

National Hog Farmer.



Getty Images/iStockphoto

Why we should sweat heat stress

Strategies that directly protect muscle, strategies intended to preserve gut health may also directly reach and protect muscle (and vice versa).

By Tori Rudolph, Robert Rhoads, Lance Baumgard and Joshua Selsby, Iowa State University | Dec 03, 2019

Core body temperature of pigs increases when the rate of heat accumulation exceeds the rate of heat loss. This increase in core body temperature removes the animal from a thermal comfort zone causing heat stress. The acute, severe effects of heat stress on humans can result in death and are generally well-reported in the news (e.g. European heat wave of 2003, Middle East- 2015, France- 2019).

Importantly, those same environmental extremes can negatively impact pig health and wellbeing as well as agricultural economics. Pigs may be more susceptible to heat stress than many species because continued selection for increased growth has resulted in increased metabolic heat production. Further, pigs are unable to effectively reduce body temperature by sweating and rely heavily on conduction and evaporation to maintain their body temperature.

While common in the southern U.S., the threat of heat stress is becoming more frequent and apparent in areas traditionally considered temperate; areas that incidentally have an extensive pig production footprint. The impact of heat stress is multifaceted, and while it can include death, more often, heat stress negatively affects animal growth efficiency, reproductive performance, carcass value and overall health. Cumulatively, these losses may exceed \$900 million annually in the United States alone, and that is with the competitive advantage of mitigation strategies. Heat stress causes multisystemic changes (reproductive, endocrine, gastrointestinal, etc.), many of which have been documented at Iowa State University and other land grant institutions. Our group has been interested in better understanding how heat stress damages muscle and impairs efficient muscle growth.

Under healthy conditions mitochondria efficiently use nutrients to produce Adenosine triphosphate, similar to an energy currency, and consume oxygen as part of that process. Cells then use ATP to support varied cellular functions, including growth. However, when mitochondria are damaged or malfunctioning, they produce ATP less efficiently and consequently produce more free radicals and heat (free radicals are similar to "oxidative stress" often described in humans).

We discovered that two hours of heat stress caused increased oxidative stress; however, with continued heat stress through six hours, oxidative stress was eliminated (Volodina et al., 2017). We think that damaged mitochondria were being removed from the muscle cells through a process called autophagy (auto — self; phagy — eating), which reestablished cell health. But heat stress remains an obvious and continuing challenge for the swine industry so we reasoned that continued heating would injure muscle despite the apparent rescue.

We discovered a re-emergence of oxidative stress with longer-term heating associated with dysfunctional autophagy (Montilla et al., 2014; Brownstein et al., 2017; Ganesan et al., 2017; Ganesan et al., 2018; Ganesan et al., 2019). Autophagy can be impaired by free radical injury,

which means excessive mitochondrial damage and the subsequent greater free radical production, may inhibit the very process intended to remove them!

As oxidative stress appears to be a driver of muscle injury during heat stress, we treated animals with an antioxidant to help preserve normal muscle function. Next, we exposed pigs to thermoneutral or heat stress conditions for 24 hours, similar to our previous experiments. To our surprise, we could not detect oxidative stress in muscles from heat-stressed pigs, regardless of control or antioxidant treatment. Additionally, autophagy was not negatively impacted.

As one can imagine, these findings were quite frustrating because they are in stark conflict with previous reports from our lab. After close review and consideration, we noted that previously obtained data were collected from gilts, but this new data set was collected in barrows, leading us to consider that the muscle response to heat stress may be at least partially dependent on the sex of the pig. Indeed, early work probing this idea indicates tissues from males are more resistant to heat stress-mediated injury than muscle from females.

We are eager to consider the role of sex further, both at the muscle level and how it impacts production systems.

The triggering events leading to heat stress-mediated muscle injury are unknown. While it is possible that increased temperature damages mitochondria directly, it is also likely there is some other initiating factor(s). We are currently intrigued by the possibility that a disruption of calcium regulation may be a driver of muscle dysfunction as this process is sensitive to heat and leads directly to impaired mitochondrial function. In addition, skeletal muscle within the heat-stressed environment of a pig appears to respond to intestinal injury and leakage of bacteria or bacterial components into circulation. Termed "leaky gut," this initiates an immune response and systemic inflammation, which may be the first domino in a series of events leading to poor muscle growth.

Hence, in addition to strategies that directly protect muscle (e.g. antioxidants), strategies intended to preserve gut health may also directly reach and protect muscle (and vice versa), but also provide indirect protection to muscle by limiting systemic inflammation and inflammatory signaling in muscle.

Research cited

Brownstein, A. J., S. Ganesan, C. M. Summers, S. Pearce, B. J. Hale, J. W. Ross, N. Gabler, J. T. Seibert, R. P. Rhoads, L. H. Baumgard, and J. T. Selsby. 2017. Heat stress causes dysfunctional autophagy in oxidative skeletal muscle. *Physiol Rep* 5(12)

Ganesan, S., A. J. Brownstein, S. C. Pearce, M. B. Hudson, N. K. Gabler, L. H. Baumgard, R. P. Rhoads, and J. T. Selsby. 2018. Prolonged environment-induced hyperthermia alters autophagy in oxidative skeletal muscle in *Sus scrofa*. *J Therm Biol* 74:160-169. doi: 10.1016/j.jtherbio.2018.03.007

Ganesan, S., C. M. Summers, S. C. Pearce, N. K. Gabler, R. J. Valentine, L. H. Baumgard, R. P. Rhoads, and J. T. Selsby. 2017. Short-term heat stress causes altered intracellular signaling in oxidative skeletal muscle. *Journal of Animal Science* 95(6):2438-2451. doi: 10.2527/jas.2016.1233

Ganesan, S., C. M. Summers, S. C. Pearce, N. K. Gabler, R. J. Valentine, L. H. Baumgard, R. P. Rhoads, and J. T. Selsby. 2019. Short-term heat stress causes altered intracellular signaling in oxidative skeletal muscle. *Journal of Animal Science* 95(6):2438-2451. doi: 10.2527/jas.2016.1233

Montilla, S. I. R., T. P. Johnson, S. C. Pearce, D. Gardan-Salmon, N. K. Gabler, J. W. Ross, R. P. Rhoads, L. H. Baumgard, S. M. Lonergan, and J. T. Selsby. 2014. Heat stress causes oxidative stress but not inflammatory signaling in porcine skeletal muscle, *Temperature (Austin)* No. 1. p. 42-50.

Volodina, O., S. Ganesan, S. C. Pearce, N. K. Gabler, L. H. Baumgard, R. P. Rhoads, and J. T. Selsby. 2017. Short-term heat stress alters redox balance in porcine skeletal muscle, *Physiol Rep* No. 5.

Source: Tori Rudolph, Robert Rhoads, Lance Baumgard and Joshua Selsby, who are solely responsible for the information provided, and wholly own the information. Informa Business Media and all its subsidiaries are not responsible for any of the content contained in this information asset.

Source URL: <https://www.nationalhogfarmer.com/animal-health/why-we-should-sweat-heat-stress>