Technical guide

Heat stress management in dairy cows

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High temperatures and humidity are detrimental to the productivity of dairy cows. According to Bernaducci et al. (2010), "heat stress can be defined as a physiological condition when the core body temperature of a given species exceeds its range specified for normal activity, which results from a total heat load (internal production and environment) exceeding the capacity for heat dissipation and this prompts physiological and behavioural responses to reduce the strain."

Heat stress affects feed intake, cow body temperature, maintenance requirements and metabolic processes, feed efficiency, milk yield, reproductive efficiency, cow behaviour, and disease incidence. These effects have been well documented.

Domestic animals have a core body temperature (CBT) range in which metabolism functions without modification. The ambient temperature range needed to maintain this is termed the thermoneutral zone. Typically, core body temperature is higher than ambient temperature to ensure that heat generated by metabolism flows out to the environment (Collier et al., 2006). Deviation outside of this range – which is relatively narrow – leads to increases in resting metabolism, modifications to the biochemistry and cellular physiology as well as the behaviour of the animal (Shearer and Beede, 1990). The thermoneutral zone lies between 5 and 25° C for dairy cattle (Roenfeldt, 1998). Above 25 °C, the body must modify physiology and behaviour to keep CBT stable.

HEAT STRESS AND THERMAL HUMIDITY INDEX

Temperature is not the only environmental factor that affects the intensity of heat stress. The temperature humidity index (THI) measures the combined effects of ambient temperature and relative humidity (RH) to ascertain heat load intensity (Berry et al., 1964). This index is now categorized into heat stress levels with an index above 72 THI (23.9 °C with 65% RH to 32.2 °C with 0% RH) established as the lower threshold of heat stress (Whittier, 1993; Armstrong, 1994). However, following the increased milk production per cow since the development of the THI, the 10 kg/d increase in milk production will decrease the threshold for heat stress by 5 °C (Berman, 2005). A recent re-evaluation of the THI has suggested that due to this improvement of milk production, the THI heat stress threshold should be lowered to 68 THI (22.2 °C with 45% RH to 26.7 °C with 0% RH; Zimbelman et al., 2009).

Figure 1: Temperature Humidity Index for lactating dairy cows
(Zimbelman and Collier, 2011)

Temperature		% Relative Humidity																		
°F	°C	0	5	10	15	20	25	30	35	40	45	50	55	60	65	70	75	80	85	90
72	22.0	64	65	65	65	66	66	67	67	67	68	68	69	69	69	70	70	70	71	71
73	23.0	65	65	66	66	66	67	67	68	68	68	69	69	70	70	71	71	71	72	72
74	23.5	65	66	66	67	67	67	68	68	69	69	70	70	70	71	71	72	72	73	73
75	24.0	66	66	67	67	68	68	68	69	69	70	70	71	71	72	72	73	73	74	74
76	24.5	66	67	67	68	68	69	69	70	70	71	71	72	72	73	73	74	74	75	75
77	25.0	67	67	68	68	69	69	70	70	71	71	72	72	73	73	74	74	75	75	76
78	25.5	67	68	68	69	69	70	70	71	71	72	73	73	74	74	75	75	76	76	77
79	26.0	67	68	69	69	70	70	71	71	72	73	73	74	74	75	76	76	77	77	78
80	26.5	68	69	69	70	70	71	72	72	73	73	74	75	75	76	76	77	78	78	79
81	27.0	68	69	70	70	71	72	72	73	73	74	75	75	76	77	77	78	78	79	80
82	28.0	69	69	70	71	71	72	73	73	74	75	75	76	77	77	78	79	79	80	81
83	28.5	69	70	71	71	72	73	73	74	75	75	76	77	78	78	79	80	80	81	82
84	29.0	70	70	71	72	73	73	74	75	75	76	77	78	78	79	80	80	81	82	83
85	29.5	70	71	72	72	73	74	75	75	76	77	78	78	79	80	81	81	82	83	84
86	30.0	71	71	72	73	74	74	75	76	77	78	78	79	80	81	81	82	83	84	84
87	30.5	71	72	73	73	74	75	76	77	77	78	79	80	81	81	82	83	84	85	85
88	31.0	72	72	73	74	75	76	76	77	78	79	80	81	81	82	83	84	85	86	86
89	31.5	72	73	74	75	75	76	77	78	79	80	80	81	82	83	84	85	86	86	87
90	32.0	72	73	74	75	76	77	78	79	79	80	81	82	83	84	85	86	86	87	88
91	33.0	73	74	75	76	76	77	78	79	80	81	82	83	84	85	86	86	87	88	89
92	33.5	73	74	75	76	77	78	79	80	81	82	83	84	85	85	86	87	88	89	90
93	34.0	74	75	76	77	78	79	80	80	81	82	83	85	85	86	87	88	89	90	91
94	34.5	74	75	76	77	78	79	80	81	82	83	84	86	86	87	88	89	90	91	92
95	35.0	75	76	77	78	79	80	81	82	83	84	85	86	87	88	89	90	91	92	93
96	35.5	75	76	77	78	79	80	81	82	83	85	86	87	88	89	90	91	92	93	94
97	36.0	76	77	78	79	80	81	82	83	84	85	86	87	88	89	91	92	93	94	95
98	36.5	76	77	78	80	80	82	83	83	85	86	87	88	89	90	91	92	93	94	95
99	37.0	76	78	79	80	81	82	83	84	85	87	88	89	90	91	92	93	94	95	96
100	38.0	77	78	79	81	82	83	84	85	86	87	88	90	91	92	93	94	95	96	98
101	38.5	77	79	80	81	82	83	84	86	87	88	89	90	92	93	94	95	96	98	99
102	39.0	78	79	80	82	83	84	85	86	87	89	90	91	92	94	95	96	97	98	100
103	39.5	78	79	81	82	83	84	86	87	88	89	91	92	93	94	96	97	98	99	101
104	40.0	79	80	81	83	84	85	86	88	89	90	91	93	94	95	96	98	99	100	101
105	40.5	79	80	82	83	84	86	87	88	89	91	92	93	95	96	97	99	100	101	102
106	41.0	80	81	82	84	85	87	88	89	90	91	93	94	95	97	98	99	101	102	103
107	41.5	80	81	83	84	85	87	88	89	91	92	94	95	96	98	99	100	102	103	104

THI = (Tdb-(0.55-(0.55*RH/100))*(Tdb-58) where Tdb is dry bulb temperature (°F) and RH is relative humidity • °C = (°F – 32)/1.8

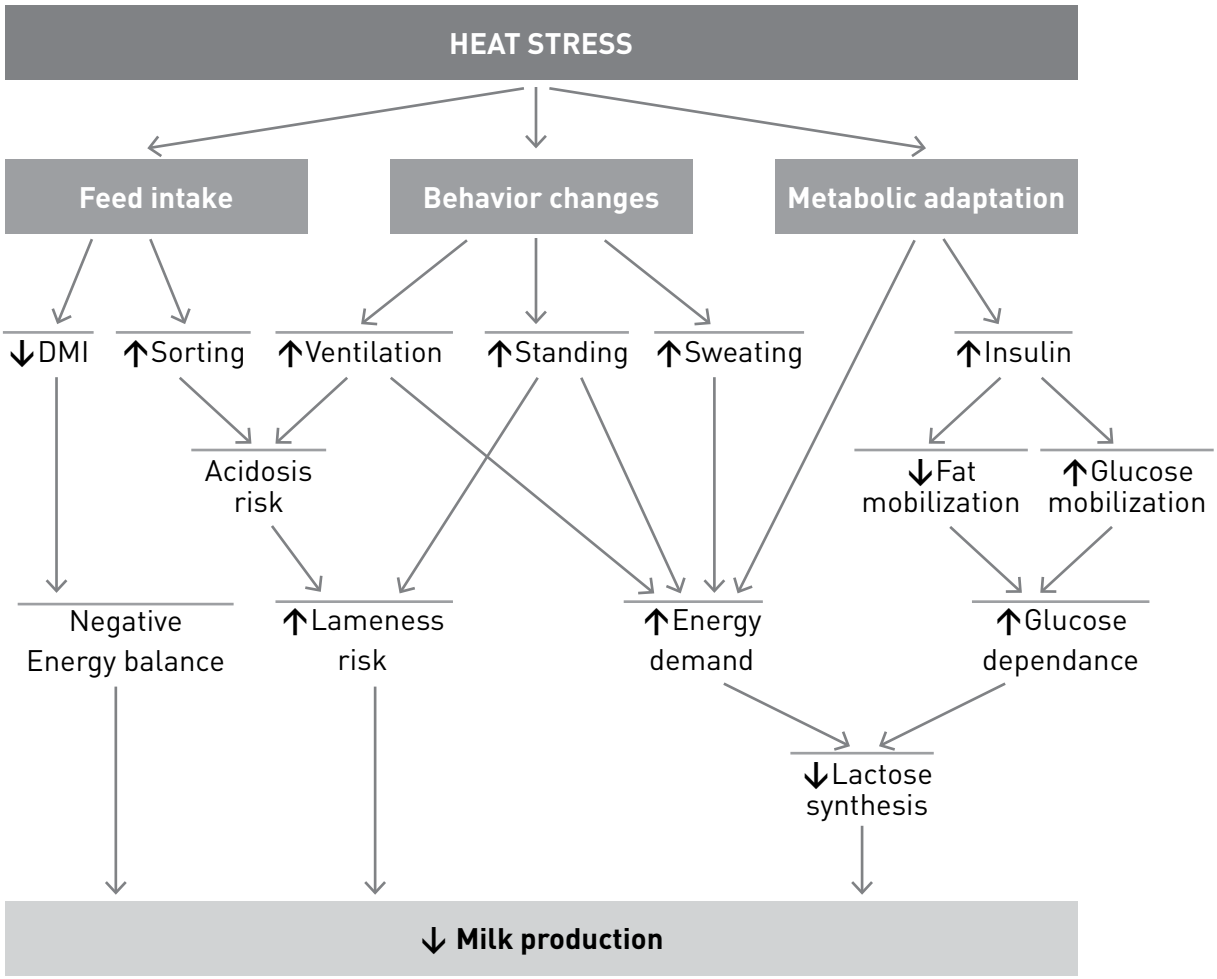
- Stress Threshold: Respiratory rate exceeds 60/min. Milk yield losses begin. Reproduction loses detectable. Rectal temperature exceeds 38.5°C
- Mild-Moderate Stress: respiratory rate exceeds 75/min. Rectal temperature exceeds 39°C
- Moderate-Severe Stress: respiratory rate exceeds 85/min. Rectal temperature exceeds 40°C
- Severe Stress: respiratory rate is 120-140 /min. Rectal temperature exceeds 41°C

CONSEQUENCES OF HEAT STRESS ON DAIRY HERDS

General consequences of heat stress

Typical signs of heat stress are hyperventilation (panting), standing position for longer periods than cows in thermoneutral environmental conditions, and sweating leading to a reduced milk production and reproductive performance, with an increased incidence of lameness (Figure 2).

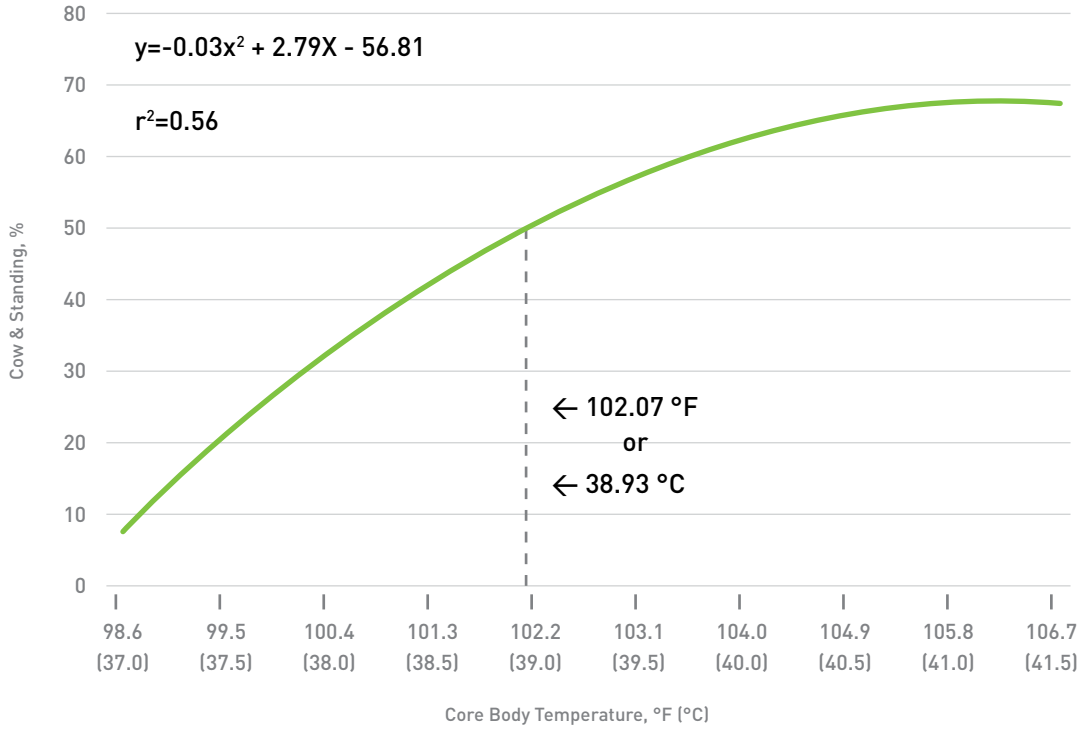
Figure 2: General effects of heat stress on dairy cows



To maximize heat loss regardless of environment, dairy cattle in areas with elevated temperature often stand to increase available surface area for heat dissipation (Figure 3) (Igono et al., 1987; Anderson et al., 2012, Smith et al., 2012). Even a mild increase in ambient temperature can invoke an increase in standing time (Smith et al. 2012). Highest incidence of lameness occurs when cattle stand longer than 45% of the day (Galindo and Broom, 2000), and locomotion scores increase during summer months relative to winter months (Cook et al., 2007). A negative correlation between time spent lying down and incidence of lameness, as well as time spent lying down and temperature humidity index, has also been reported (Privolo and Riva, 2009). This suggests that cattle exposed to higher temperatures are more likely to stand to improve heat dissipation, and are also more likely to experience periods of lameness during the same time frame. Reducing resting time has been reported to reduce milk production (Bach et al., 2008). It was estimated that for each hour of increased resting time, milk production increased by 1.7 kg.

Figure 3: Percent of animals standing in relation to core body temperature

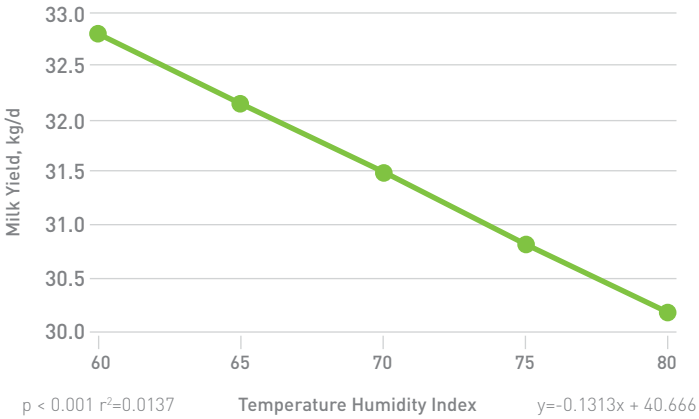
Data are representative of cows in Arizona (n = 56), California (n = 37), and Minnesota (n = 64). (Anderson et al., 2012), (Smith et al., 2012)



Heat stress impact on production

Figure 4: Impact of the increase of THI on milk yield

(Zimbelman et al., 2009)

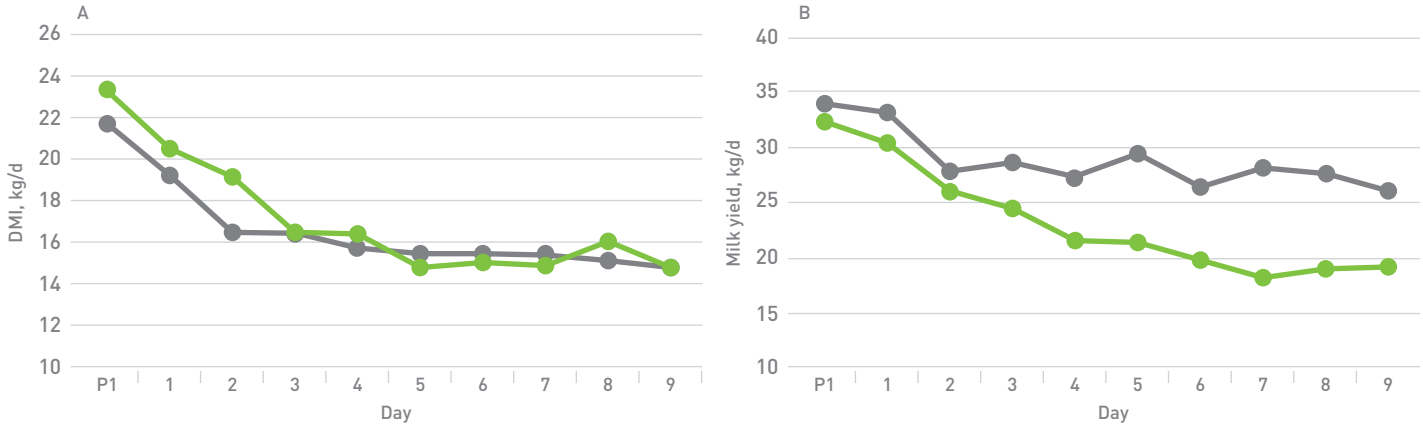


Heat stress is directly correlated with reduced feed intake and reduced milk yield in dairy cows (Figure 4). A reduction in milk yield by up to 35 to 40% is not unusual (West, 2003). Recent climate controlled experiments indicate that milk yield starts to decrease (Zimbleman et al., 2009) at a THI of 68 and are supported by field observations evaluating the THI when cow standing time increases, a classic response to a thermal load (Cook et al., 2007). The lower THI at which cows are thought to become heat-stressed is consistent with the hypothesis that higher producing cows are more susceptible to a thermal load.

Heat-stressed animals reduce their feed intake and this is presumably a survival strategy as digesting, especially in ruminants, and processing nutrients, generate heat (i.e. thermic effect of feed). It has traditionally been assumed that inadequate feed intake caused by the thermal load was responsible for decreased milk production (Fuquay, 1981; DeShazer et al., 2009).

Recent experiments (Wheelock et al., 2010; Baumgard et al., 2011) with heat stressed cows compared with underfed cows consuming the same dry matter as the heat stressed cows demonstrated that reduced feed intake only explains approximately 35 to 50% of the decreased milk yield during environmental-induced hyperthermia (Figure 5). The other 50% are explained by heat stress effect on nutrient partitioning, which corresponds to a coordination of metabolism to unsure a uniform flow of nutrients in support of a physiological state.

Figure 5: Effects of heat stress (HS) or pair-feeding (PF) on dry matter intake (DMI) (A) and milk yield (B) in lactating Holstein cows

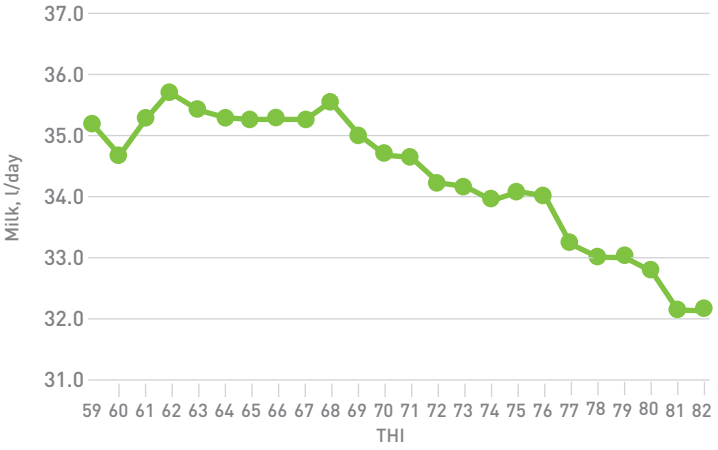


Heat-stressed (HS) cows: Cows submitted to a heat stress (THI = ~80) for 16 hours/day for 9 days

Pair-feeding (PF) cows: Cows, not in heat stress conditions (THI = ~ 64) for 24 hours/day) underfed to consume the same quantity of feed as the heat-stressed cows

The mean value from day 1 to 9 of the thermoneutral ad lib. period (P1) was used as a covariate and is represented by P1 on the X axis. The day 1 to 9 results are from the next period (P2), when cows were either exposed to heat stress (line HS) or pair-fed as described above (group PF)

Figure 6: Effect of temperature humidity index on milk production in Holstein cows
(Bernabucci et al., 2010)



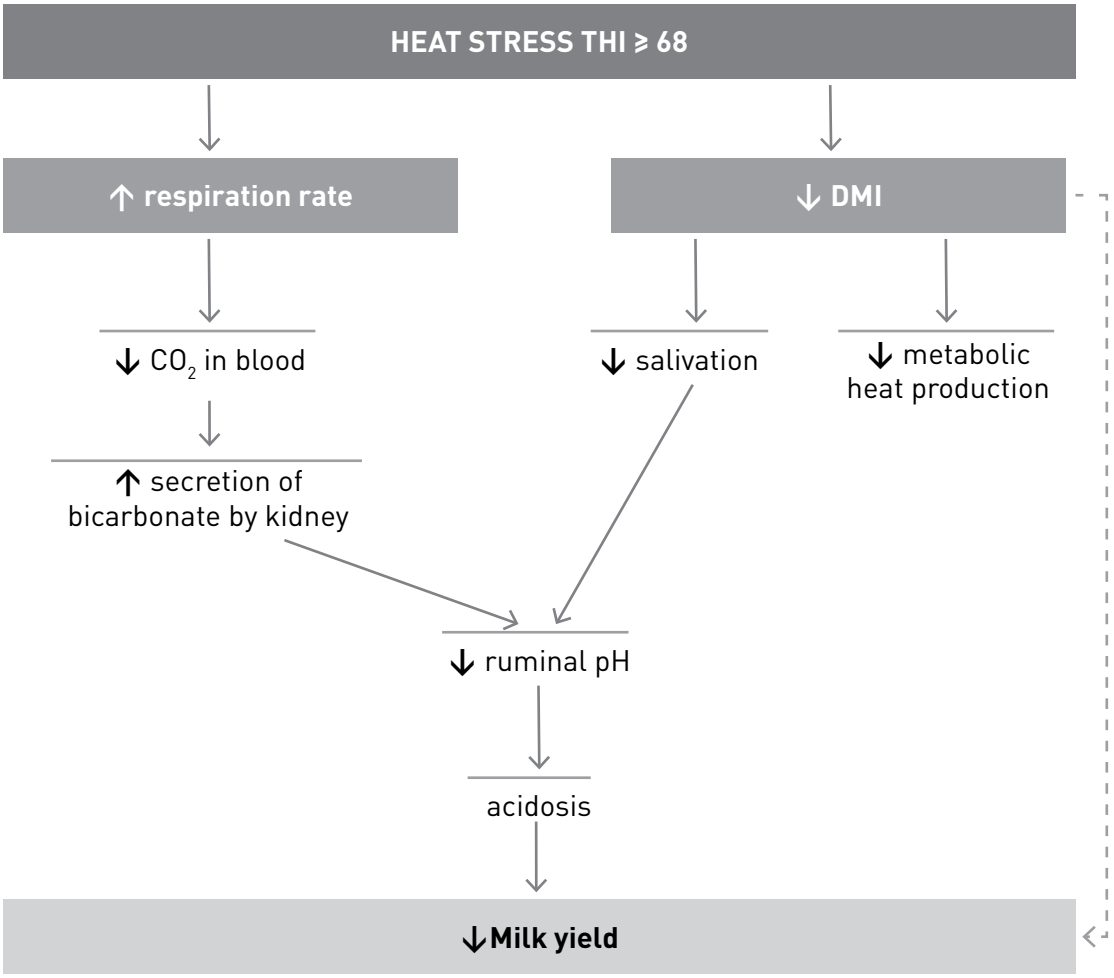
In a 2-year study conducted under field conditions, Bernabucci et al. (2010) found a decrease of 0.27 kg of milk per THI unit only if Holstein cows were exposed to a THI higher than 68 (Figure 6), and similar results were reported by Ravagnolo et al. (2000). Additionally, Bouraoui et al. (2002) in a 2-year study, found a negative correlation between milk yield and daily THI (r^2=-0.76). In particular, milk yield decreased by 0.41 kg per cow per day for each THI unit increase of above 69. Bohmanova et al. (2007) reported different rates of milk production decline per unit of THI, ranging from 0.27 to 0.40 kg and from 0.23 to 0.59 kg in Georgia and Arizona, respectively.

Heat stress: impact on rumen health

One way that cows dissipate heat is via panting and this increased respiratory rate results in enhanced CO2 (carbon dioxide) being exhaled. In order to be an effective blood pH buffering system, the body needs to maintain a 20:1 HCO3- (bicarbonate) to CO2 ratio. Due to the hyperventilation-induced decrease in blood CO2, the kidney secretes HCO3- to maintain this ratio. This reduces the amount of HCO3- that can be used (via saliva) to buffer and maintain a healthy rumen pH. In addition, panting cows drool, and this reduces the quantity of saliva that would normally be deposited in the rumen. Furthermore, due to reduced feed intake, heat-stressed cows ruminate less and therefore generate less saliva. The reductions in the amount of saliva produced and salivary HCO3- content make the heat stressed cow much more susceptible to sub-clinical and acute rumen acidosis (Kadzere et al., 2002).

Heat stress has long been known to adversely affect rumen health (Figure 7).

Figure 7: Effect of heat stress on rumen functions



Due to the reduced feed intake caused by heat stress and the heat associated with fermenting forages, nutritionists typically increase the energy density of the ration. This is often accomplished with extra concentrates and a reduction in forage levels. However, this needs to be conducted with care as this type of diet can be associated with a lower rumen pH. The combination of a "hotter" ration and the cows reduced ability to neutralize the rumen contents (because of the reduced saliva HCO3- content and increased drooling) directly increases the risks of rumen acidosis and indirectly enhances the risk of negative side effects of an unhealthy rumen (i.e. laminitis, milk fat depression, etc.).

A change in the cow's eating behaviour probably also contributes to rumen acidosis. Cows in thermoneutral conditions typically consume 12 to 15 meals per day but decrease eating frequency to 3 to 5 meals per day during heat stress. Also the cows feed with more sorting. The decreased frequency is accompanied by larger meals and thus more acid production post-eating. Furthermore, cows will typically gorge (over eat) the day following a heat wave and this behaviour is well known to cause rumen acidosis (Bernabucci et al., 2010).

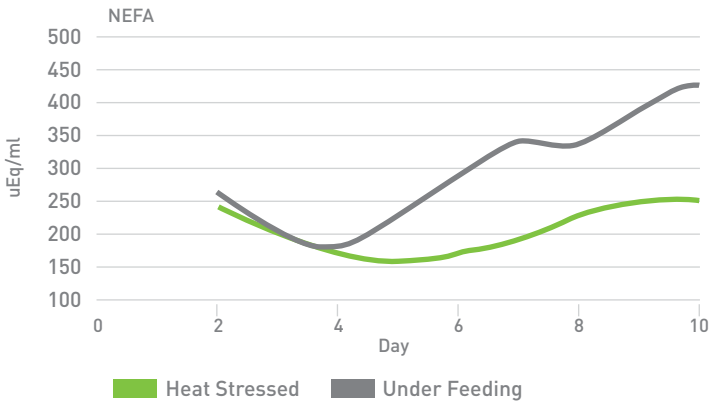
Effect of heat stress on metabolic functions

Maintenance costs

Heat stress is thought to increase maintenance requirements in cattle (Morrison, 1983). The enhanced energy expenditure during heat stress is believed to originate from panting, sweating, and from increased rates of chemical reaction, as predicted by the Arrhenius equation (Kleiber, 1961; Fuquay, 1981). Although it is difficult to quantify, maintenance costs in lactating dairy cattle are estimated to increase by as much as 25% during heat stress and some suggest it may be greater than 30% (Fox and Tylutki, 1998).

Lipid metabolism

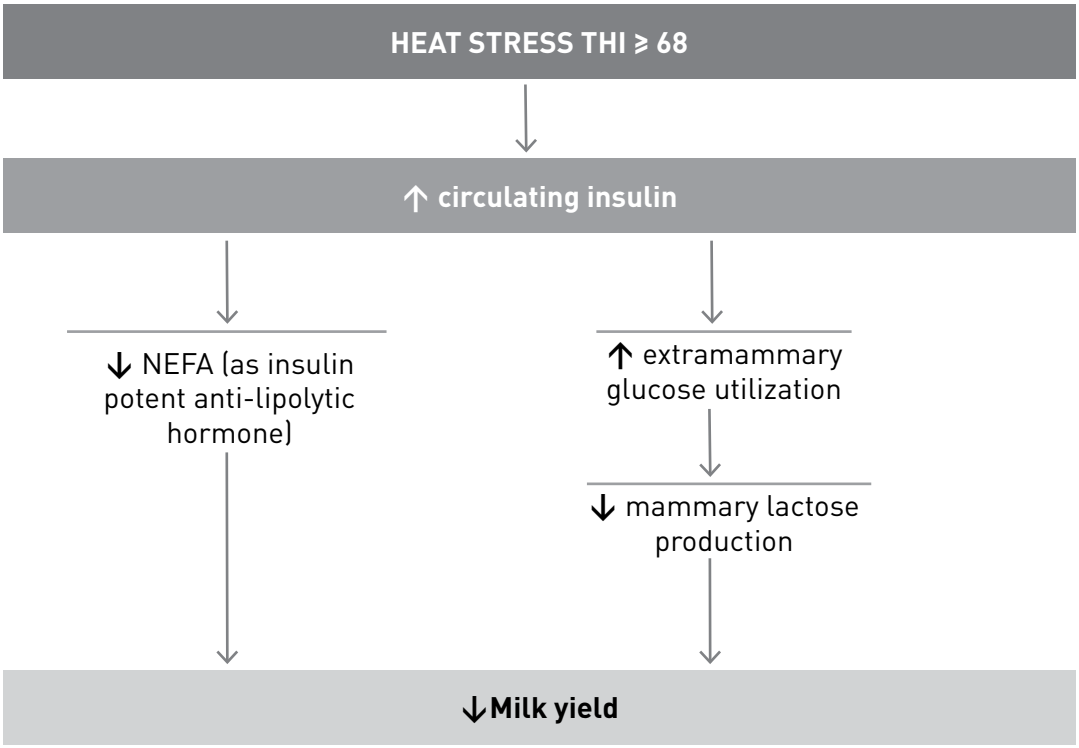
Figure 8: Effects of heat stress and pair-feeding in thermoneutral conditions on circulating non-esterified fatty acids (NEFA) in lactating Holstein cows
(Adapted from Wheelock et al., 2006)



During Heat Stress, because of the reduction of feed intake, the dairy cows are in negative energy balance (NEBAL). Several studies demonstrated that despite the NEBAL, basal plasma non-esterified fatty acid (NEFA) concentrations are typically reduced in heat-stressed cattle, especially when compared with pair-fed thermoneutral controls (Figure 8) (Wheelock et al., 2010). Pair-fed cows were restricted to the same DMI as the heat stressed cows, but without being exposed to heat stress. In this condition, as they were underfed they developed a negative energy balance (NEBAL). In this scenario the pair-fed cows reacted with increased NEFA from activated fat mobilization to meet the energy demands for body maintenance and milk production. The heat-stressed cows on the other hand did not respond with fat utilization and showed no significant increase in NEFA.

Also heat stress increases adipose tissue lipoprotein lipase (Christon, 1988), indicating adipose tissue of hyperthermic animals has an increased capacity to liberate fatty acids from circulating triglycerides and use these for storage. So instead of utilizing fats for energy as the underfed cows, heat-stressed cows actually stored more fats (Figure 9).

Figure 9: Effects of heat stress on lipid metabolism



Carbohydrate metabolism

As is observed in several studies of heat-stressed cows, fat mobilization and utilization as an energy source during heat stress is suppressed. Thus heat-stressed cows rely mainly on glucose as an energy source for maintenance and milk production.

Basal insulin values gradually increase in lactating heat-stressed cows (Wheelock et al., 2010), and this has also been confirmed in growing heat-stressed calves (O'Brien et al., 2010).

In addition, heat-stressed cows and calves have an increased insulin response to a glucose tolerance test (Figure 10) (O'Brien et al., 2010; Wheelock et al., 2010).

The increased insulin may be an essential part of the heat stress adaptation mechanism. For example, diabetic humans are more susceptible to heat-related illness and death (Kovats and Hajat, 2008).

Insulin is a potent regulator of both carbohydrate and lipid metabolism and may play an important role in mediating heat stress regulation of post-absorptive nutrient partitioning. Insulin stimulates glucose uptake and is likely responsible for the heat-induced hypoglycemia.

This occurs despite an increase in intestinal glucose absorptive capacity (Garriga et al., 2006) and enhanced renal glucose rebsorptive ability (Ikari et al., 2005). Insulin is also a potent anti-lipolytic hormone (Vernon, 1992) and may explain why heat-stressed animals do not mobilize adipose tissue triglycerides.

Figure 10: Effects of heat stress (HS) and pair-feeding in thermoneutral conditions (PF) on the insulin response to a glucose tolerance test (GTT) in growing Holstein bull calves
(Adapted from O'Brien et al., 2010)

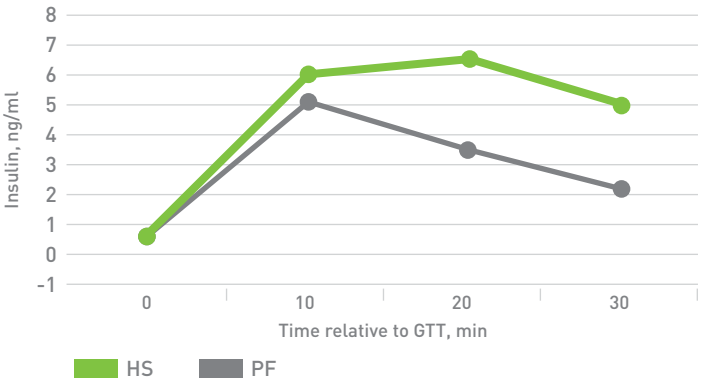
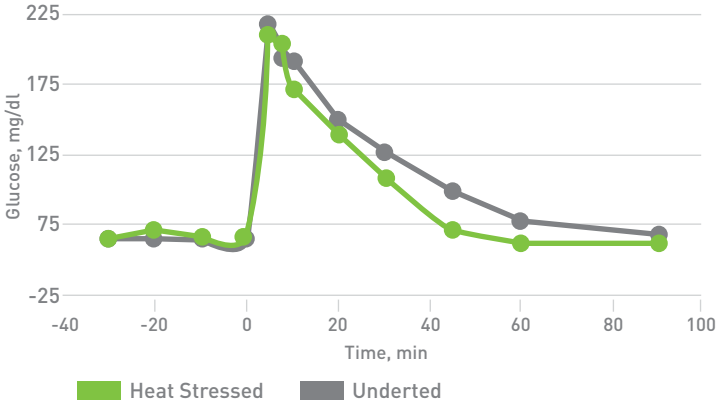
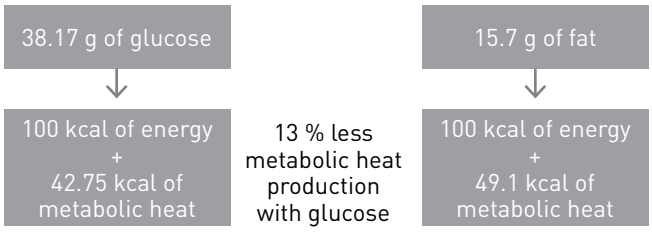


Figure 11: Effects of heat-stressed and under-fed (pair-feeding in thermoneutral conditions) cattle on plasma glucose response to a glucose challenge
(Adapted from Wheelock et al., 2006)



In addition, using an IV glucose tolerance test, Wheelock et al., 2006 (Figure 11) demonstrated that glucose disposal (rate of cellular glucose entry) is greater in heat stressed cows compared to thermoneutral pair-fed cows.

Figure 12: Bioenergetics and metabolic heat production from oxidizing either glucose or fatty acids (i.e. stearic acid), assuming 100 kcal of energy was needed and assuming 38 ATP/mole of glucose



The mammary gland requires glucose to synthesize milk lactose, and lactose production is the primary osmoregulator and thus determinant of milk yield. However, in an attempt to generate less metabolic heat, the body (primarily skeletal muscle) appears to utilize glucose at an increased rate. As a consequence, the mammary gland may not receive adequate amounts of glucose and thus mammary lactose production and subsequent milk yield are reduced. This may be the primary mechanism which accounts for the additional reductions in milk yield that cannot be explained by decreased feed intake.

Heat-stressed animals secreted approximately 200 to 400 g/d less milk lactose than did pair-fed thermoneutral controls (Rhoads et al., 2009; Wheelock et al., 2010).

Cellular response to heat stress

Exposure to increased ambient temperatures can result in significant alterations and damage at the cellular level. Many intracellular molecular structures rely on a variety of relatively weak interactions for stabilization, and these interactions are easily disrupted by changes in the microenvironment (i.e., increases or decreases in temperature and pH). Heat can negatively affect cell components directly, such as unfolding and subsequent aggregation of proteins (Caspani et al., 2004, Roti Roti, 2008). Protein synthesis appears to be particularly impaired by heat.

The cellular response to a heat load includes activation of transcription factors such as heat shock factors (HSFs) (Pirkkala et al. 2001), expression of proteins associated with acute homeostatic response such as heat shock proteins (HSPs), and altered gene expression (Collier et al. 2008).

Of particular importance to cell survival during hyperthermia are Heat Shock Proteins (HSPs). Members of this protein family are ubiquitously expressed across species and are present at low levels in cells under normal conditions, but their levels increase greatly, but transiently, on a cellular insult (Moseley et al., 1997). HSPs bind to unfolded or misfolded proteins and help restore their native conformation (Bouchama et al., 2002).

In addition to their role in protecting cells from heat-induced protein misfolding, HSPs may enhance insulin function. Several studies demonstrate that HSPs are important for proper insulin function and suggest that strategically manipulating the insulin-HSP axis may improve human and animal health and productivity during heat stress (Kavanagh et al. 2011).

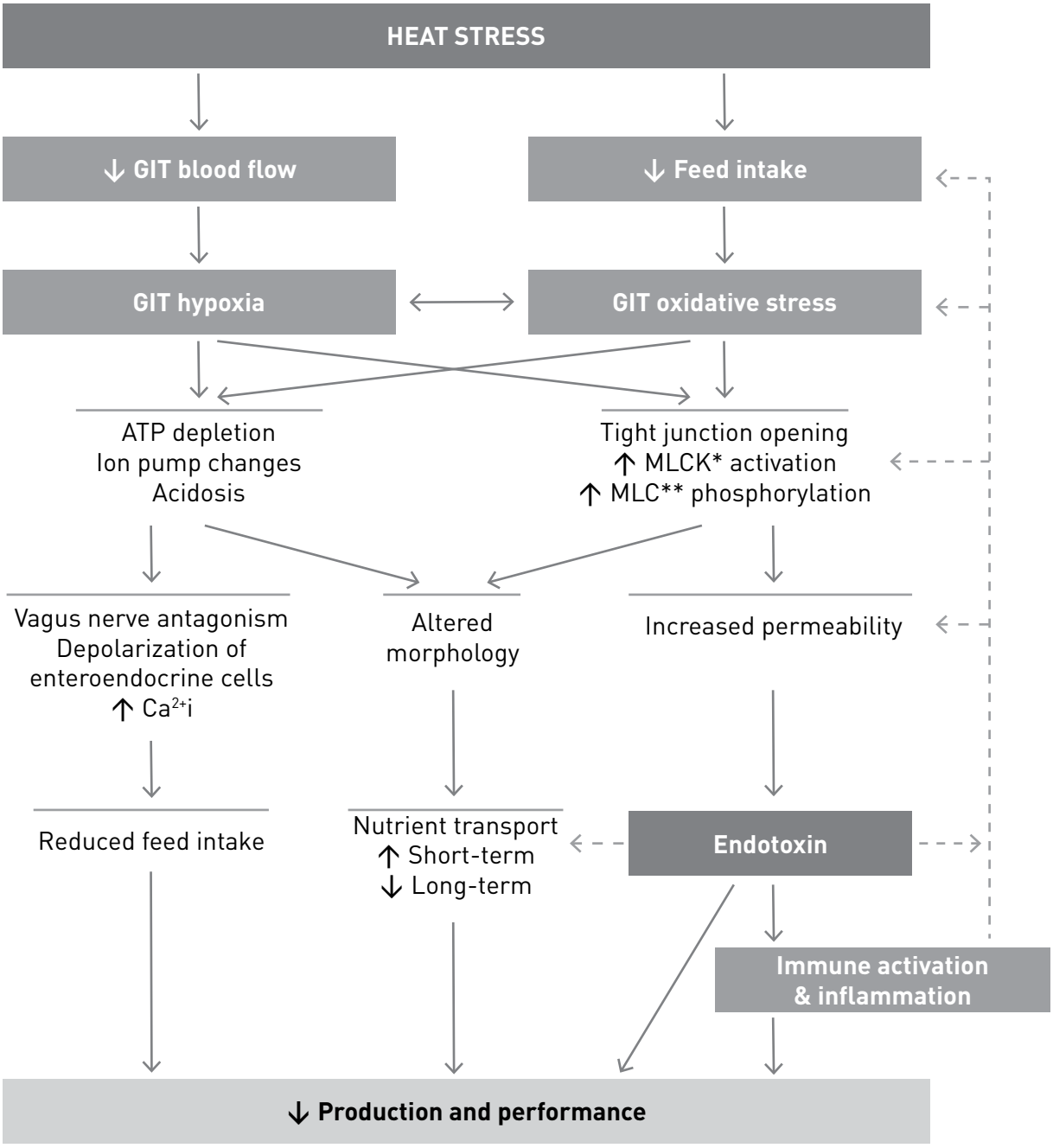
Niacin supplementation may improve heat tolerance through elevation of cellular heat shock proteins and peripheral vasodilation (Zimbelman et al. 2008).

Leaky gut and inflammation during heat stress

The mechanisms that alter nutrient partitioning during heat stress are not entirely clear, but they are certainly mediated by the effects of heat stress on gut health and function (Stoakes et al., 2014).

During heat stress, blood flow is diverted towards the periphery to dissipate heat through the skin (Lambert et al., 2002). The small intestine is the first to suffer from reduced blood flow to the viscera, and enterocytes are particularly sensitive to hypoxia and nutrient deficiency. This impairs tight junctions and reduces intestinal barrier function (Lambert et al., 2002; Pearce et al., 2013b), which causes lipopolysaccharides (LPS) to be translocated from the intestines to the systemic circulation (Hall et al., 2001; Pearce et al., 2013b). LPS are endotoxins found in Gram-negative bacteria and, when translocated to the systemic circulation, they induce a systemic immune reaction (Andersen et al. 1994). This translocation stimulates the release of proinflammatory cytokines, such as interleukin-1 (IL-1), IL-6, and tumor necrosis factor-α (TNF-α) (Gabay C. et al., 1999). Translocation of endotoxins into the bloodstream causes metabolic changes and induces a systemic inflammatory response (Ametaj et al., 2009).

Figure 13: LPS-induced metabolic changes (adapted from Baumgard et al., 2011)

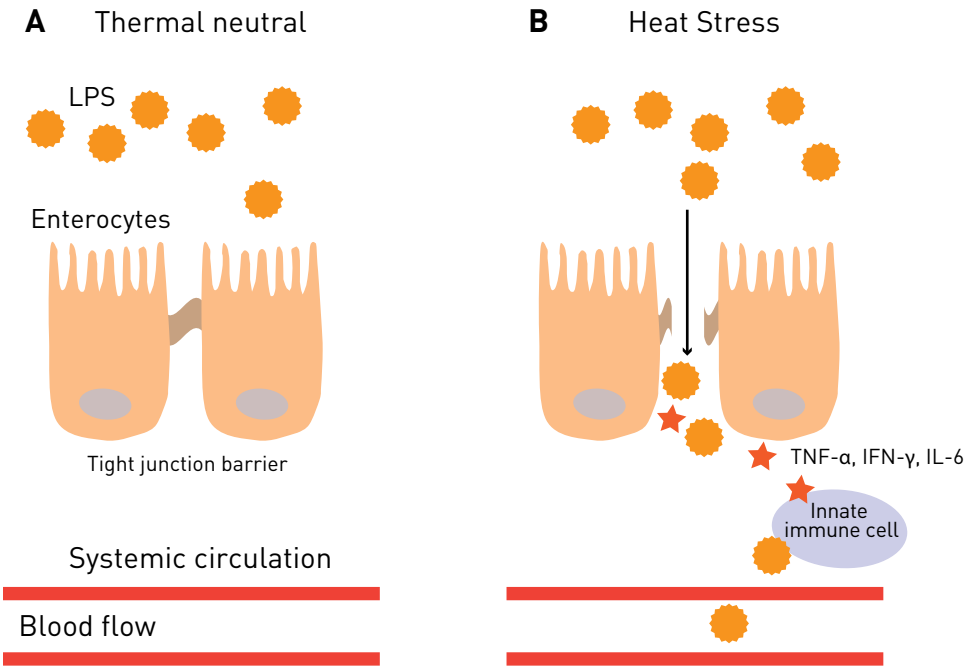


*MLCK: Myosin light-chain kinase
**MLC: Myosin light-chain

High levels of LPS in dairy cows also increase blood cortisol (Gross et al., 2015).

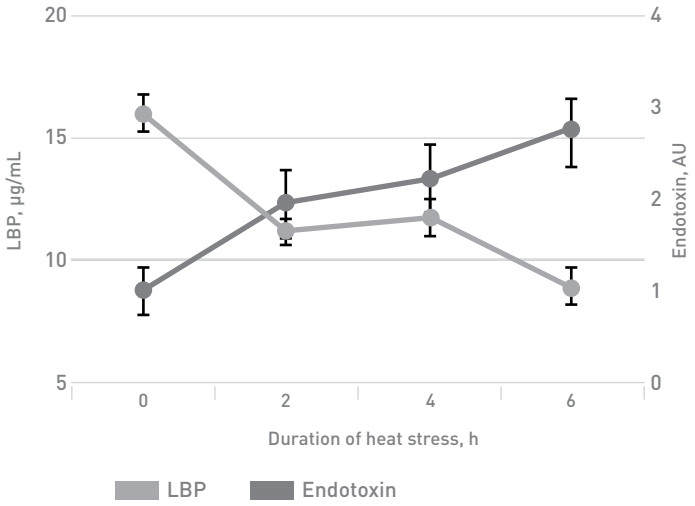
During heat stress, cows are also fed a more concentrated diet to cope with the negative energy balance. High-grain ruminal acidosis in dairy cows increases *E. coli* LPS in both the GIT and the systemic circulation and causes systemic inflammation (Khafipour et al., 2009b).

Figure 14: Effect of heat stress on tight junctions, proinflammatory cytokines and, therefore, intestinal permeability



Enterocytes are particularly sensitive to heat stress. During heat stress, intestinal integrity is compromised and LPS bacterial endotoxin are translocated to the systemic circulation causing inflammation.

Figure 15: LPS-binding protein (LBP) and endotoxin levels during heat stress



During heat stress, LPS levels in the blood increase and LBP levels decrease. (Pearce at al., 2014)

Systemic inflammation caused by bacterial endotoxin translocation during heat stress raises body temperature and increases levels of proinflammatory cytokines. The changes in insulin, glucose, and cortisol levels in ruminants during heat stress are similar to the changes seen in systemic inflammation. This suggests that some of the negative effects of heat stress are linked to the inflammatory reactions caused as an immune response to the translocated endotoxins. Inflammation reduces performance and increases the demand for energy, which is supplied to the immune system in the form of glucose, leaving less energy available for lactose synthesis and milk production. Procedures to reduce systemic inflammation should be combined with management and nutritional strategies to minimize production losses during heat stress. These may include improving intestinal integrity, reducing LPS translocation, and modulating the immune system for more effective reaction to the endotoxin challenge.

Effect of heat stress on oxidative stress

Heat stress is thought to affect oxidative status in dairy cows, which can affect health and performance (Bouchard et al., 1999).

Heat stress has been demonstrated to have a negative effect on intestinal integrity in dairy cows, and it may lead to LPS translocation and systemic inflammation. Inflammatory processes are endogenous sources of reactive oxygen species (ROS). High levels of ROS cause cell damage, reduced immune responses, fertility problems, and mastitis. The best known causes of oxidative stress in veterinary medicine are metabolic and inflammatory events, and environmental factors (heat stress and nutrition) (Pietro Celi, 2015).

HEAT STRESS MANAGEMENT

High risk groups during heat stress

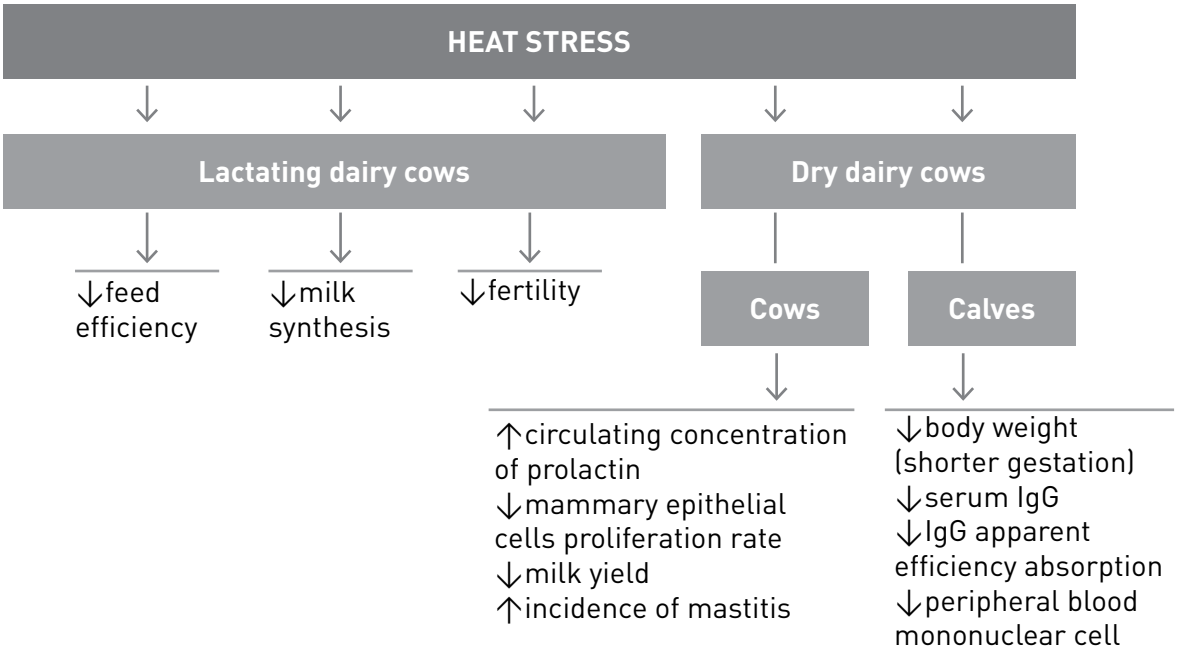
Milk cows under heat stress are frequently studied, as there is a loss of milk and this affects farm profit directly. However, it is important not to overlook other groups in the farm (Figure 16):

Dry cows Research from the University of Florida (Tao et al., 2011) showed that cows housed in naturally ventilated barns with fans and sprinklers programmed to cool dry cows once temperatures reached 21 °C, produced 5 kg more milk per day in early lactation. Heat stress during the dry period compromises mammary gland development before parturition, which decreases milk yield in the subsequent lactation.

Nursery calves University of Arizona researchers (O'Brien et al., 2010) reported a 12% decrease in starter intake in heat-stressed calves (29.4 to 40 °C) vs. control calves housed at 19.4 °C. To increase intake during heat stress, research at the University of Washington (Moore et al., 2012) reported that when calf hutches were elevated, internal hutch temperatures were cooler than external temperatures, hutch carbon dioxide levels were lower and respiratory rates were lower, particularly during the afternoon.

Fresh cows Heat stress reduces feed intake and therefore reduces energy intake, compromising cow health and performance in fresh cows. Iowa State University (Baumgard et al., 2011) housed cows in environmental chambers at either 20 °C (control) or a consistent cycle ranging from 29.4 to 38.9 °C (heat-stressed). Heat-stressed cows consumed 28% less dry matter intake and had a 29% reduction in milk yield. This dramatic effect of heat stress on energy intake would likely be additive in fresh cows that are already in negative energy balance often for the first 40 days of lactation.

Figure 16: General effects of heat stress on lactating and dry dairy cows



Environmental strategies

- Roof insulation for summer housed herds can reduce solar penetration dramatically.
- Increase the amount of available water. One water point is recommended per 20 heads. This may not be enough during extreme heat stress when animals will increase their water intake. When the temperature reaches dangerous levels, an additional water source is required near the feeding area.
- Increase the airflow by installing fans or open the sides of the barn at low level to allow a flow of air to pass through the recumbent cows.
- Adequate number of fans should be spaced at about 3.5 - 3.7 M high along the length of the freestall barn. Fans should be installed so that they are spaced longitudinally down the shelter with a spacing of no more than 10 times their blade diameter. The recommended distance between fans is 9.1 M for 91 cm fans and 12.2 M for 120 cm fans.
- Misters are another solution that can reduce heat stress. It is important to take into consideration the type of the barn and the floor to avoid creating mud where the cows are lying. A combination of misters with fans can even further decrease the temperature in the barn. Misters and fans are more beneficial if placed over the lying area instead of the feeding area, as the cows spend more time lying down and ruminating than eating.

Feeding strategies

- Alter feeding times to deliver feed during the coolest part of the day.
- Increase the number of daily feeding times (mix smaller loads) to keep feed in the trough cool.
- Make sure high moisture feed ingredients (distillers and corn gluten) are fed before they undergo secondary fermentation (heating).
- Minimize total mixed ration (TMR) discrimination by increasing feed presentation and evaluating particle size.

- Avoid mist spray settling onto feed. Excessive wetting of the TMR can reduce palatability and accelerate bacterial growth in the trough reducing feed quality. Remove all refused feed daily before any new TMR is fed.
- Plan on lower dry matter intake during heat stress and work with a nutritionist to adjust diets accordingly. Increase the nutrient density of the diet to reflect actual dry matter intake, which is typically reduced during heat stress.
- Add yeast probiotic to the diet to increase DMI, milk yield, and reduces negative impact of heat stress.

EFFECT OF YEAST SOLUTIONS ON HEAT STRESS MANAGEMENT

Actisaf® yeast probiotic improves fibre digestion and stabilizes the rumen environment. In heat-stressed dairy cows, supplementation with live yeast reduces respiratory rate and increases milk production and milk solids.

Actisaf® is a rumen-specific live yeast which acts as a probiotic and, in many studies, has shown significant improvement in rumen function:

- Improves fibre digestion with increased production of acetate
- Reduces the rumen reduction potential (E_h), which is associated with a well-balanced and active rumen microflora
- Balances the pH reducing the incidence of rumen acidosis

Actisaf® effects in high producing cows (>40 l) in heat stress

Materials and methods

In a study (Moallem et al., 2009), forty-two Holstein dairy cows (14 primiparous and 28 multiparous) in heat stress conditions were fed either a control lactation diet or were supplemented with 1 g of Actisaf® per 4 kg of dry matter consumed (corresponding to 6 g of Actisaf®/cow/day). The cows were 114 (±54) days in milk and were group housed in shaded loose pens with adjacent outside yards, equipped with a real-time electronic individual feeding system. After 10 days of adaptation to the diets and the electronic feeding system, the cows were assigned to 2 groups of 21 cows each to begin treatment. The temperature humidity index (THI) in the morning (06:00 am) averaged 69.4 ± 3.5, and the THI in the afternoon (16:00 pm) averaged 79.3 ± 2.1. The temperature, relative humidity, and THI values during the study period were relatively stable. The cows were exposed to heat stress during the entire time of the trial.

Results

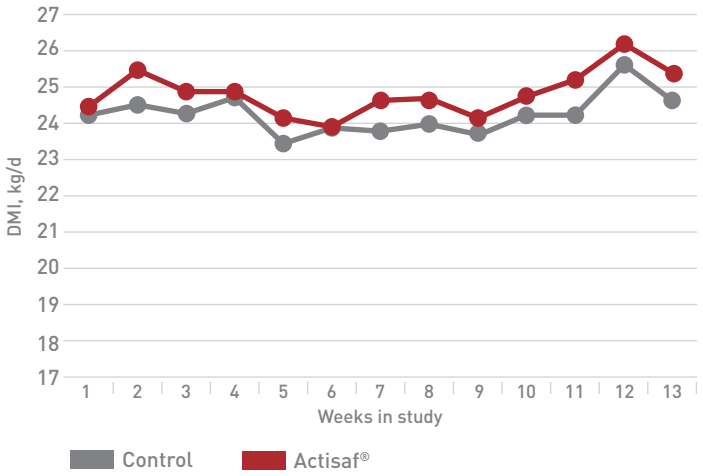
The results are presented in the following table:

Indicator	Control	Actisaf®	p <
DMI, kg/d	24.1	24.7	0.0001
Milk yield			
Milk, kg/d	36.3	37.8	0.007
FCM 4%, kg/d	32.8	34.8	0.0001
Milk solids g/d			
Fat g/d	1237	1368	0.03
Protein g/d	1172	1220	0.12
Lactose g/d	1810	1887	0.15
Feed efficiency			
FCM 4% per kg of DMI	1.36	1.41	0.03

FCM: Fat Corrected Milk; DMI: Dry Matter Intake

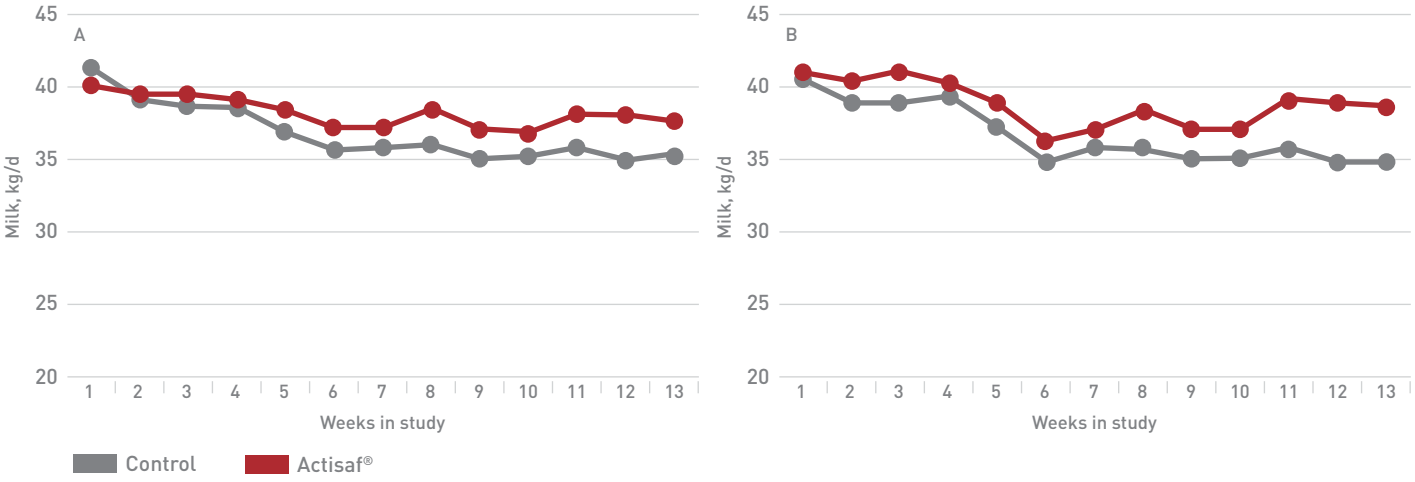
The average daily DMI in the Actisaf® group was greater by 0.6 kg per day (2.5%) than in the control ($p < 0.0001$) (Figure 17).

Figure 17: Impact of the addition of Actisaf® in the feed of heat-stressed cows on dry matter intake (DMI) (Moallem et al. 2009)



The average daily milk production was 1.5 kg/day greater in the Actisaf® group than in the control group (4.1%; $p < 0.007$). Also the fat corrected milk at 4% yield was 6 % greater in the Actisaf® group than in the control ($p < 0.03$) (Figure 18).

Figure 18: Impact of the addition of Actisaf® in the feed of heat-stressed cows on milk yield (A) and fat-corrected milk (FCM) at 4% (B) (Moallem et al. 2009)



Feed efficiency, as defined by production of fat-corrected milk at 4 % from DMI was also greater (3.7%) in Actisaf® group than in the control ($p < 0.03$).

The rumen pH values tended to be higher in the Actisaf® cows than in the control; 6.67 and 6.54, respectively (pooled SEM = 0.03; $p < 0.1$).

Conclusion

In heat stressed cows, the addition of Actisaf® in the feed of cows allowed a reduction of the negative impact of heat stress by increasing the dry matter intake and milk yield while improving the feed conversion ratio. Actisaf® limits the drop of pH during the heat stress period, lessening the risk of ruminal acidosis.

Actisaf® effects in mid-range milk-producing dairy cows (~30 kg) in heat stress

The benefits of adding Actisaf® to the diet of cows in heat stress conditions have been confirmed in another trial performed in Brazil (Salvati et al., 2015).

Materials and methods

The THI was above 68 for 75% of the time, and above 72 for 40% of the time during the trial. The maximum THI reached was 85.1 with a maximum temperature of 36 °C and a maximum humidity of 98.5%.

In this trial twenty-eight Holsteins were housed in a sand-bedded tie stall barn with fans and high pressure sprinklers. The cows (207 ± 87 days in milk) received a standardization diet for 14 days as an adaptation period. At the end of the adaptation period, cows were paired blocked based on calving order and milk yield, and assigned to a treatment for 10 weeks.

Treatments were:

- Actisaf® group: basal diet plus Actisaf® at 10g/cow/day top dressed

- Control group: fed with the same basal diet.

Results

The results are presented in the following table:

Parameters	Control	Actisaf®	p <
DMI, kg/d	19.0	19.5	0.53
Milk, kg/d	25.4	26.7	0.03
ECM, kg/d	23.0	24.4	0.05
4% FCM, kg/d	21.7	23.1	0.05
Fat, kg/d	0.777	0.824	0.09
Protein, kg/d	0.801	0.828	0.06
Solids, kg/d	2.921	3.062	0.05
Milk/DMI, kg/kg	1.34	1.37	0.77

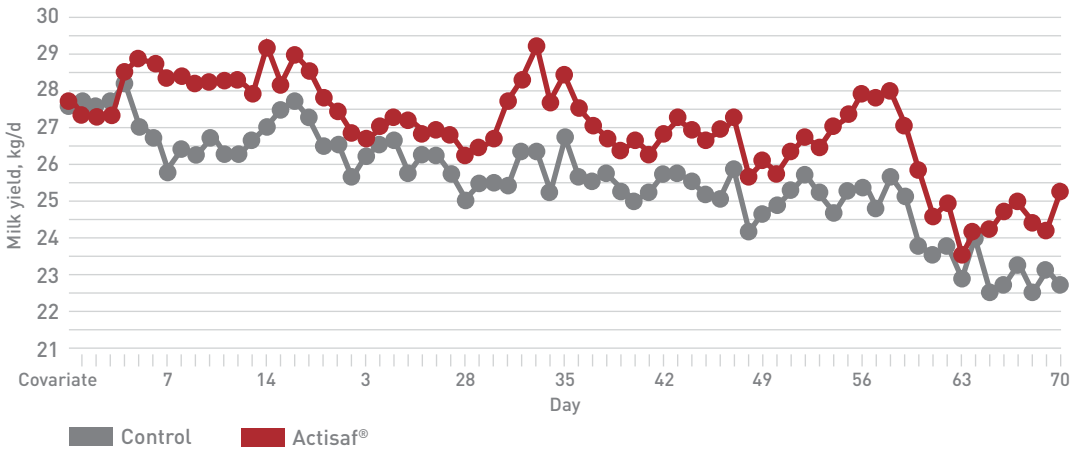
ECM: Energy Corrected Milk; FCM: Fat Corrected Milk; DMI: Dry Matter Intake

The authors have observed similar results from the previous trial with DMI increase of 0.5kg per day or 3.3%).

The milk yield was increased by 1.3 kg or 5.12%. Once more, an increase in daily fat and protein production was observed by the authors, which again confirmed the Actisaf® benefits from the previous study.

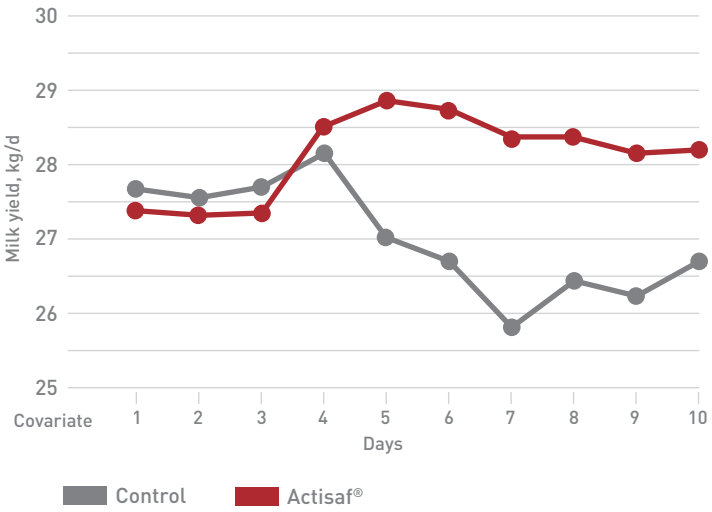
In addition, the results were consistent during the whole study, showing a constant increase in the milk production (Figure 19).

Figure 19: Impact of the addition of Actisaf® in the feed of dairy cows on milk yield



This result showed a fast response to Actisaf® after 4 days of Actisaf® supplementation (Figure 20).

Figure 20: Milk yield in the first 10 days after including Actisaf® in the diet



Feed efficiency was also improved as demonstrated in the previous study: Milk production per kg of DMI were 1.34 / 1.37 for the control and treated group respectively.

The average rumen pH in the Actisaf® group was also higher as expected: pH 6.35 for the control and pH 6.44 for the Actisaf® group.

CONCLUSION

This trial confirmed the benefits of the addition of Actisaf® in the feed of dairy cows, during a heat stress period. Actisaf® improved the milk production and daily milk solids production.
During these and other trials, the authors have also evaluated many other parameters in order to better understand and explain the mode of action of Actisaf® during heat stress.

Actisaf® mode of action during heat stress

Actisaf® and rumen function

Adding Actisaf® to the diet of dairy cows improves rumen function by regulating the pH and reducing the redox potential (Eh). The rumen operates best at pH levels of 6 and above. At levels below pH 6, there is a reduction in the digestion of fibre and in feed conversion. The ability of yeast probiotic Sc 47 to regulate rumen pH has been demonstrated in a study comparing it with the use of 150 g of sodium bicarbonate in dairy cows fed an acidosis-inducing high starch diet (Figure 21).

Actisaf® at 5g/cow/day shows similar results to 150g of sodium bicarbonate in its capability to maintain the rumen pH above 6 after feeding.

Actisaf® regulates the rumen pH by stabilizing and promoting the beneficial rumen microflora, as assessed by the reduction potential (Eh) ,which shows better rumen environment for anaerobic bacterial activity (Figure 22).

Figure 21: Comparison of 3 types of diet on their effect on rumen pH
(Marden et al., 2008)

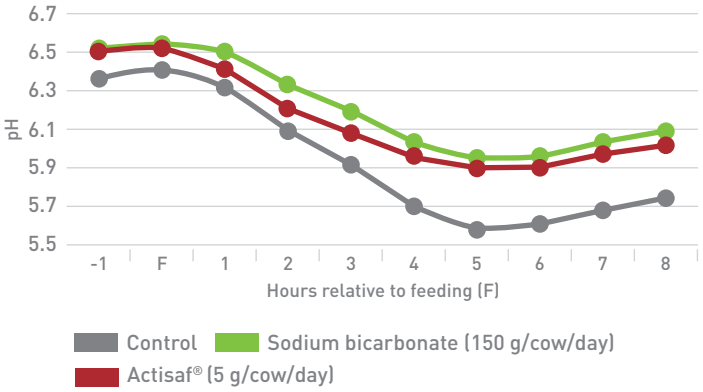
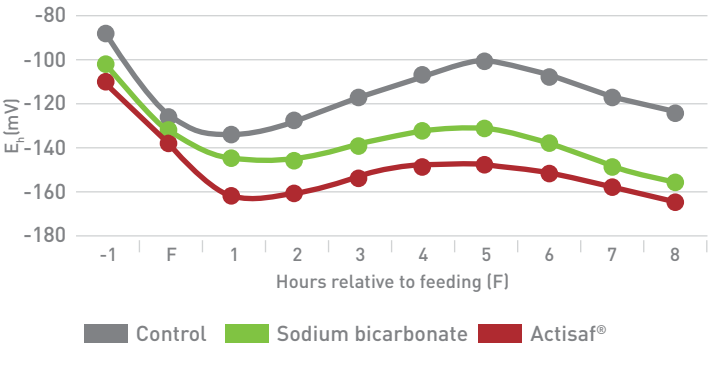


Figure 22: Comparison of 3 types of diet on their effect on rumen reduction potential (Eh)
(Marden et al., 2008)



This is a clear indication of better rumen environment. The ability of yeast probiotic to promote and stimulate the beneficial rumen microflora activity explains the increased concentration of volatile fatty acids (VFAs) and decrease of lactic acid. This is achieved by promoting the growth and activity of lactic acid utilizing bacteria which transform the lactic acid to propionate, thus reducing the risk of ruminal acidosis (Figure 23).

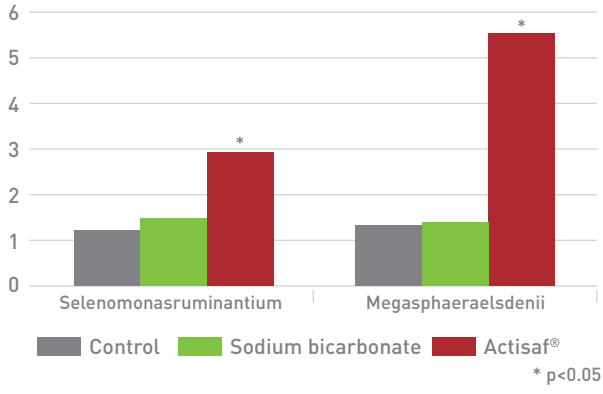
The authors observed an increase in numbers of fibre digesting bacteria after adding Actisaf®, which leads to better feed conversion and more acetate available as energy source for the cow.

The evaluation of the effect of Actisaf® on the microbiota revealed that some bacterial groups were more affected than others (Figure 24). The relative abundance of the lactate-utilizing bacteria (Megasphaera and Selenomonas) increased with yeast supplementation as well as the fibrolytic groups (Fibrobacter and Ruminococcus). (Pinloche et al., 2013)

Figure 23: Volatile Fatty Acids (VFAs) profile in control, Actisaf® and bicarbonate supplemented cows
(Marden et al., 2008)

(mM)	Control	Bicarbonate	Actisaf®
Total VFA	85.3[a]	95.3[b]	99.4[b]
Acetate	53.2[a]	60.8[b]	59.1[b]
Propionate	18.0[a]	20.0[a]	25.8[b]
Butyrate	10.6	10.1	10.2
Lactate	16.5[b]	12.2[b]	5.4a

Figure 24: Abundance of lactate utilizing bacteria in control, Actisaf® and bicarbonate supplemented cows
(Pinloche et al., 2013)



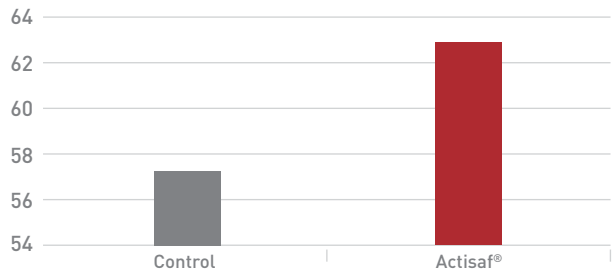
* p<0.05

Actisaf® and energy balance

Adding Actisaf® to the diet of heat stressed cows also changes some important blood parameters like glucose (Figure 25), which have a direct impact on physiology and metabolism of the cows during heat stress.

During heat stress, as previously explained, glucose is the major source of energy. Glucose is used by the mammary gland to produce lactose which controls the milk production. Increased blood glucose in heat stressed cows could explain not only an increased milk production but also better health due to more available metabolic energy for maintenance.

Figure 25: Plasma glucose in heat stressed cows after feeding Actisaf®
(Salvati et al., 2015)



Actisaf® and heat regulation

Niacin, also known as nicotinic acid or vitamin B3, is known to increase blood flow to the epidermis in humans. Consequently, recently, it was suggested that a higher level of niacin should increase vasodilatation, thus cooling the dairy cow more efficiently in case of heat stress. At animal level, some niacin comes from feed but most is from microbial synthesis. In addition, plasma, total blood, red blood cell and leukocyte-niacin concentrations decreased during heat-stress (Rungruang et al., 2014).

Niacin is synthesized by the microflora in the rumen of ruminants. Actisaf®, by reducing the redox potential and balancing the pH, promotes the growth and activity of rumen microflora, which leads to increased synthesis of niacin (Figure 26) which is involved in heat dissipation through the skin and possibly elevation of cellular heat shock proteins (as mentioned above).

Figure 26: Plasma niacin in heat stressed cows after feeding Actisaf®
(Salvati et al., 2015)

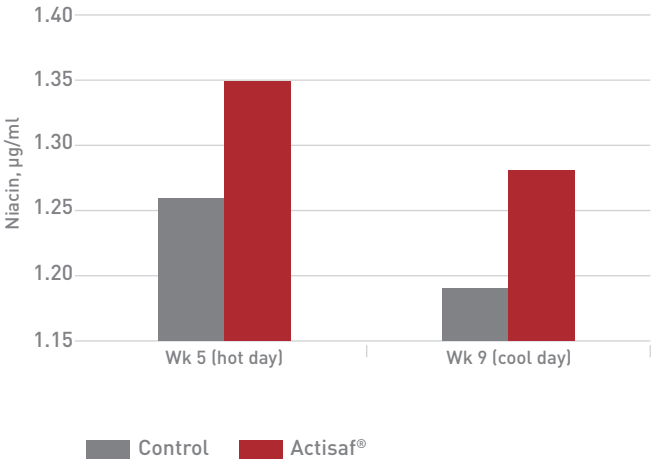
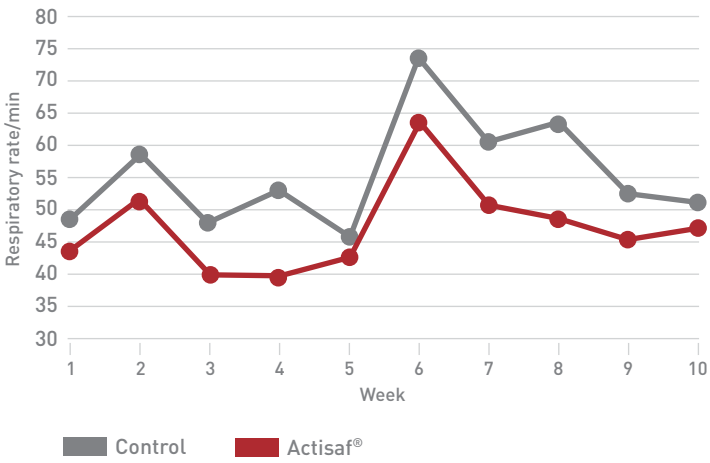


Figure 27: Impact of the addition of Actisaf ® on respiratory rate of dairy cows (p treat = 0.02) (Salvati et al., 2015)



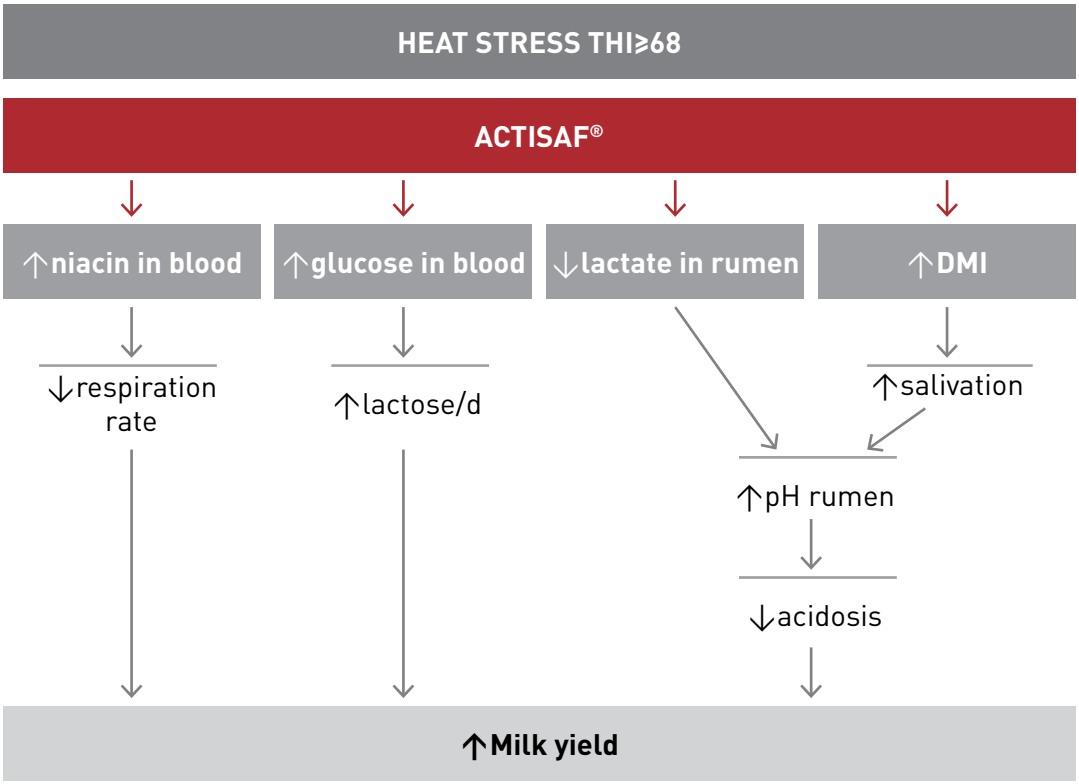
The elevated plasma levels of niacin can also explain the decrease of respiratory frequency in Actisaf® supplemented cows during heat stress, which indicates greater comfort (Figure 27).

Conclusion

Through stabilizing the rumen environment and supporting microflora activity, Actisaf® improves rumen function which leads to improved metabolism.

Improved metabolism and heat stress adaptation of dairy cows leads to better comfort (higher DMI, lower respiratory rate) and higher production (milk quantity and quality).

Higher feed intake with better feed conversion, improved rumen function, and lower respiratory rate shows better comfort for dairy cows supplemented with yeast probiotic. This leads to increased health and productivity.



The effect of the yeast probiotic Actisaf® and paraprobiotic* Safmannan® on inflammation and LPS translocation

Yeast probiotics and paraprobiotics have been demonstrated to improve intestinal integrity by strengthening tight junctions, which may reduce LPS translocation to the systemic circulation. They have also been shown to reduce pathogenic bacteria, such as *E. coli*, *in vitro* and *in vivo*.

*Paraprobiotic: inactivated (non-viable) microbial cells or cell fractions, used to confer a health benefit when administered orally (Taverniti and Guglielmetti, 2011).

Figure 28: Effects of Actisaf® and Safmannan® on the growth of *Escherichia coli* O157:H7, *Salmonella Typhimurium*, and *Listeria monocytogenes* (Carroll, 2015. In vitro study, USDA)

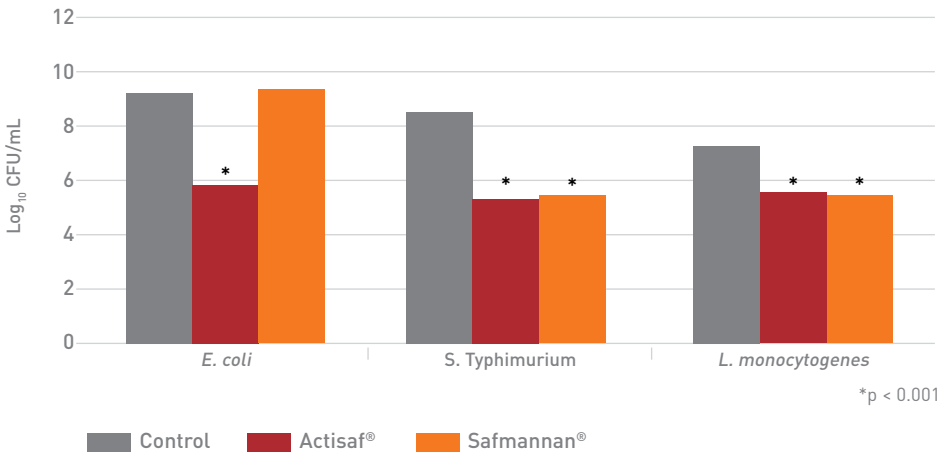
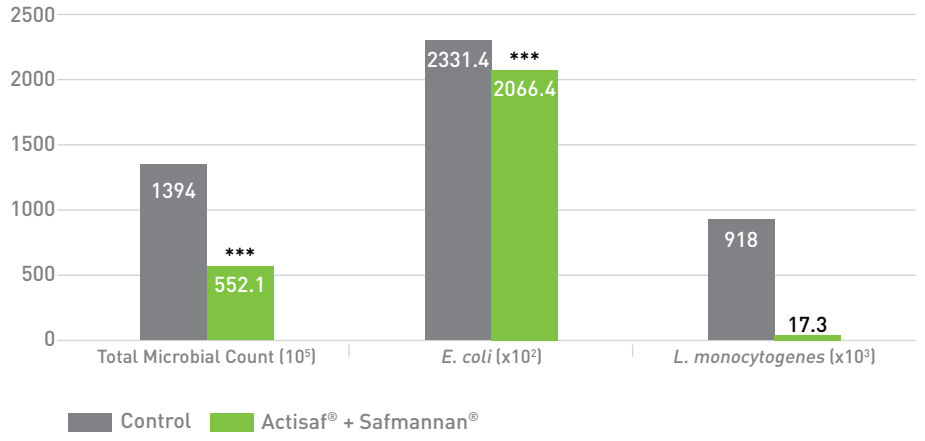


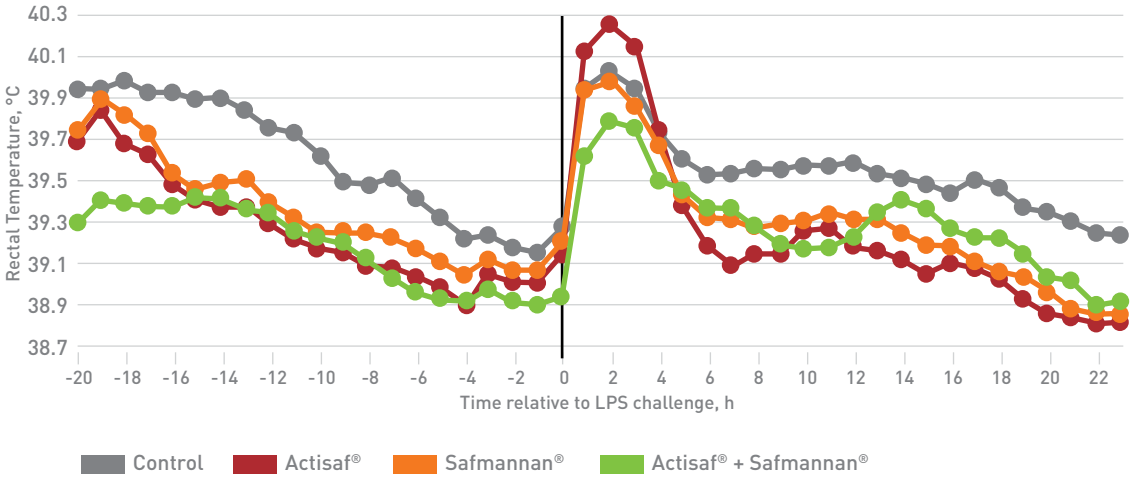
Figure 29: Bacteria present in the rectum at slaughter in beef cattle that received Actisaf® and Safmannan® (Morgante, 2016. Department of Animal Medicine, Production and Health, University of Padua, Italy)



Yeast probiotics and para-probiotics may reduce systemic inflammation after an LPS challenge. Certain yeast strains can modulate the immune system, and they demonstrated an anti-inflammatory effect during an intra-venous challenge with *E. coli* LPS in a trial in 24 beef steers.

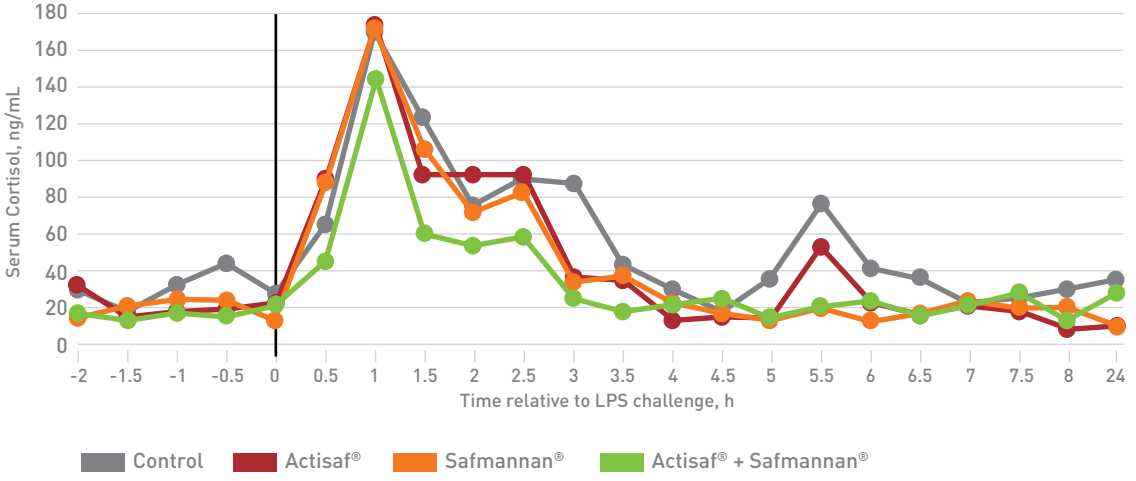
A trial was set up at Texas Tech University (Finck et al., 2014), where the steers were divided into 4 groups: Control, Actisaf®, Safmannan®, and Actisaf® + Safmannan® and fed the same basal diet, plus the relevant treatments (if any). The steers were fitted with jugular vein catheters and individual rectal temperature (RT) recording devices (Reuter et al., 2010) and moved into individual stanchions (2.13 × 0.76 m), where they had ad libitum access to feed and water. An LPS was administered (0.25 µg/kg of BW LPS; *Escherichia coli* O111:B4; Sigma-Aldrich, St. Louis, MO) as the challenge at time 0 h. Blood samples were then analyzed for proinflammatory cytokines.

Figure 30: Effect on rectal temperature



Before administration of the LPS, basal RT tended ($p = 0.06$) to differ between the groups, with Control steers having a higher RT than the Actisaf® + Safmannan® ($p \leq 0.01$) and Actisaf® ($p \leq 0.04$) steers. RT increased ($p < 0.01$) in all steers within 1 h after LPS administration, with peak RT occurring at approximately 2 h, and RT remained higher in Control steers ($p \leq 0.05$) than all other treatment animals throughout the post-LPS period. Animals that received Actisaf® and Safmannan® (alone or in combination) were healthier before the challenge, and recovered normal body temperature much quicker after the challenge.

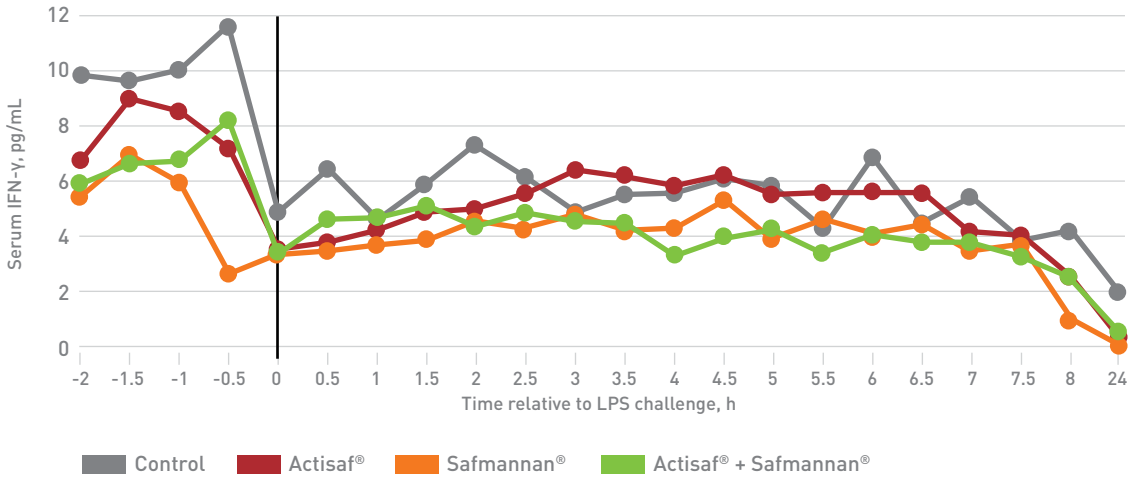
Figure 31: Effect on serum cortisol (Finck et al., 2014)



Serum cortisol concentrations were the same in all treatment groups before LPS administration ($p > 0.05$). Cortisol levels increased in response to LPS, and peaked 1 h post-LPS in all treatment groups, with higher peak cortisol concentrations observed in the Control animals ($p < 0.04$) than the steers supplemented with Actisaf® + Safmannan®.

Cortisol is an indicator of stress in ruminants, and raised cortisol levels are related to an increased requirement for glucose. Cortisol regulates gluconeogenesis and increases glucose production from amino acids in response to the increased demand for glucose during inflammation and stress.

Figure 32: Effect on serum IFN-γ (Finck et al., 2014)



IFN-γ is a proinflammatory cytokine that increases during inflammation. Before LPS administration, basal serum IFN-γ levels were the same ($p > 0.10$) in the Control and treatment groups. However, the IFN-γ levels tended ($p \leq 0.06$) to be higher in the Control group than in the steers supplemented with Safmannan® and Actisaf® + Safmannan®. This indicates lower levels of inflammation due to the immunomodulatory effect of yeast probiotics and paraprobiotics.

Conclusion

Supplementing with yeast probiotics (Actisaf®) and paraprobiotics (Safmannan®) during periods of heat stress may help to reduce leaky gut syndrome and control the inflammation related to bacterial endotoxin translocation. Yeast solutions are multifunctional ingredients that can combine several beneficial properties in different parts of the ruminant digestive tract. The kinetics of Actisaf® and Safmannan® in the dairy cow GIT were studied, and the products were shown to be readily available and metabolically active throughout the GIT, from the rumen to the faeces. This multifunctionality makes Actisaf® and Safmannan® even more relevant to include in a heat stress management program, together with other strategies to reduce the heat overload and better utilize the diet.

Improving the antioxidant status of dairy cows during heat stress with Selsaf®

Supplementing dairy cows under heat stress (THI 72) with selenium-enriched yeast (Selsaf®) improved their antioxidant status and reduced oxidative stress more effectively than an alternative selenium source (sodium selenite).

40 lactating Holstein dairy cows were divided into two groups: a Sodium selenite group and a Selsaf® group. The two groups were balanced for parity, days in milk (DIM), and average daily milk yield. The treatments were as follows:

- Sodium selenite: 20 cows fed a basal diet + sodium selenite (0.3 mg Se/kg DM)
- Selsaf® group: 20 cows fed the same basal diet + Selsaf® (0.3 mg Se/kg DM)

During the trial period, the mean THI was 77.3 at 6:00 am; 83.3 at 2:00 pm and 80.0 at 8:00 pm. Therefore, the cows were subjected to heat stress conditions during the whole experiment (THI ≥ 68).

The cows that received Selsaf® had lower body temperatures and higher serum glutathione peroxidase (GPx) activity.

Figure 33: Effect of Selsaf® on rectal temperature (RT)

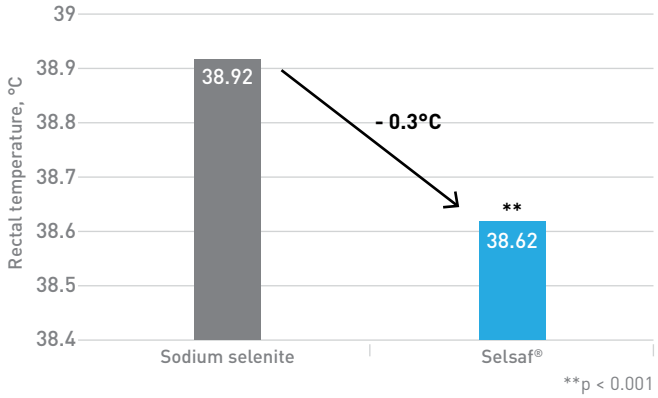
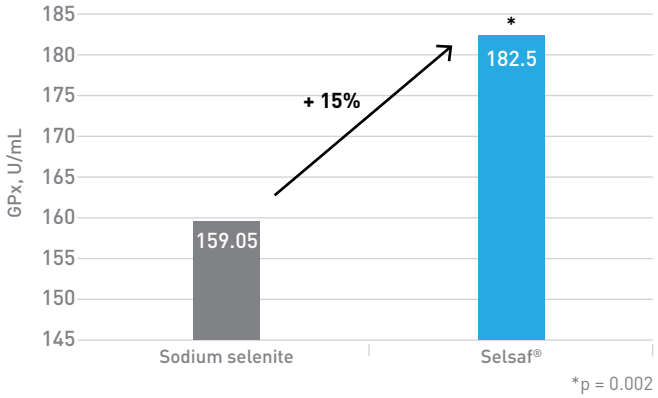


Figure 34: Effect of Selsaf® on serum glutathione peroxidase (GPx) activity



An improvement in antioxidant status leads to better overall health, and the cows that received Selsaf® had significantly lower milk somatic cell counts (SCC) and serum cortisol levels. Serum cortisol is a stress hormone that increases during stress conditions and inflammation.

Figure 35: Effect of Selsaf® on milk SCC

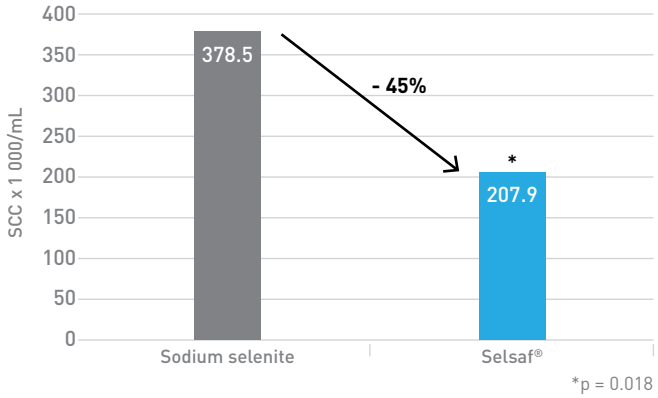
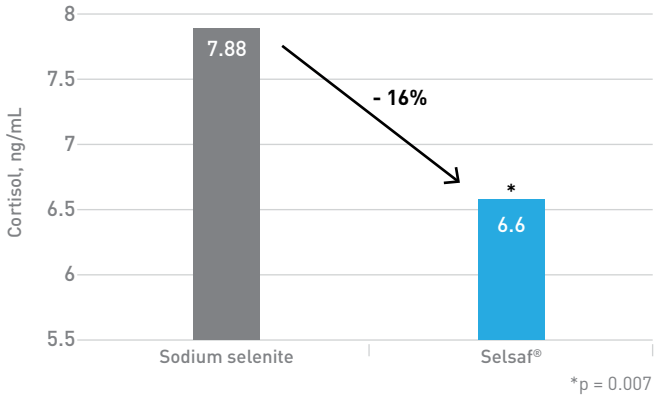


Figure 36: Effect of Selsaf® on serum cortisol levels



CONCLUSION

High temperatures and humidity are detrimental to dairy cow productivity. Heat stress affects feed intake, body temperature, maintenance requirements, metabolic processes, feed efficiency, milk yield, reproductive efficiency, behavior and susceptibility to disease.

A nutritional strategy using yeast products is a promising solution: yeast probiotics and paraprobiotics, as well as selenium-enriched yeast are good candidates to alleviate the negative effects of heat stress on dairy cows.

- Through stabilizing the rumen environment and supporting microflora activity, Actisaf® (yeast probiotic) improves rumen function, which leads to improved metabolism, better comfort (higher DMI, lower respiratory rate) and higher milk production and quality.

- Supplementing animals with Actisaf® and Safmannan® (paraprobiotic) during periods of heat stress may help to reduce leaky gut syndrome and control the inflammation related to bacterial endotoxin translocation.

- Heat stress is thought to affect oxidative status in dairy cows: Selsaf® (selenium enriched yeast) supplementation under heat stress helps to improve their antioxidant status more effectively than an inorganic selenium source (sodium selenite), leading to better overall health.

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