Review

Heat stress in swine- a review

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ABSTRACT

Heat stress (HS) is one of the major economic impacts on production efficiency in swine industry by reducing voluntary feed intake with subsequent negative consequences on growth, production and reproductive performance. HS reduces the blood flow to the intestine, disrupts intestinal barrier function, increases permeability of endotoxin and leads to endotoxemia and systemic inflammatory response. HS increases glucose utilization by enhancing pancreatic insulin secretion, decreases carcass composition by reduction of protein synthesis, induces oxidative stress and reduces endogenous antioxidants. HS in swine can be managed by provision of adequate ventilation, adlibitum water, adequate cooling of the animal, reduced protein diet with balanced amino acid, reduced fibre content of diet, supplementation of electrolytes and exogenous antioxidants.

Keywords: Heat stress; antioxidants; heat shock protein

INTRODUCTION

The swine industry is one of the fast growing industries in the world in livestock sector. Heat stress (HS) is a major limiting factor on production efficiency in the swine industry by reducing voluntary feed intake which indirectly affects the growth, production and reproductive performance of pig. HS is a state of physiological strain within an animal's body when exposed to high ambient temperature, high humidity, low wind speed and high solar radiation and animals are not able to regulate their heat homeostasis passively (Ondruska et al 2011). This is an occasional problem in temperate whereas chronic issue in tropical countries (Renaudeau et al 2012). Pigs are much more sensitive to hot weather than other livestock species largely due to absence of sweating and presence of relatively small lungs compared to their body size. The present review article describes heat stress in swine.

Mechanism of action

When the environmental temperature exceeds the critical level (18 to 24°C) the animals are susceptible to heat stress. Heat-stressed animal redistributes the blood to the periphery in an attempt to maximize heat loss through skin and vasoconstriction of splanchnic vessels in gastrointestinal tract (Hall et al 2001). Consequently reduced flow of blood and nutrients to the intestinal epithelium leads to hypoxia, damages the intestinal barrier and increases the permeability of endotoxin that leads to endotoxemia and systemic inflammatory response (Hall et al 2001, Pearce et al 2013). Heat-stressed pigs show symptoms of high rectal temperaure, lieing on the cool place, panting, increased respiration, convulsion and death (Anon 2008).

Effect of heat stress on metabolism

Carbohydrates are readily available energy source that can be converted into a number of metabolic intermediates used for the production of ATP (Slimen et al 2015). Heat stress appears to markedly alter intracellular energetics characterized by a decrease in ATP production via oxidative phosphorylation and an increase in energy production via aerobic glycolysis (Sanz Fernandez et al 2015).

Heat stress enhances the pancreatic insulin secretion rather than decreased systemic insulin clearance (Rhoads et al 2013), increases level of basal and stimulated (ie in response to a metabolic challenge) circulating insulin and decreases adipose tissue

mobilization in a variety of species (Baumgard and Rhoads 2013) including pigs (Pearce et al 2013). An increased action of circulating insulin causes increase in glucose utilization by the body (Febbraio 2001). The increased hepatic glucose output during heat stress originates from both increased glycogenolysis and gluconeogenesis and is due to upregulation of hepatic pyruvate carboxylase gene expression, a rate-limiting enzyme controlling lactate and alanine entry into the gluconeogenic pathway. Insulin is a potent lipogenic and antilipolytic signal and is frequently elevated in HS animals as compared with thermo-neutral counterparts. Heat stress depresses protein deposition by reduction of protein synthesis, increases fat accretion through lipogenesis and decreases carcass composition (Baumgard and Rhoads 2013).

Cellular response to heat stress

Heat stress is likely to induce oxidative stress by production of reactive oxygen species (ROS) such as superoxide dismutase, catalase and glutathione peroxidase which can damage protein, DNA, lipids and mitochondrial function in cell (Addabbo et al 2009). Generally oxidative stress occurs when accumulation of reactive oxygen species (ROS) exceeds the cellular detoxification mechanism. Endogenous antioxidants like glutathione, vitamin E, C and A and ß-carotenes decrease during hot season in many animal species is due to mobilization of cellular antioxidant to detoxify free radicals generated by heat exposure (Kumar et al 2011, Slimen et al 2015).

The cellular response to heat load includes activation of transcription factors such as heat shock factors (HSFs) and expression of heat shock protein associated with acute homeostatic response. The production of heat shock protein (HSP) during thermal stress is important for cell survival during hyperthermia. HSP is usually present at lower level under normal cell conditions which is transiently increased on cellular insult (Rhoads et al 2013). HSP is generally accepted as molecular chaperones known to confer cellular tolerance to intracellular injury (Bao et al 2008). Major HSPs in mammalian cells include HSP 110, 90, 70, 60 and 27 (Arya et al 2007). HSP 70 is frequently used as a biomarker of cellular stress (Bao et al 2008).

Effect on growth and reproduction

Heat stress induces reduced voluntary feed intake in an attempt to produce less heat. This has direct effect on growth rate as smaller quantities of feed are ingested over a period of time (Le Bellego et al 2002). The negative effects of heat stress on growth are increased when the relative humidity is high. Huynh et al (2005) reported that greatest decrease in average daily gains (ADGs) was found at RH of 80 per cent with high ambient temperature.

Heat stress decreases fertility in sows and gilts that is typically manifested as seasonal infertility and delayed onset of puberty in growing pigs (Ross et al 2015). Decreased conception rates and litter sizes were also found in gilts exposed to heat stress between 0 and 16 days after mating (Wettemann and Bazer 1985). The decreased dry matter intake in lactating sows response to heat stress causes weight loss as the energy input is no longer sufficient for body maintenance and lactation. Piglets born from heat stressed sows take longer time to return to oestrus after weaning than sows unexposed to heat stress (Spencer et al 2003).

Hematological and electrolyte changes

Heat stress increases reticulocyte count, erythrocyte deformity and reduction of blood viscosity in growing pig in an attempt to reduce tissue hypoxia (Waltz et al 2014). Heat-stressed pig shows lower level of plasma bicarbonate and potassium due to increased excretion (Ma et al 2015).

Management of heat stress

The management practice to reduce heat stress in pig includes provision of adequate ventilation and air velocity over the animals raised in confinement building during warm weather. Swine must have access to large quantities of water during periods of high environmental temperatures. Much of the water is needed to evaporative heat loss via respiration to help the pig cool off. Cooling system like water sprinkler and dripping of water on the neck and shoulder along the air movement is an effective way to cool the skin and maximize heat loss.

Alteration in feeding management such as a change in feeding frequency is an efficient tool to avoid excessive heat load and improve survival rate especially in poultry (Renaudeau et al 2012). It is recommended that reduce the energy and fibre content and increase fat in the diet to reduce heat production (Slimen et al 2015). Dietary supplementation of lipoic or dihydrolipoic acid may improve heat tolerance and animal performance during heat stress by enhancing insulin action (Rhoads et al 2013). Adding value to the pig's diet with low protein and balanced amino acids does not reduce the pig net energy intake and production

performance. Supplementation of synthetic lysine instead of natural protein improves the pig production performance (Ma et al 2015).

Dietary supplementation of exogenous antioxidants like vitamins C, A and E, Zn and chromium may attenuate negative consequence of heat stress (Sunil Kumar et al 2011). Supplementation of vitamin E and C reduces the rectal temperature and respiratory rate by directly altering thermal set point (hypothalamic thermoregulatory zone) by decreasing prostaglandin output especially of PGE series (Sivakumar et al 2010). Cottrell et al (2015) reported that the supplementation of vitamin E and Selenium (Se) in diet at supra-nutritional level mitigates HS in sheep by upregulation of HSP mRNA expression and down regulation of pro-inflammatory cytokine. Zinc may be used as feed additive for alleviating the negative effects of intestinal-related stresses and decrease the circulating endotoxin by improving intestinal barrier function (Ma et al 2015). Wang et al (2004) reported that supplementation of 530 mg/kg zinc methionine and 200 mg/kg pyridine chromium carboxylate to pig's diet increased the average daily feed intake and the average daily gain and decreased the feed-gain ratio under the high temperature condition.

Supplementation of electrolytes is one among the nutritional strategies to combat heat stress in animals. The sodium and potassium are given in the form of bicarbonate/carbonate to regulate acid-base balance in the blood (Sunil Kumar et al 2011). Potassium chloride or sodium bicarbonate electrolyte should be added to the pig diets appropriately to reduce the damage caused by heat stress. Oral supplementation of 0.1 to 0.2 per cent sodium bicarbonate or 0.1 to 0.2 per cent vitamin E to the pig diets had beneficial effect on preventing heat stress. The piglets drinking electrolyte solution (containing sugar, sodium chloride, potassium chloride, sodium bicarbonate and citric acid) increases average daily weight gain by about 15.64 per cent (Ma et al 2015).

CONCLUSION

Heat stress increases glucose utilization by enhancing pancreatic insulin secretion, decreases carcass composition and causes reduction in the endogenous antioxidants. Heat stress in pig can be reduced by adopting adequate shelter management practices, supplementation of antioxidants and electrolyte solution.

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