. While cattle can acclimatize to hotter conditions, an individual animal's adjustment period encompasses anywhere from 2-7 weeks (Blackshaw and Blackshaw 1994) [1]. Additionally, animals exhibiting higher levels of performance tend to generate more heat due to their inherently higher levels of productivity; hence, they experience more heat stress.

Table 1: Cattle comfort category (Mader *et al*. 2010) [2]

Mesonet cattle comfort categories	Comprehensive Climate Index Categoriesa	Impacts	Cattle Comfort Index (℃)	Cattle Comfort Index (℉)
Heat Danger	Hot conditions: Extreme danger	Animal deaths may exceed 5%	>40	>105
Heat Caution	Hot conditions: Moderate to Severe	Decreased production, 20% or more Reduced conception, as low as 0%	30 to 40	85 to 105
Comfortable	Mild conditions		-10 to 30	15 to 85
Cold Caution	Cold conditions: Moderate to Severe	18-36% increase in dry matter intake	-10 to -30	15 to -20
Cold Danger	Cold conditions: Extreme danger		<-30	<-20

Genes responsible for heat tolerance in cattle

During heat stress gene expression changes include

- Activation of heat shock transcription factor 1(HSF1)
- Increased expression of heat shock proteins (HSP) and decreased expression and synthesis of other proteins
- Increased glucose and amino acid oxidation and reduced fatty acid metabolism
- Endocrine system activation of the stress response
- Immune system activation via extracellular secretion of HSP.

Heat shock proteins (HSP) plays a central role in cytoprotection during HS by protecting the animal against hyperthermia, circulatory shock, and cerebral ischemia by overexpression of HSP (Lee *et al.,* 2006) [3]. Thermotolerance appears to be a quantitative trait influenced by many regions of the genome, and genomics studies have identified regions of the genome that appear to be important for regulation of body temperature in both beef and dairy cattle (Dikmen *et al., 2012* [13]; Howard *et al.,* 2014 [6, 11]; Hayes *et al.,* 2009) [5]. Additionally, some breeds may be segregating for genes of large effect on heat tolerance. Howard *et al.* (2014) [6, 11] showed that only a small number of the most influential (top 5%) genomic regions involved in predicting body temperature regulation during the summer and winter were shared between both traits (9%), which means that advancements in selection for both heat and cold tolerance traits

While there are undoubtedly many factors and pathways that influence thermotolerance in cattle, several factors that have been implicated in cellular processes regulating thermotolerance include peroxisome proliferator-activated receptor alpha (PPARα; Fang *et al.,* 2014) [8], heat shock proteins (Hansen 1999; 2014) [9, 10], glutathione (Hansen, 1999) [9], and the insulin-like growth factor 1 system (Hansen, 2014) [10]. Heat shock proteins have been moderately well-studied in the context of bovine embryo development because reproduction is very easily disrupted by heat stress (Hansen 2014) [10].

Table 2: The genes and/or pathways and functions identified in genome-wide association studies are outlined in below.

Pathway/Function	Gene(s)	Publication
Cellular response to stress	STAC, WRNIP1, MLH1, RIPK1, SMC6, GEM1	Howard *et al*. 2014 [6, 11]
Response to heat	STAC	Howard *et al*. 2014 [6, 11]
Gap junction	TUBB2A, TUBB2B	Howard *et al*. 2014 [6, 11]
Cellular response to stress	CCNG, TNRC6A	Howard *et al*. 2014 [6, 11]
Apoptosis	FGD3, G2E3, RASA1, CSTB, DAPK1, MLH1, RIPK1, SERPINB9, HMGB1	Howard *et al*. 2014 [6, 11]
Ion transport	CACNG3, CLCN4, PRKCB, TRPC5, KCNS3, SLC22A23, TRPC4	Howard *et al*. 2014 [6, 11]
Thyriod hormone regulation	DIO2	Howard *et al*. 2014 [6, 11]
Body weight and feed intake	NBEA	Howard *et al*. 2014 [6, 11]
Heat shock protein response	HSPH1, TRAP1	Howard *et al*. 2014 [6, 11]
Respiration	ITGA9	Howard *et al*. 2014 [6, 11]
Calcium ion and protein binding	NCAD	Dikmen *et al*. 2012 [13]
Protein ubiquitination	RFWD12, KBTBD2	Dikmen *et al*. 2012 [13]
CEP170, PLD5		Dikmen *et al*. 2012 [13]
Thyriod hormone regulation	SLCO1C1	Dikmen *et al*. 2012 [13]
Insulin signaling	PDE3A	Dikmen *et al*. 2012 [13]
RNA metabolism	LSM5, SNORD14, SNORA19, U1, SCARNA3	Dikmen *et al*. 2012 [13]
Transaminase activity	GOT1	Dikmen *et al*. 2012[13]
Apoptosis, cell signaling	FGF4	Hayes *et al*., 2009[5]

Several reports showed associations of SNP in the HSP genes with thermal stress response

and tolerance in cattle. Association of polymorphisms in Hsp90AB1 with heat tolerance has also been reported in Sahiwal and Frieswal cattle (Deb *et al*., 2014) [15], HSF1 gene (Li *et al*., 2011a) [16], HSP70A1A gene (Li *et al*., 2011b) [17], HSBP1 (Wang *et al*., 2013) [18] in Chinese Holstein cattle. There are non-Hsps genes also revealed to undergo changes in expression in response to HS. For example ATP1B2 gene in Chinese Holstein cows (Wang *et al*., 2011) [20] and ATP1A1 gene in jersey crossbred cows (Das *et al.,* 2015) [22] were observed to have associated with thermotolerance. These SNPs could be used as markers in marker assisted selection to developed thermotolerant animal in early ages. Further, thermotolerant bull can be used in breeding policy to have thermal adapted offspring.

Effect of Heat Stress on Cattle Population

- HS reduces the length and intensity of estrus besides increases incidence of anestrous and silent heat in farm animals.
- It increases ACTH and cortisol secretion and blocks estradiol-induced sexual behavior. It has been reported that developed follicles suffer damage and become non-viable when the body temperature exceeds 40 °C (Roth *et al*., 2000) [24].
- Embryonic growth and survival also affected during thermal stress in dairy animals. HS causes embryonic death by interfering with protein synthesis (Edwards, 1996) [25], oxidative cell damage (Wolfenson, 2000) [24], reducing interferon- tau production for signaling pregnancy recognition (Bilby, 2008) [29] and expression of stress-related genes associated with apoptosis (Fear, 2011) [29].
- In bull, increased testicular temperature results from thermal stress could changes in seminal and biochemical parameters leads to infertility problems in bulls and significant seasonal difference in semen characteristics

Approaches to ameliorate HS

Physical modifications of the environment, nutritional management and genetic development of thermotolerant breeds are key components for sustainable livestock production in tropical hot climates.

Conclusion

Heat stress is a multi-faceted challenge that can be mitigated utilizing a variety of available tools and resources. Heat stress not only causes production losses, but is also an important animal well-being issue that merits consideration in management and breeding programs. Proper understanding of genetic regulation of cutaneous evaporative heat loss (EVHL), determining the role of HSF in coordinating cellular metabolism and survival during HS and genetic regulation of nutrient partitioning during thermal stress can help to decrease thermal stress in cattle. Determining the basis for altered energy metabolism during thermal stress will lead to opportunities for improved animal performance via altered nutritional management.

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