Journal of Thermal Biology 61 (2016) 29–37

Contents lists available at ScienceDirect

Journal of Thermal Biology

journal homepage: www.elsevier.com/locate/jtherbio

Effects of rapid temperature fluctuations prior to breeding on reproductive efficiency in replacement gilts ☆

J.S. Johnson [a,*], K.L. Martin [b], K.G. Pohler [c], K.R. Stewart [b]

[a] USDA-ARS, Livestock Behavior Research Unit, 125 S. Russell St., Rm. 219C, West Lafayette, IN 47907, United States
[b] Purdue University, Department of Animal Sciences, West Lafayette, IN 47907, United States
[c] University of Tennessee, Department of Animal Science, Knoxville, TN 37996, United States

ARTICLE INFO

Article history:
Received 17 June 2016
Received in revised form
11 August 2016
Accepted 11 August 2016
Available online 19 August 2016

Keywords:
Breeding
Fetal viability
Pigs
Rapid cooling
Temperature fluctuations

ABSTRACT

Rapidly cooling pigs after heat stress (HS) results in a pathophysiological condition, and because rapid temperature fluctuations may be associated with reduced reproductive success in sows, it lends itself to the hypothesis that these conditions may be linked. Objectives were to determine the effects of rapid cooling on thermal response and future reproductive success in pigs. Thirty-six replacement gilts (137.8 ± 0.9 kg BW) were estrus synchronized and then 14.1 ± 0.4 d after estrus confirmation, pigs were exposed to thermoneutral conditions (TN; n = 12; 19.7 ± 0.9°C) for 6 h, or HS (36.3 ± 0.5°C) for 3 h, followed by 3 h of rapid cooling (HSRC; n = 12; immediate TN exposure and water dousing) or gradual cooling (HSGC; n = 12; gradual decrease to TN conditions) repeated over 2 d. Vaginal (T_V) and gastrointestinal tract temperatures (T_{GI}) were obtained every 15 min, and blood was collected on d 1 and d 2 during the HS and recovery periods at 180 and 60 min, respectively. Pigs were bred 8.3 ± 0.8 d after thermal treatments over 2 d. Reproductive tracts were collected and total fetus number and viability were recorded 28.0 ± 0.8 d after insemination. HS increased T_V and T_{GI} ($P = 0.01$; 0.98 °C) in HSRC and HSGC compared to TN pigs. During recovery, T_V was reduced from 15 to 105 min ($P = 0.01$; 0.33 °C) in HSRC compared to HSGC pigs, but no overall differences in T_{GI} were detected ($P < 0.05$; 39.67 °C). Rapid cooling increased ($P < 0.05$) TNFα compared to HSGC and TN pigs during recovery-d 1 (55.2%), HS-d 2 (35.1%), and recovery-d 2 (64.9%). Viable fetuses tended to be reduced ($P = 0.08$; 10.5%) and moribund fetuses tended to be increased ($P = 0.09$; 159.3%) in HSRC compared to HSGC and TN pigs. In summary, rapid cooling prior to breeding may contribute to reduced fetal viability and reproductive success in pigs.
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1. Introduction

Climate change threatens the sustainability of animal agriculture and can negatively impact the health and development of livestock at all stages of production (Johnson et al., 2015). In particular, heat stress (HS) resulting from the imbalance between thermal energy flowing into and out of an animal (Kleiber, 1961) can slow growth, decrease reproductive efficiency, alter metabolism and body composition, and in severe cases, morbidity and mortality can occur (Baumgard and Rhoads, 2013; Johnson et al., 2015). As climate models predict an increase in the frequency of heat waves and periods of extreme high temperatures for most U.S. swine producing areas (U.S. EPA, 2014), incidences of heat-related maladies for pigs are likely to increase. Therefore, it is imperative that current HS management practices are evaluated and improved to mitigate the effects of HS on pig health and improve overall welfare and productivity.

The agriculture sector contributes $200 billion annually to the US economy (USGCRP, 2009); however, it is the most vulnerable to climate change (IPCC, 2014) and it is estimated that the U.S. swine industry loses > $300 million/year due to HS-related losses in productivity (St-Pierre et al., 2003). Of these losses, reproductive inefficiency (commonly referred to as seasonal infertility) due to HS is estimated to cost producers $55 per sow each year (Pollmann, 2010) despite improved cooling systems and management practices. Seasonal infertility has been recognized as a potential economic loss to the swine industry since the late 1970s (Love, 1978; Stork, 1979) and is characterized by prolonged weaning-to-mating intervals, reduced conception rates, reductions in farrowing rate and embryonic survival, and a reduction in pigs weaned per litter (as reviewed by Bertoldo et al., 2012). Alterations in nutrition, photoperiod, housing, insulin action, and rapid

☆ Mention of trade names or commercial products in this article is solely for the purpose of providing specific information and does not imply recommendation or endorsement by the U.S. Department of Agriculture. All procedures involving animal use were approved by the Purdue University Animal Care and Use Committee (protocol #1410001136).

* Corresponding author.
E-mail address: Jay.Johnson@ars.usda.gov (J.S. Johnson).

http://dx.doi.org/10.1016/j.jtherbio.2016.08.003
0306-4565/Published by Elsevier Ltd.

temperature fluctuations have been suggested as potential causes (Sanford, 1982; Almond, 1985; Auvigne et al., 2010; Nteeba et al., 2015), although there appears to be little consensus regarding the specific mechanism of action. Therefore, determining the potential mechanisms and specific stressors that contribute to HS-induced reproductive inefficiency is a prerequisite to developing mitigation strategies.

Recently, we determined that rapidly cooling pigs after acute HS resulted in a pathophysiological condition characterized by intestinal damage and an increase in circulating cytokines (Johnson et al., 2016). Because rapid fluctuations in temperature due to greater day and nighttime temperature variation may be associated with reduced reproductive success in late summer and autumn months (Sanford, 1982; Almond, 1985), the previously observed increase in cytokine response due to rapid cooling (Johnson et al., 2016) may be a contributing factor, especially when low nighttime temperatures are combined with evaporative cooling technology. Therefore, study objectives were to determine the effects of rapid reductions in ambient temperature after acute HS on thermal response and future reproductive success in replacement gilts. We hypothesized that rapid cooling after acute HS for 2 consecutive days prior to breeding would reduce overall reproductive efficiency in replacement gilts.

2. Materials and methods

2.1. Animals and pre-treatment procedures

All procedures involving animal use were approved by the Purdue University Animal Care and Use Committee (protocol #1410001136). Thirty-six crossbred replacement gilts (1/2 Large White x 1/2 Landrace; 137.8 ± 1.9 kg BW) were housed in individual gestation crates (2.44 m × 0.61 m), allowed ad libitum access to water, and were limit-fed 2.3 kg/d during the entire trial to prevent excessive maternal weight gain (standard industry practice; Brendemuhl and Myer, 2009). The diet consisted of primarily corn and soybean meal and was formulated to meet or exceed nutrient requirements for gestating gilts (NRC, 2012). To synchronize and establish estrus, all gilts were orally administered 6.8 mL of Matrix® per manufacturer's instructions (Merck Animal Health; Williamsburg, KS) once daily for 14 d, and then Matrix® was withdrawn and gilts were monitored for estrus by two trained individuals twice daily (0800 and 1500 h).

2.2. Thermal treatments

Thermal treatments were administered on 2 consecutive days, 14.1 ± 0.4 d after estrus was confirmed in all pigs on trial to mimic a 2 d heat wave. For thermal treatments, gilts were moved from their gestation crates to individual pens (0.91 × 1.83 m) at the Purdue University swine farm environmental rooms 1 d prior to thermal treatments, and then all gilts were orally administered a calibrated CorTemp® temperature sensor (model HT150002; accuracy: ± 0.1 °C; HQ Inc.; Palmetto, FL) at 1500 h to monitor gastrointestinal temperature (T_{GI}). In each environmental room, percent relative humidity (RH) and ambient temperature (T_a) were recorded every 5 min using two mounted data loggers (Hobo®; data logger temp/RH; Onset®; Bourne, MA) for the duration of the experiment.

To evaluate the effects of T_a fluctuations on future reproductive success, replacement gilts were subjected to 2 consecutive days of either thermoneutrality (TN; n = 12; 129.9 ± 2.5 kg BW; 19.7 ± 0.9 °C; 49.5 ± 5.9% RH) for 6 h, or HS (36.3 ± 0.5 °C; 33.4 ± 3.3% RH) for 3 h, followed by a 3 h recovery period of either rapid cooling (HSRC; n = 12; 130.2 ± 2.4 kg BW) or gradual cooling

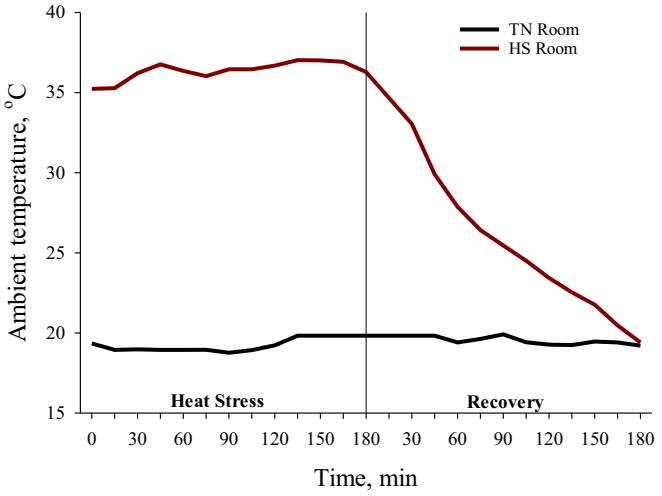

Fig. 1. Ambient temperature temporal pattern on d 1 and d 2 of thermal treatments by time (min) in the heat stress and recovery periods. Abbreviations are thermoneutral room (TN Room) and heat stress room (HS Room).

(HSGC; n = 12; 130.4 ± 1.8 kg BW) as previously described (Johnson et al., 2016). Briefly, rapid cooling was achieved by moving pigs from the HS room directly into the TN room (1.5 m walking distance) and then pouring 15 gallons of water (4.0 °C) on the backs of individual pigs every 30 min for 1.5 h (total of 4 times), and gradual cooling was accomplished by reducing the HS room T_a by 5.0 °C every 30 min until TN environmental conditions (20.8 ± 0.7 °C; 42.7 ± 3.5% RH) were reached (Fig. 1). Throughout the 6 h thermal treatment period on d 1 and d 2, vaginal temperature (T_V), T_{GI}, and whole-body skin temperature (T_{SK}) of individual pigs were measured every 15 min. Vaginal temperature was measured with a calibrated thermochron temperature recorder (iButton; accuracy: ± 0.1 °C; Dallas Semiconductor; Maxim®; Irving, TX) attached using non-absorbable polyamide suture (Braunamid; B. Braun Medical Ltd., 146 Sheffield, UK) to a blank sheep CIDR® (Eazi-Breed; Zoetis, New York, NY) and then sterilized, lubricated, and inserted intravaginally in unrestrained pigs 1 d prior to thermal treatments at 1500 h. Gastrointestinal temperature was measured using orally administered CorTemp® temperature sensors (given at 1500 h 1 d prior to thermal treatments) monitored with a wireless core body temperature data recorder (CorTemp® Data Recorder 262 K; model HT130003; HQ Inc.; Palmetto, FL). Whole-body T_{SK} was measured by taking a broadside photo of individual pigs using an infrared camera (FLIR-T62101; FLIR Systems Inc.; Wilsonville, OR), and photos were analyzed with FLIR Tools software (version 2.1). A thermal circulation index (TCI) was calculated as previously described (Johnson et al., 2013, 2016) using T_{GI}, T_{SK}, and T_a, to quantify blood and heat transfer to the skin as described for livestock species [$TCI = T_{SK} - T_a)/(T_{GI} - T_{SK})$; Curtis, 1983].

2.3. Blood sampling and analysis

Two blood tubes (serum and EDTA; 5 mL) were obtained on restrained pigs via jugular venipuncture (BD® vacutainers; Franklin Lakes, NJ; K_3EDTA; serum) at 180 min during the HS period, and at 60 min during the recovery period on d 1 and d 2 of the thermal treatment period. Plasma and serum were harvested by centrifugation at 4 °C and 2500 rpm for 15 min, aliquoted and stored at −80 °C. Plasma glucose concentrations were measured enzymatically using a commercially available kit (Wako Chemicals, Richmond, VA). ELISA kits were used to determine serum TNFα (Swine TNFα ELISA Kit; Invitrogen™; Thermo Fisher Scientific; Waltham, MA) and plasma insulin (Mercodia Porcine Insulin