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Does heat stress alter the pig's response to dietary fat?

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Abstract

Heat stress (HS) results in major losses to the pork industry via reduced growth performance and possibly carcass fat quality. The experimental objective was to measure the effects of HS on the pig's response to dietary fat in terms of lipid digestion, metabolism, and deposition over a 35 d finishing period. A total of 96 PIC 337 × C22/C29 (PIC, Inc., Hendersonville, TN) barrows (initial BW of 100.4 ± 1.2 kg) were allotted randomly to 1 of 9 treatments arranged as a 3 × 3 factorial: [TN (thermonetural: constant 24°C; ad libitum access to feed), PFTN (pair-fed thermoneutral: constant 24°C; limit-fed based on previous HS daily feed intake), or HS (cyclical 28°C nighttime, 33°C d 0 to 7, 33.5°C d 7 to 14, 34°C d 14 to 21, 34.5°C d 21 to 28, 35°C d 28 to 35 daytime; ab libitum access to feed)] and diet [a corn-soybean meal based diet with 0% added fat (CNTR), 3% added tallow (TAL; iodine value (IV) = 41.8), or 3% added corn oil (CO; IV = 123.0)]. No interactions between environment and diet were evident for any major response criteria ($P \geq 0.063$). Rectal temperature increased due to HS (HS = 39.0, TN = 38.1, PFTN = 38.2°C; $P < 0.001$). Heat stress decreased ADFI (27.8%; $P < 0.001$), ADG (HS = 0.72, TN = 1.03, PFTN = 0.78 kg/d; $P < 0.001$), and G:F (HS = 0.290, TN = 0.301, PFTN = 0.319; $P = 0.006$). Heat stress barrows required 1.2 Mcal of ME intake more per kg of BW gain than PFTN ($P < 0.001$). Heat stress tended to result in the lowest ATTD of AEE (HS = 59.0, TN = 60.2, PFTN = 61.4%, $P = 0.055$). True total tract digestibility of AEE of CO-based diets (99.3%) was greater than that of CNTR (97.3%) and TAL-based diets (96.3%; $P = 0.012$). Environment had no impact on TTTD of AEE ($P = 0.118$). Environment had no impact on jowl IV at market (HS = 69.2, TN = 69.3, PFTN = 69.8 g/100 g; $P = 0.624$). Jowl IV at market increased with increasing degree of unsaturation of the dietary fat (CNTR = 68.5, TAL = 68.2, CO = 71.5 g/100 g; $P < 0.001$). Heat stress decreased mRNA abundance of ATGL and HSL ($P \leq 0.041$). HS and CO increased mRNA abundance of SCD ($P \leq 0.047$), and CO increased abundance of FASN ($P = 0.011$). In conclusion, HS does not alter the pig's response to dietary fat. However, HS leads to reduced ADG, ADFI, G:F, caloric efficiency, and a suppression of mRNA abundance of genes involved in the lipolytic cascade, which resulted in a phenotype that was fatter than PFTN.

Keywords

de novo lipogenesis, dietary fat, heat stress, iodine value, lipid metabolism, swine

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Running Head: Heat stress impacts on fat metabolism in pigs

Does heat stress alter the pig's response to dietary fat?

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ABSTRACT: Heat stress (HS) results in major losses to the pork industry via reduced growth performance and possibly carcass fat quality. The experimental objective was to measure the effects of HS on the pig's response to dietary fat in terms of lipid digestion, metabolism, and deposition over a 35 d finishing period. A total of 96 PIC 337 × C22/C29 (PIC, Inc., Hendersonville, TN) barrows (initial BW of 100.4 ± 1.2 kg) were allotted randomly to 1 of 9 treatments arranged as a 3 × 3 factorial: [TN (thermonetural: constant 24°C; ad libitum access to feed), PFTN (pair-fed thermoneutral: constant 24°C; limit-fed based on previous HS daily feed intake), or HS (cyclical 28°C nighttime, 33°C d 0 to 7, 33.5°C d 7 to 14, 34°C d 14 to 21, 34.5°C d 21 to 28, 35°C d 28 to 35 daytime; ab libitum access to feed)] and diet [a corn-soybean meal based diet with 0% added fat (CNTR), 3% added tallow (TAL; iodine value (IV) = 41.8), or 3% added corn oil (CO; IV = 123.0)]. No interactions between environment and diet were evident for any major response criteria ($P \geq 0.063$). Rectal temperature increased due to HS (HS = 39.0, TN = 38.1, PFTN = 38.2°C; $P < 0.001$). Heat stress decreased ADFI (27.8%; $P < 0.001$), ADG (HS = 0.72, TN = 1.03, PFTN = 0.78 kg/d; $P < 0.001$), and G:F (HS = 0.290, TN = 0.301, PFTN = 0.319; $P = 0.006$). Heat stress barrows required 1.2 Mcal of ME intake more per kg of BW gain than PFTN ($P < 0.001$). Heat stress tended to result in the lowest ATTD of AEE (HS = 59.0, TN = 60.2, PFTN = 61.4%, $P = 0.055$). True total tract digestibility of AEE of CO-based diets (99.3%) was greater than that of CNTR (97.3%) and TAL-based diets (96.3%; $P = 0.012$). Environment had no impact on TTTD of AEE ($P = 0.118$). Environment had no impact on jowl IV at market (HS = 69.2, TN = 69.3, PFTN = 69.8 g/100 g; $P = 0.624$). Jowl IV at market increased with increasing degree of unsaturation of the dietary fat (CNTR = 68.5, TAL = 68.2, CO = 71.5 g/100 g; $P < 0.001$). Heat stress decreased mRNA abundance of ATGL and HSL ($P \leq 0.041$). HS and CO increased mRNA abundance of SCD ($P \leq 0.047$), and CO

increased abundance of FASN ($P = 0.011$). In conclusion, HS does not alter the pig's response to dietary fat. However, HS leads to reduced ADG, ADFI, G:F, caloric efficiency, and a suppression of mRNA abundance of genes involved in the lipolytic cascade, which resulted in a phenotype that was fatter than PFTN.

Key words: de novo lipogenesis, dietary fat, heat stress, iodine value, lipid metabolism, swine

INTRODUCTION

Heat stress (**HS**) affects a plethora of swine production variables (Baumgard et al., 2012); its negative impact on ADG has been known for over 110 yrs (Grisdale, 1904; Heitman et al., 1958). Despite improvements in barn design, genetics, management, and nutrition, HS remains one of the most costly issues for American pork producers (St-Pierre et al., 2003; Renaudeau et al., 2012).

To reduce heat stress's negative impact on energy intake (Hao et al., 2014; Pearce et al., 2014), producers formulate diets utilizing ingredients that are energy dense and low in heat increment (Forbes and Swift, 1944; Stahly et al., 1981). Because dietary fat and oils are energy dense and have a low heat increment, (NRC, 2012; Kerr et al., 2015), their use increases in the hotter months of the year. Adding dietary fat has been shown to reduce the negative effects of HS on ADG (Stahly et al., 1981; Spencer et al., 2005). What is unknown is whether high ambient temperature affects the pig's utilization of fat, and if a fat source that is more unsaturated will be more effective at alleviating the negative effects of HS.

A review by Baumgard and Rhodes (2013) concluded that pigs that experience HS deposit more lipid than predicted based on their energy consumption. It is also known that the composition of dietary fat will be highly reflective of pork fat composition (Ellis and Isbell, 1923; Kellner et al., 2014). This creates a scenario where high fat diets are employed to alleviate HS and HS pigs deposit even greater amounts of fat than expected, increasing the risk of carcass fat quality issues when HS occurs (Spencer et al., 2005; White et al., 2008).

The experimental objective was to determine if HS would impact the pig's response to a more saturated or a less saturated dietary fat source in terms of growth performance, caloric efficiency, lipid metabolism, carcass quality, and carcass iodine value (**IV**).

MATERIALS AND METHODS

All experimental procedures adhered to guidelines for the ethical and humane use of animals for research, and were approved by the Iowa State University Institutional Animal Care and Use Committee (#1-14-7703-S).

Animals, Housing, and Experimental Design

A total of 96 PIC 337 × C22/C29 (PIC, Inc., Hendersonville, TN) barrows, with an average initial BW of 100.4 ± 1.2 kg were allotted by BW and pre-experiment ADG to 1 of 9 treatments arranged as a 3 × 3 factorial. The first factor was environmental treatment: thermoneutral (**TN**; ad libitum access to feed), pair-fed thermoneutral (**PFTN**; limit-fed based on HS feed intake on the previous day), or HS (ab libitum access to feed). The second factor was diet: a corn-soybean meal based diet with 0% added fat (**CNTR**), CNTR with 3% added tallow (**TAL**; IV = 41.8), or CNTR with 3% added corn oil (**CO**; IV = 123.0). There were 2 sequential replications of 48 barrows each.

Pigs were housed in 2 identical rooms where temperature was controlled (Figure 1), but humidity, while similar between the 2 rooms, was not regulated (Figure 2). Each room contained 24 individual pens. Each pen provided 1.25 m^2 of floor space, a nipple drinker, a stainless steel feeder, and had mesh metal flooring. Pigs were given ad libitum access to water throughout the experiment.

The control room housed TN and PFTN barrows and was maintained within the thermoneutral temperature zone for pigs of this age (24°C; Comberg et al., 1972; Renaudeau, 2012). The HS room housed HS barrows and was heated in a diurnal pattern (Figure 1) at 28°C from 2000 h to 800 h and at 33°C d 0 to 7, 33.5°C d 7 to 14, 34°C d 14 to 21, 34.5°C d 21 to 28,

35°C d 28 to 35 from 800 h to 2000 h. The temperature of the HS room was set greater than estimated upper critical temperature point from 800 h to 2000 h and set slightly less than the estimated upper critical temperature point from 2000 h to 800 h based on multiple studies complied by Renaudeau (2012). Additionally, the upper temperature of the HS room was increased 0.5°C every 7 d to minimize acclimation to the environmental conditions during the 35 d experiment. Temperature and humidity in both rooms were recorded every 30 min using a data logger (Lascar EL-USB-2-LCD, Erie, PA).

Diets and Feeding

All experimental diets (Table 1) were formulated on a constant ME to standardized ileal digestible lysine ratio and met or exceeded all nutrient requirements for pigs of this size (NRC, 2012). Diets contained 0.40% titanium dioxide as an indigestible marker to determine the apparent total tract digestibility (**ATTD**) of acid hydrolyzed ether extract (**AEE**), DM, and GE. All experimental diets were offered to the pigs in mash form. Dietary fat sources were selected to provide a diverse range of unsaturation, while keeping in mind choices relevant to current production practices. Representative feed samples were collected at the time of mixing and stored at -20°C for later analysis. Prior to the initiation of the study, the pigs were fed a common diet, similar to the experimental CNTR diet.

Sample Collection

Pigs were weighed individually on d 0, 7, 14, 21, and 35. Feeders in the TN pens were weighed on d 0, 7, 14, 21, and 35. Feeders in the HS room were weighed daily to determine daily feed intake for the next d PFTN feed allotment. If any feed was remaining in the feeders of PFTN barrows at 800 h, it was measured and discarded before the next daily allotment of feed was added. These measurements allowed for the determination of ADG, ADFI, and G:F. Fecal

grab samples were collected fresh from each pig on d 16 to 18, and immediately stored at -20°C for later analysis.

Rectal temperature was measured daily with a dual-scale digital thermometer at 1100 h (VetOne; MWI Veterinary Supply, Boise, ID). Daily respiration rate was determined by counting flank movements at 1200 h. Both measurements were taken in duplicate and condensed into daily averages if numerical differences occurred.

Subcutaneous fat samples from the jowl were collected on d 7 and 21 by biopsy, using local anesthesia. The skin was removed from each 10 g lipid sample. Once the skin was removed a ~200 mg cross section was taken and placed into a 2.5 mL RNAase free microcenterfuge tube (FisherBrand; Fisher Science, Hanover Park, IL) with 2 mL of TRIzol reagent (Invitrogen, Carlsbad, CA). The remaining lipid sample was inserted into a 7.62 by 12.70 cm plastic bag (FisherBrand; Fisher Science, Hanover Park, IL) and snap-frozen using liquid nitrogen. These samples were immediately placed on dry ice and then stored at -80°C for later analysis.

On d 35, pigs were marketed at the JBS professing plant in Marshalltown, IA, where HCW, loin depth, and back fat thickness were measured. Following carcass chilling, a 100 g sample of fat from the right jowl of each carcass was collected, vacuum packaged, and stored at -20°C until analyzed. The loin from the right side of each carcass was measured for pH using a Hanna HI925 meter with an FC200 hard glass electrode (Hanna Instruments, Woonsocket, RI), for loin color score (Japanese color bar 1 to 6, with 1 = extremely light and 6 = extremely dark; Sullivan et al., 2007), and for loin marbling score according to National Pork Board Standards (NPPC, 2000). The right side of the belly from each carcass was collected and measured for weight, temperature, and thickness. Belly thickness was measured in 2 locations in the center of

the belly for middle thickness and at the center of the scribe edge of the belly for edge thickness. A belly firmness test was conducted using a durometer (model 1600-OOO-S; Electromatic Equipment Co., Inc., Cedarhurst, NY) which measured compression of the belly (1 to 100, with 1 = least firm and 100 = firmest; Semen et al., 2013; Kellner et al., 2015). A subjective belly firmness test was conducted by assigning a visual score (1 to 3, 1 = firmest and 3 = least firm) based on the degree of flop of the belly (Kellner et al., 2014). Objective color measures were obtained using a Minolta Chromameter CR 310 (Minolta Corp., Ramsey, NJ) equipped with a 50 mm orifice calibrated against a white tile. Objective color and durometer measures were taken in the middle of the belly with skin removed 3 cm from the proximal edge. Temperature of each belly analyzed was recorded with a thermometer (model 7937; Fisher Science, Hanover Park, IL). No treatment differences among belly temperatures were evident (2.5 ± 0.7°C; $P = 0.580$).

Analytical Methods

Fatty acids were extracted from adipose tissue and feed samples by a 1-step direct transesterification procedure (Lepage and Roy, 1986). The fatty acid profile was then determined by gas chromatography using a model 3900 gas chromatograph fitted with a CP 8400 automatic injector (Varian Analytical Instruments, Walnut Creek, CA) and a 60 m capillary column (0.25 mm diameter; model DB-23; Agilent, Santa Clara, CA). Helium was utilized as a carrier gas at 0.5 mL/mm (1:50 split ratio). Oven temperature started at 50°C and increased to 235°C across a 26 min period. The injector and detector were maintained at 250°C. Identification of fatty acid peaks was performed by comparison with purified fatty acid samples obtained from Sigma-Aldrich, Co. (St. Louis, MO).

Prior to analysis, fecal and feed samples were homogenized and then finely ground through a 1 mm screen in a Retsch grinder (model ZM1; Retsch Inc., Newtown, PA). Acid

hydrolyzed ether extract (method 2003.06, AOAC International, 2007) was analyzed using a SoxCap SC 247 hydrolyzer and a Soxtec 255 semiautomatic extractor (FOSS North America, Eden Prairie, MN). Dry matter was determined according to modified methods (930.15, AOAC International, 2007) by drying samples in an oven at 105°C to a constant weight. Gross energy was determined using a bomb calorimeter (model 6200; Parr Instrument Co., Moline, IL). Benzoic acid (6.318 Mcal/kg; Parr Instruments) was used as the standard for calibration (determined GE: 6.320 ± 0.006 Mcal/kg). Titanium dioxide was determined by spectrophotometer (synergy 4; BioTek, Winooski, VT) according to the method of Leone (1973). All chemical analyses were performed in duplicate and repeated when intra-duplicate CV was greater than 1%.

Adipose tissue stored in TRIzol was homogenized using a Clean PowerGen 700D homogenizer (Fisher Science, Hanover Park, IL). Total RNA was then isolated from adipose tissue using TRIzol reagent following the manufacturer's protocol with the modification of repeating the RNA pellet wash step 3 times to reduce 230 nm contamination. Isolated RNA was then utilized for cDNA synthesis employing the QuantiTect Reverse Transcription Kit (Qiagen, Hilden, Germany). Abundance differences of mRNA were determined using qPCR (BioRad iCycler; Hercules, CA) on 12 genes. Expression normalization across samples within tissue was performed by calculating a delta Ct value for each sample using RPL32, as transcript abundance proved to be similar among treatments ($P < 0.05$).

Calculations

According to the equation of Oresanya et al. (2007), ATTD, % of AEE, DM, and GE was calculated as 100 – {100 × [concentration (g) of TiO_2 in diet × concentration of (g) of AEE, DM, or GE in feces]/[concentration (g) of TiO_2 in feces × concentration of AEE, DM, or GE in diet]}.

True total tract digestibility (**TTTD**; %) of AEE was calculated by correcting ATTD of AEE for endogenous fat losses at 20 g of AEE/kg of DM intake (Acosta Camargo et al., 2015).

Delta delta Ct (**ΔΔCt**) values were calculated from delta Ct values using a reference sample. Fold differences among treatments were calculated using the following equation $2^{|\Delta\Delta Ct(\text{treatment A}) - \Delta\Delta Ct(\text{treatment B})|}$. The fold difference among treatments are expressed where a positive value indicates an increase in transcript abundance and negative value indicates a decrease.

Iodine value was calculated from the fatty acid profile using the following equation: IV = (C16:1 × 0.95) + (C18:1 × 0.86) + (C18:2 × 1.732) + (C18:3 × 2.616) + (C20:1 × 0.785) + (C22:1 × 0.723); (AOCS, 1998).

Statistical Analysis

Analysis of the 9 treatments arranged as a 3 × 3 factorial, the main effects of environment (TN vs. PTFN vs. HS) and dietary fat (CNTR vs. CO vs. TAL), and their interactions (**E × DF**) were analyzed using PROC MIXED (SAS 9.4; SAS Inst. Inc., Cary, NC) with replicate as a random effect. Pig was the experimental unit. For each variable, normal distribution of residuals was tested using PROC UNIVARIATE.

Non-detectable fatty acid values were treated in all statistical analyses as 0. All *P*-values less than 0.05 were considered significant and *P*-values between 0.05 and 0.10 were considered trends.

RESULTS

Environment and Dietary Fat Effects on Rectal Temperature and Respiration Rate

As expected, during the 35 d experimental period HS pigs had an increased rectal temperature and greater than twice the respiration rate of TN and PFTN pigs ($P < 0.001$; Table 2). Dietary fat had no impact on either rectal temperature or respiration rate ($P \geq 0.203$). There was no E × DF interaction evident for rectal temperature or respiration rate, which indicates that HS pigs sustained a heat load indicative of marked heat stress and that dietary fat did not increase or decrease the degree of heat stress ($P \geq 0.192$).

Environment and Dietary Fat Effects on Growth Performance, Feed Intake, and Feed Efficiency

There were no E × DF interactions for ADG, ADFI, or gain to feed ratio ($P \geq 0.157$; Table 2). As expected, the ADG of TN was greater than PFTN and HS ($P < 0.001$). Dietary fat had no impact on ADG ($P \geq 0.413$; Table 2). As expected, the ADFI of TN was greater than HS, and by design the ADFI of HS and PFTN were not different ($P < 0.001$; Table 2). Overall, PFTN converted gain from feed with greater efficiency than HS ($P < 0.001$; Table 2). Overall, a CO-based diet tended to increase gain to feed ratio with TAL as the intermediate and CNTR as the least efficient ($P = 0.073$; Table 2). Part of the difference between the fat sources could be due to slight differences in their available energy content.

Environment and Dietary Fat Effects on Energy Intake and Caloric Efficiency

No E × DF interactions were evident for energy intake or caloric efficiency ($P \geq 0.477$; Table 3). By design, ME intake of HS and PFTN were similar and both were less than TN ($P < 0.001$). Barrows in the HS environment required more Mcal of ME to deposit 1 kg of BW or 1 kg of carcass weight than PFTN ($P \leq 0.021$). There was a tendency for barrows fed a TAL-based diet to consume less energy/d ($P = 0.090$), but there was no impact of dietary fat on caloric efficiency ($P \geq 0.654$).

Environment and Dietary Fat Effects on Digestibility of Dry Matter, Energy, and Lipids

No E × DF interactions were evident for digestibility of DM, GE, or AEE ($P \geq 0.253$; Table 4). No differences were evident among environment or dietary fat treatments for ATTD of DM ($P \leq 0.223$). The ATTD of GE was decreased in TN when compared with PFTN and HS ($P = 0.008$). Barrows in the HS environment compared to a TN environment tended to have decreased ATTD of AEE ($P = 0.055$), but not TTTD of AEE ($P = 0.118$).

The ATTD of GE, ATTD of AEE, and TTTD of AEE was increased for a CO-based diet compared with CNTR and TAL-based diet ($P \leq 0.012$; Table 4). Barrows on the CNTR diet had decreased ATTD of AEE than a TAL-based diet, but the difference between the 2 diets was not evident for TTTD of AEE ($P < 0.050$).

Environment and Dietary Fat Effects on Belly, Carcass, and Loin Characteristics

No interactions between E × DF were evident for any belly, carcass, or loin characteristics ($P \geq 0.215$; Table 5). The HCW and back fat was greater for TN than both PFTN and HS ($P \leq 0.011$). Carcasses from PFTN pigs tended to yield less ($P = 0.096$) and have increased fat free lean ($P = 0.089$). Loin depth was unaffected by environmental treatment ($P = 0.261$). The 3% CO diets resulted in decreased loin depth ($P = 0.006$), but HCW, yield, back fat depth, and fat free lean, were unaffected by dietary fat ($P \geq 0.129$).

Loin characteristics were unaffected by E × DF ($P \geq 0.495$; Table 5). Bellies from TN barrows had increased weight, middle thickness, and a star values ($P \leq 0.029$), and tended to have increased edge thickness ($P = 0.055$) than PFTN and HS bellies. Bellies from PFTN and HS barrows had increased l star values than TN bellies ($P = 0.021$). Environment did not affect b star values or belly firmness ($P \geq 0.243$). Bellies from barrows fed a CO-based diet were

heavier than bellies from those fed a TAL-based diet ($P = 0.018$). However, belly thickness, fat color, nor belly firmness was unaffected by dietary fat ($P \geq 0.215$).

Environment and Dietary Fat Effects on Fatty Acid Profile and Calculated Carcass Iodine Value

Oleic acid (**C18:1**) concentrations in jowl fat on d 7 collected from HS barrows tended to be less when fed either a CO-based diet or a TAL-based diet, but was greater in concentration when no additional fat was added in comparison to PFTN, resulting in a E × DF interaction ($P = 0.063$; Table 6). The sum of other minor saturated fatty acids increased in TN and HS pigs compared with PFTN ($P = 0.014$). Additionally, myristic acid tended to be greater in concentration in TN and HS jowl fat than PFTN ($P = 0.055$). The sum of other minor unsaturated fatty acids tended to increase in concentration in TN jowl fat ($P = 0.060$). Three percent TAL increased the concentration of eicosatrienoic acid ($P = 0.039$), while 3% CO tended to increase the concentration of linoleic acid (**C18:2**) ($P = 0.093$) in jowl fat collected on d 7. Environment or dietary fat did not alter IV, unsaturated to saturated fatty acid ratio (**U:S**), or omega-3 to omega-6 fatty acid ratio (**n-3:n-6**; $P \geq 0.167$).

In jowl fat collected on d 21 and d 35, no E × DF interactions were evident for fatty acid concentrations, IV, U:S, or n-3:n-6, and none of these parameters were impacted by environmental treatment ($P \leq 0.102$; Table 7 and 8). On d 21, C18:1 decreased ($P = 0.022$; Table 7), but C18:2 increased ($P < 0.001$) in barrows fed CO-based diets. These changes on d 21 caused jowl IV to increase and n-3:n-6 to decrease ($P < 0.001$); the U:S ($P = 0.063$) tended to decrease in barrows fed CO. On d 35, the use of 3% dietary CO resulted in decreased C18:1 ($P < 0.001$; Table 8). Feeding a CO-based diet also increased linoleic, linolenic and eicosadienoic

acid concentrations in jowl fat on d 35 ($P \leq 0.003$). These effects on d 35 caused jowl IV to increase and n3:n6 to decrease ($P < 0.001$).

Environment and Dietary Fat Effects on mRNA Abundance in Adipose Tissue

Interactions between E × DF were not evident for the mRNA abundance of *ACLY*, *ACSS2*, *ACACA*, *FASN*, *SCD*, *FADS2*, *EVOLV6*, *PRKAG1*, *PLIN1*, *ATGL*, *HSL*, and *INSR* in adipose tissue collected on d 7 ($P \geq 0.150$; Table 11). After 7 d of environmental treatment, the mRNA abundance of *ATGL* and *HSL* were less abundant in TN and HS barrows than in PFTN barrows ($P \leq 0.041$). The abundance of *SCD* mRNA was increased in HS barrows compared to TN barrows ($P = 0.047$). After 7 d of dietary treatment, mRNA abundance of *FASN* and *SCD* decreased in adipose tissue from barrows fed CO compared with barrows consuming the CNTR and TAL diets ($P \leq 0.011$; Table 12).

DISCUSSION

Pigs dissipate heat poorly, are highly insulated, lack functional sweat glands, and are densely housed during late finishing causing a high risk of susceptibility to HS (White et al., 2008; Qu et al., 2015). Heat stress imposes substantial changes in the physiological status of pigs, such as acid-base homeostasis (Patience et al., 2005) and is noted for suppressing feed intake (Hao et al., 2014; Pearce et al., 2014) and therefore energy intake of the pig (Renaudeau et al., 2013). Heat stress has a greater impact on pigs with a high rate of lean gain, resulting in reduced carcass lean gain and protein accretion (Nienaber et al., 1997; Brown-Brandl et al., 2000). Due to HS shifting the ratio of protein accretion to lipid deposition ratio and the reduced protein accretion rate, the AA requirement for TN pigs may be different than HS pigs (Nienaber

et al., 1997; Kerr et al., 2003). This assumes that the efficiency with which pigs use dietary amino acids does not change under heat stress conditions.

To alleviate HS suppressing feed intake, producers typically formulate diets on seasonal basis using ingredients with a low heat increment and greater energy density during the summer months (Stahly et al., 1981). Dietary fats and oils are ideal in meeting this ingredient description (Forbes and Swift, 1944; Kerr et al., 2015), and are therefore used more frequently and at higher dietary concentrations during the seasonally warm periods of the year. Unexpectedly, the data reported herein show that the pig's response to dietary fat is similar whether housed in a TN or a HS environment. Therefore, these data indicate that producers can anticipate that the inclusion of dietary fat in HS conditions will result in the same outcomes as including dietary fat in TN conditions.

However, it must be noted that while HS suppressed dietary energy intake by approximately 30% in comparison to contemporaries raised in TN conditions, the energy intake of HS barrows was still relatively high for this size of pig (Patience, 2012). This high energy intake is probably due to this experiment being conducted using pigs with a high health status housed in individual pens, where other stressors outside of ambient room temperature were kept to a minimum (White et al., 2015).

Certainly, the response to dietary energy intake is not easy to predict (Collins et al., 2009; Beaulieu et al., 2009), and it has been recently suggested that pigs that consume less energy are more likely to respond to increases in dietary energy concentration (Patience, 2012). Thus, the data reported herein should be complemented with data collected under differing feed intake conditions, including those representative of the industry, where daily ME intake for pigs of this

size may be between 9.0 (Graham et al, 2014) and 9.7 Mcal ME/d (Kellner et al, unpublished data).

Heat stress barrows had decreased mRNA abundance of genes involved in the lipolytic cascade (adipose triglyceride lipase and hormone sensitive lipase), which was similarly found by Sanz Fernandez et al. (2015a). These lipases hydrolyze fatty acids from the stored triglycerides in adipose tissue to be utilized as energetic fuel for protein accretion and maintenance processes throughout the body (Zimmerman et al., 2004). This result provides mechanistic evidence as to why HS pigs have decreased muscle mass and increased adiposity, a phenotype which has been demonstrated in HS pigs for nearly half a century (Close and Mount, 1971; Bridges et al., 1998). However, we did not find any upstream alteration of the lipolysis pathway via quantifying mRNA abundance of the AMPK regulatory subunit which has be implicated in regulating lipolytic lipases (Gaidhu et al., 2012; Sanz Fernandez et al., 2015a). The retention of stored triglycerides in adipose tissue during HS when energy intake is decreased is the opposite of what occurs during TN conditions when energy intake is decreased; unexpectedly, under TN conditions, there is a classic catabolic response where stored lipids are mobilized and circulating non-esterified fatty acid concentrations and whole-body oxidation is increased (Vernon, 1992). Reduced lipolysis in adipose tissue may be an attempt to reduce thermogenesis during mitochondrial fatty acid transport and beta-oxidation (Mujahid and Furuse, 2008). Another potential explanation, is insulin, an acute anabolic and anti-lipolytic hormone, which circulating concentrations are increased in a variety of species during HS (Baumgard and Rhoads, 2013).

Previous research has indicated that HS in pigs is not simply a suppression of lipolysis, it directly suppresses protein accretion and the rate of lean carcass gain (Neinaber et al., 1997), and results in a whole-body alteration of nutrient partitioning to a phenotype of increased adiposity

due to increased insulin activity (Pearce et al., 2013; Sanz Fernandez et al., 2015a, b). An increase in whole-body insulin action is a conserved HS response across a multitude of species (Baumgard and Rhodes, 2013). Recent findings support this whole-body change in HS pigs. For example, Qu et al. (2015) found that HS increased the expression of genes involved in de novo lipogenesis and fatty acid uptake in adipose tissue, and Sanz Fernandez et al. (2015b) found HS increased whole-body insulin sensitivity. Furthermore, in utero HS alters the hierarchy of future nutrient partitioning resulting in a fatter phenotype at market (Johnson et al., 2015).

The direction of storing recently digested dietary lipids and retaining stored body lipids versus mobilizing and then utilizing lipids as an energy source for protein deposition and maintenance processes may explain why HS pigs are less caloric efficient. The energetic cost of a gram of deposited lipid is approximately 1.6 kcal of ME more than a gram of deposited protein (van Milgen and Noblet, 2003; Barea et al., 2010; Patience, 2012).

Despite HS altering lipid metabolism and increasing mRNA abundance of stearoyl CoA desaturase (delta-9-desaturase) in adipose tissue, HS had no significant effect on the carcass IV and fatty acid composition on d 7, 21, or at market (d 35). This suggests that any seasonal pork fat quality issues are most likely due to decreased carcass weight and belly weight and thickness and not due to HS resulting in carcass fat with increased concentrations of unsaturated fatty acids. A recent finding by Seibert et al. (2015) demonstrated that adipose tissue of HS pigs contained a greater percentage of water than their TN contemporaries; which is consistent with pigs that are limit fed or leaner in phenotype having less lipid relative to water, indicative of small adipocyte size (Gnaedinger et al., 1963). Seibert et al. (2015) also reported that exposure to HS did not alter the fatty acid profile of adipose tissue. Similar to the data reported herein, White et al. (2008) found that when stocking density was adequate, HS increased stearoyl CoA

desaturase mRNA abundance, but did not alter fatty acid synthase or carcass IV. However, when floor space was reduced from 0.93 m^2/pig to 0.66 m^2/pig in combination with HS, there was a further decrease in energy intake, and a significant increase in adipose tissue stearoyl CoA desaturase mRNA abundance, fatty acid synthase mRNA abundance, and carcass IV by approximately 4 g/100g (White et al., 2008). Under commercial stocking densities (eg, 0.70 m^2/pig) carcass IV values can be 2 to 10 g/100g greater than individually fed pigs under TN conditions (Kellner et al., 2016). Thus, HS pigs densely stocked in commercial production maybe at a greater risk of exceeding carcass IV standards. An interaction between stocking density and HS was also reported to reduce rate of gain (Kerr et al., 2005). In sum, these studies suggest that if HS pigs have adequate floor space and additional stressors are minimal, the pig can sustain a minimum level of energy intake such that no impact of carcass IV will be evident.

Pigs that are limit fed have been noted to have carcasses that are leaner and have greater carcass IVs (Madsen et al., 1992). The data herein agree with this phenotype as the PFTN carcasses tended to be leaner and had numerically higher carcass IVs than TN and HS carcasses.

Since the first demonstration by Ellis and Isbell (1923), it has become accepted that the fatty acid composition of carcass fat will be highly reflective of the dietary fatty acid composition (Apple et al., 2009; Kellner et al., 2015). The data reported herein reveal that the degree of unsaturation in dietary fat also modulate genes involved in de novo lipogenesis (Jump, 2002; Duran-Montge et al. 2009). Use of an unsaturated dietary fat (CO) versus a saturated fat (TAL) increased the mRNA abundance of fatty acid synthase. It has been demonstrated that dietary saturated fatty acids, in comparison with unsaturated fatty acids and in particular omega-6 fatty acids, suppress fatty acid synthase and de novo lipogenesis (Waterman et al.; 1975, Kouba et al., 1999; Duran-Montge et al, 2009). Dietary saturated fatty acids suppressing

lipogenesis is not always a consistent response as Hsu et al. (2004) has shown; in their study, the mRNA abundance of fatty acid synthase was similar between diets with TAL or docosahexaenoic acid. Similarly, Allee et al. (1972) showed that CO and TAL suppressed lipogenesis to the same degree. De novo lipogenesis in the pig synthesizes saturated or monounsaturated fatty acids of either 16 or 18 carbons (Kloreag et al., 2007). Thus, feeding a saturated fat source would suppress the further production of similar saturated and monounsaturated fatty acids via lipogenesis and feeding an unsaturated dietary fat source would not have the same effect.

Heat stress has been reported to compromise the pig's intestinal integrity and morphology (Pearce et al., 2014); these negative effects are largely independent of reduced feed intake (Pearce et al., 2015). The data reported herein indicates the differences between HS and TN barrows in terms of the ATTD of GE and AEE were minimal after 17 d of HS exposure, and that there was no significant difference evident for TTTD of AEE. The use of CO resulted in greater ATTD of GE and AEE and TTTD of AEE. The increase in digestibility of a more unsaturated dietary fat source versus a saturated fat source has been previously shown (Wiseman et al., 1990; Kerr et al., 2009; Kil et al., 2010). However, more work is needed to validate if unsaturated dietary fat sources have increased levels of DE and ME than saturated fat sources (Powels et al., 1995; NRC, 2012).

In conclusion, HS does not alter the pig's response to dietary fat. However, HS results in reduced growth, feed intake, caloric and feed efficiency, and a suppression of mRNA abundance of genes involved in the lipolytic cascade which may contribute to fatter carcasses.

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Table 1. Ingredient composition (as-fed basis) of the experimental diets formulated with no added fat (control), 3% corn oil, or 3% tallow

Ingredient, %	Control	3% Corn oil	3% Tallow
Corn	84.36	79.74	79.74
Soybean meal (46.5% CP)	12.71	14.35	14.35
Corn oil	-	3.00	-
Tallow	-	-	3.00
Limestone	0.90	0.90	0.90
Monocalcium phosphate	0.56	0.53	0.53
Salt	0.50	0.50	0.50
L-lysine HCL	0.15	0.15	0.15
DL-methionine	-	0.01	0.01
L-threonine	0.01	0.01	0.01
Vitamin premix[1]	0.20	0.20	0.20
Trace mineral premix[2]	0.15	0.15	0.15
Titanium dioxide	0.40	0.40	0.40
Santoquin[3]	0.06	0.06	0.06
Formulated composition			
Standard ileal digestible AA, %			
Lysine	0.61	0.64	0.64
Methionine	0.20	0.21	0.21
Methionine + Cysteine	0.41	0.42	0.42
Threonine	0.39	0.41	0.41
Tryptophan	0.12	0.12	0.12
Calculated composition			
NE, Mcal/kg	2.54	2.67	2.67
Heat increment[4], Mcal/kg	1.16	1.34	1.18
ME[5], Mcal/kg	3.70	3.90	3.85
Analyzed composition			
DM, %	88.65	89.01	88.39
GE, Mcal/kg	3.81	4.01	3.95
Crude protein (N × 6.25), %	13.16	13.56	13.55
Crude fat, %	3.18	6.21	6.22

Dietary fat IV[6], g/100g	-	123.0	41.8
Diet IV[7], g/100g	117.9	120.8	84.6
Diet IVP[8]	37.5	75.0	52.6

[1]Provided 6,614 IU vitamin A, 827 IU vitamin D, 26 IU vitamin E, 2.6 mg vitamin K, 29.8 mg niacin, 16.5 mg pantothenic acid, 5.0 mg riboflavin, and 0.023 mg vitamin B12 per kg of diet.

[2]Provided 165 mg Zn (zinc sulfate), 165 mg Fe (iron sulfate), 39 mg Mn (manganese sulfate), 17 mg Cu (copper sulfate), 0.3 mg I (calcium iodate), and 0.3 mg Se (sodium selenite) per kg of diet.

[3]Santoquin Mixture 6 (Feed and forage Anti-oxidant; NOVUS International, Saint Charles, MO).

[4]Heat increment = ME - NE

[5]ME = DE × [1.003 – (0.0021 × CP)] (Noblet and Perez, 1993).

[6]Iodine value (IV) determined via titration (Barrow-Agee Labs, Memphis, TN).

[7]Iodine value calculated from fatty acid composition: IV = [C16:1] × 0.95 + [C18:1] × 0.86 + [C18:2] × 1.732 + [C18:3] × 2.616 + [C20:1] × 0.785 + [C22:1] × 0.723; brackets indicate concentration (AOCS, 1998).

[8]Iodine value product (IVP) = (IV of the dietary lipids) × (% dietary lipid) × 0.10 (Christensen, 1962; Madsen et al., 1992).

Table 2. Effects of ad libitum feed intake in thermal neutral conditions (TN)[1], pair feeding in thermal neutral conditions (PFTN)[1,2], or heat stress (HS)[3], additional inclusion of no dietary fat (CNTR), 3% tallow (TAL), or 3% corn oil (CO) on daily respiration rate (RR), rectal temperature (RT), growth performance, and feed efficiency d 0 to 35

	Environment					Dietary fat					E × DF[4]
	Treatment					Treatment					
Item	TN	PFTN	HS	SEM	*P*-value	CNTR	CO	TAL	SEM	*P*-value	*P*-value
Initial BW, kg[5]	101.5	99.9	100.5	0.9	0.406	100.6	101.2	100.0	0.9	0.644	0.903
Final BW, kg[6]	137.0	127.2	125.0	1.3	<0.001	129.5	131.1	128.6	1.3	0.366	0.867
RR, breaths/min	36.3[b]	34.2[b]	78.3[a]	1.6	<0.001	50.2	49.0	49.6	1.7	0.692	0.904
RT, °C	38.1[b]	38.2[b]	39.0[a]	0.1	<0.001	38.4	38.4	38.5	0.1	0.653	0.192
ADG, kg	1.03[a]	0.77[b]	0.72[b]	0.03	<0.001	0.83	0.87	0.83	0.03	0.492	0.413
ADFI, kg	3.46[a]	2.49[b]	2.49[b]	0.10	<0.001	2.89	2.82	2.72	0.10	0.124	0.978
G:F	0.301[ab]	0.319[a]	0.290[b]	0.013	0.006	0.292	0.314	0.303	0.013	0.073	0.500

[a-c]Within a row, least squares means lacking a common superscript letter differ due to effect of environment, $P < 0.05$.

[1]Constant thermal neutral environment of ~24.0°C.

[2]Limit-fed based on HS feed intake on the previous day

[3]Diunral heat stress environment of ~33.0°C between 0800 h to 2000 h and ~28.0°C 2000 h to 0800 h from d 0 to d 7, ~33.5°C between 0800 h to 2000 h and ~28.0°C 2000 h to 0800 h for d 7 to d 14, ~34.0°C between 0800 h to 2000 h and ~28.0°C 2000 h to 0800 h for d 14 to d 21, ~34.5°C between 0800 h to 2000 h and ~28.0°C 2000 h to 0800 h for d 21 to d 28, and ~35.0°C between 0800 h to 2000 h and ~28.0°C 2000 h to 0800 h for d 28 to d 35.

[4]Probability value for environment × dietary fat interaction (E × DF).

[5]d 0.

[6]d 35.

Table 3. Effects of ad libitum feed intake in thermal neutral conditions (TN)[1], pair feeding in thermal neutral conditions (PFTN)[1,2], or heat stress (HS)[3], additional inclusion of no dietary fat (CNTR), 3% tallow (TAL), or 3% corn oil (CO) on energy intake and caloric efficiency

	Environment					Dietary fat					E × DF[4]
	Treatment					Treatment					
Item	TN	PFTN	HS	SEM	*P*-value	CNTR	CO	TAL	SEM	*P*-value	*P*-value
ME intake, Mcal/d	13.1[a]	9.6[b]	9.5[b]	0.4	<0.001	10.7	11.0	10.4	0.4	0.090	0.990
ME intake:BW gain	12.8[ab]	12.2[b]	13.4[a]	0.7	0.013	12.8	12.6	13.0	0.7	0.654	0.477
ME intake:carcass gain	17.2[ab]	16.6[b]	18.1[a]	1.0	0.021	17.4	17.1	17.5	1.0	0.786	0.509

[a-c]Within a row, least squares means lacking a common superscript letter differ due to effect of environment, $P < 0.05$.

[1]Refer to Footnote 1 in Table 2.

[2]Refer to Footnote 2 in Table 2.

[3]Refer to Footnote 3 in Table 2.

[4]Probability value for environment × dietary fat interaction (E × DF).

Table 4. Effects of ad libitum feed intake in thermal neutral conditions (TN)[1], pair feeding in thermal neutral conditions (PFTN)[1,2], or heat stress (HS)[3], additional inclusion of no dietary fat (CNTR), 3% tallow (TAL), or 3% corn oil (CO) on apparent total tract digestibility (ATTD)[4] and true total tract digestibility (TTTD)[5] of DM, GE, and acid hydrolyzed ether extract (AEE)

	Environment					Dietary fat					E × DF[6]
	Treatment					Treatment					
Item	TN	PFTN	HS	SEM	*P*-value	CNTR	CO	TAL	SEM	*P*-value	*P*-value
ATTD, %											
DM	88.0	88.7	88.4	0.2	0.223	88.4	88.5	88.2	0.2	0.524	0.253
GE	88.2^{b}	89.1^{a}	88.8^{a}	0.2	0.008	88.4^{y}	89.1^{x}	88.6^{y}	0.2	0.011	0.525
AEE	60.2	61.4	59.0	0.8	0.055	41.5^{z}	71.2^{x}	67.8^{y}	0.8	<0.001	0.886
TTTD, %											
AEE	97.9	98.5	96.7	0.7	0.118	97.3^{y}	99.3^{x}	96.3^{y}	0.7	0.012	0.932

[a-c]Within a row, least squares means lacking a common superscript letter differ due to effect of environment, $P < 0.05$.

[x-z]Within a row, least squares means lacking a common superscript letter differ due to effect of dietary fat, $P < 0.05$.

[1]Refer to Footnote 1 in Table 2.

[2]Refer to Footnote 2 in Table 2.

[3]Refer to Footnote 3 in Table 2.

[4]Apparent total tract digestibility (ATTD; %) of either AEE, DM, or GE was calculated as 100 – {100 × [concentration (g) of TiO_2 in diet × concentration of (g) of AEE, DM, or GE in feces]/[concentration (g) of TiO_2 in feces × concentration of AEE, DM, or GE in diet]}; (Oresanya et al. 2007).

[5]Calculated via correcting ATTD of AEE for endogenous fat losses at 20 g of AEE/kg of DM intake.

[6]Probability value for environment × dietary fat interaction (E × DF).

Table 5. Effects of ad libitum feed intake in thermal neutral conditions (TN)[1], pair feeding in thermal neutral conditions (PFTN)[1,2], or heat stress (HS)[3], additional inclusion of no dietary fat (CNTR), 3% tallow (TAL), or 3% corn oil (CO) on carcass characteristics

	Environment					Dietary fat					E × DF[4]
	Treatment					Treatment					
Item	TN	PFTN	HS	SEM	*P*-value	CNTR	CO	TAL	SEM	*P*-value	*P*-value
HCW, kg	101.5[a]	93.1[b]	92.1[b]	1.2	<0.001	95.2	96.5	95.1	1.2	0.554	0.827
Yield, %	74.1	73.2	73.7	0.4	0.096	73.5	73.6	74.0	0.4	0.407	0.600
Loin depth, cm	6.23	5.95	5.97	0.23	0.261	6.11[x]	5.73[y]	6.30[x]	0.24	0.006	0.387
Back fat, cm	2.29[a]	1.99[b]	2.10[b]	0.21	0.011	2.19	2.14	2.06	0.21	0.353	0.854
Fat free lean, %	52.4	53.9	53.2	1.5	0.089	52.9	52.7	52.9	1.6	0.129	0.774
Loin characteristics											
Ultimate pH	5.6	5.6	5.6	0.1	0.873	5.6	5.6	5.7	0.1	0.199	0.640
LCS[5]	3.2	3.0	3.1	0.1	0.561	3.0	3.1	3.1	0.1	0.806	0.693
LMS[6]	1.8	1.8	1.7	0.1	0.495	1.7	1.7	1.8	0.1	0.829	0.515
Belly characteristics											
Belly weight, kg	8.6[a]	7.5[b]	7.8[b]	0.2	<0.001	8.0[xy]	8.3[x]	7.7[y]	0.2	0.018	0.372
Belly ET[7], cm	3.11	2.81	2.76	0.25	0.055	2.94	2.88	2.86	0.25	0.856	0.313
Belly MT[8], cm	2.47[a]	2.23[b]	2.20[b]	0.08	0.029	2.28	2.36	2.25	0.08	0.568	0.919
l star	71.8[b]	73.2[a]	73.4[a]	0.6	0.021	73.4	72.6	72.4	0.6	0.177	0.309
a star	11.6[a]	9.9[b]	10.4[b]	0.4	0.003	10.3	10.7	10.9	0.4	0.452	0.318
b star	7.7	7.3	7.4	0.2	0.303	7.3	7.6	7.5	0.2	0.210	0.215
Durometer	44.4	41.9	42.7	2.5	0.682	44.7	42.6	41.8	2.4	0.547	0.687
Belly firmness[9]	2.2	2.4	2.4	0.1	0.243	2.3	2.5	2.2	0.1	0.220	0.720

[a-c]Within a row, least squares means lacking a common superscript letter differ due to effect of environment, $P < 0.05$.

[x-z]Within a row, least squares means lacking a common superscript letter differ due to effect of dietary fat, $P < 0.05$.

[1]Refer to Footnote 1 in Table 2.

[2]Refer to Footnote 2 in Table 2.

[3]Refer to Footnote 3 in Table 2.

[4]Probability value for environment × dietary fat interaction (E × DF).

[5]Loin Color Score; evaluated postmortem according to the Japanese color bar 1 to 6 scale, 1 = extremely light, 6 = extremely dark (Sullivan et al., 2007).

[6]Loin Marbling Score; evaluated postmortem according to National Pork Board Standards (NPPC, 2000). The marbling standards correspond to percentage of intramuscular lipid.

[7]Measured in the middle scribe side of the belly.

[8]Measured in the middle of the belly.

[9]Measured by a subjective flop test with a score of 1, 2, or 3 with 1 being the firmest.

Table 6. Effects of ad libitum feed intake in thermal neutral conditions (TN)[1], pair feeding in thermal neutral conditions (PFTN)[1,2], or heat stress (HS)[3], additional inclusion of no dietary fat (CNTR), 3% tallow (TAL), or 3% corn oil (CO) on fatty acid profile and calculated iodine value (IV)[4] of jowl fat on d 7

	Environment					Dietary fat					E × DF[5]
	Treatment					Treatment					
Item	TN	PFTN	HS	SEM	*P*-value	CNTR	CO	TAL	SEM	*P*-value	*P*-value
Fatty acid[6], %											
C12:0, %	0.04	0.04	0.04	0.01	0.655	0.04	0.04	0.04	0.01	0.925	0.112
C13:0, %	0.04	0.04	0.04	0.01	0.623	0.04	0.04	0.04	0.01	0.936	0.372
C14:0, %	1.10	1.05	1.12	0.02	0.055	1.11	1.06	1.10	0.02	0.210	0.557
C15:0, %	0.04	0.04	0.04	0.01	0.516	0.03	0.04	0.04	0.01	0.592	0.398
C16:0, %	22.37	22.03	22.36	0.20	0.440	22.41	22.25	22.09	0.20	0.525	0.566
C16:1, %	2.44	2.22	2.32	0.13	0.270	2.46	2.29	2.23	0.13	0.169	0.848
C17:0, %	0.54	0.55	0.53	0.07	0.845	0.54	0.52	0.56	0.07	0.477	0.786
C17:1, %	0.36	0.36	0.37	0.04	0.882	0.37	0.35	0.38	0.04	0.323	0.372
C18:0, %	10.83	11.23	11.20	0.34	0.461	10.98	11.13	11.15	0.34	0.861	0.475
C18:1, %	44.36	44.61	43.66	0.35	0.101	44.50	43.63	44.50	0.35	0.140	0.063
C18:2, %	14.80	14.86	15.23	0.36	0.527	14.51	15.55	14.84	0.36	0.093	0.752
C18:3, %	0.63	0.64	0.67	0.03	0.117	0.64	0.66	0.65	0.03	0.556	0.957
C20:0, %	0.12	0.09	0.14	0.03	0.250	0.08	0.13	0.14	0.03	0.124	0.291
C20:1, %	0.93	0.92	0.90	0.06	0.468	0.93	0.89	0.93	0.06	0.305	0.495
C20:2, %	0.78	0.79	0.76	0.03	0.659	0.77	0.80	0.77	0.03	0.669	0.444
C20:3, %	0.11	0.09	0.09	0.01	0.369	0.07^{y}	0.10^{xy}	0.12^{x}	0.01	0.039	0.760
C22:1, %	0.30	0.30	0.30	0.02	0.958	0.29	0.30	0.29	0.02	0.814	0.450
Other SFA[7], %	0.15^{a}	0.11^{b}	0.14^{a}	0.02	0.014	0.13	0.13	0.13	0.02	0.939	0.186
Other UFA[8], %	0.07	0.05	0.04	0.01	0.060	0.05	0.05	0.06	0.01	0.795	0.194
U:S[9]	1.84	1.85	1.81	0.03	0.544	1.83	1.83	1.84	0.03	0.956	0.311
IV, g/100g	68.7	68.8	68.8	0.50	0.976	68.4	69.3	68.7	0.05	0.425	0.929
n-3:n-6[10]	0.049	0.048	0.049	0.003	0.563	0.048	0.048	0.050	0.003	0.167	0.757

[a-c]Within a row, least squares means lacking a common superscript letter differ due to effect of environment, $P < 0.05$.

[x-z]Within a row, least squares means lacking a common superscript letter differ due to effect of dietary fat, $P < 0.05$.

[1]Refer to Footnote 1 in Table 2.

[2]Refer to Footnote 2 in Table 2.

[3]Refer to Footnote 3 in Table 2.

[4]Iodine value was calculated by: [C16:1] × 0.95 + [C18:1] × 0.86 + [C18:2] × 1.732 + [C18:3] × 2.616 + [C20:1] × 0.785 + [C22:1] × 0.723; brackets indicate percentage concentration (AOCS, 1998).

[5]Probability value for environment × dietary fat interaction (E × DF).

[6]Lauric acid (C12:0), tridecanoic acid (C13:0), myristic acid (C14:0), pentadecanoic acid (C15:0), palmitic acid (C16:0), palmitoleic acid (C16:1), margaric acid (C17:0), heptadecenoic acid (C17:1), stearic acid (C18:0), oleic acid (C18:1), linoleic acid (C18:2), linolenic acid (C18:3), arachidic acid (C20:0), gadoleic acid (C20:1), eicosadienoic acid (C20:2), eicosatrienoic acid (C20:3), docosenoic acid (C22:1).

[7]Saturated fatty acids.

[8]Unsaturated fatty acids.

[9]Unsaturated to saturated fatty acid ratio.

[10]Omega-3 fatty acid to Omega-6 fatty acid ratio.

Table 7. Effects of ad libitum feed intake in thermal neutral conditions (TN)[1], pair feeding in thermal neutral conditions (PFTN)[1,2], or heat stress (HS)[3], additional inclusion of no dietary fat (CNTR), 3% tallow (TAL), or 3% corn oil (CO) on fatty acid profile and calculated iodine value (IV)[4] of jowl fat on d 21

	Environment					Dietary fat					E × DF[5]
	Treatment					Treatment					
Item	TN	PFTN	HS	SEM	*P*-value	CNTR	CO	TAL	SEM	*P*-value	*P*-value
Fatty acid[6], %											
C12:0, %	0.05	0.04	0.04	0.01	0.102	0.05	0.04	0.04	0.01	0.479	0.829
C13:0, %	0.04	0.03	0.05	0.01	0.158	0.04	0.04	0.04	0.01	0.917	0.986
C14:0, %	1.13	1.07	1.12	0.03	0.109	1.12	1.10	1.10	0.03	0.785	0.454
C15:0, %	0.04	0.03	0.03	0.01	0.867	0.03	0.04	0.03	0.01	0.886	0.949
C16:0, %	22.22	21.80	21.90	0.20	0.370	22.18	21.73	22.00	0.20	0.294	0.768
C16:1, %	2.57	2.43	2.46	0.11	0.574	2.47	2.49	2.51	0.11	0.951	0.382
C17:0, %	0.49	0.47	0.51	0.05	0.540	0.48	0.48	0.51	0.05	0.496	0.264
C17:1, %	0.34	0.35	0.36	0.04	0.525	0.36	0.33	0.36	0.04	0.311	0.778
C18:0, %	10.44	10.49	10.42	0.36	0.970	10.64	10.12	10.58	0.36	0.162	0.662
C18:1, %	45.91	46.06	45.36	0.49	0.349	45.73^{xy}	45.01^{y}	46.60^{x}	0.49	0.022	0.251
C18:2, %	13.78	14.24	14.65	0.36	0.197	13.89^{y}	15.57^{x}	13.20^{y}	0.36	<0.001	0.473
C18:3, %	0.58	0.61	0.63	0.03	0.125	0.61	0.63	0.58	0.03	0.124	0.818
C20:0, %	0.11	0.09	0.12	0.03	0.659	0.10	0.09	0.14	0.03	0.420	0.810
C20:1, %	0.99	0.98	0.96	0.06	0.697	0.98	0.95	1.01	0.06	0.340	0.194
C20:2, %	0.72	0.77	0.75	0.04	0.696	0.73	0.77	0.74	0.04	0.717	0.159
C20:3, %	0.10	0.08	0.09	0.02	0.618	0.10	0.08	0.09	0.02	0.449	0.149
C22:1, %	0.26	0.27	0.27	0.02	0.848	0.26	0.27	0.27	0.02	0.857	0.310
Other SFA[7], %	0.12	0.11	0.13	0.01	0.238	0.13	0.12	0.12	0.01	0.537	0.508
Other UFA[8], %	0.07	0.05	0.05	0.01	0.134	0.05	0.05	0.06	0.01	0.804	0.169
U:S[9]	1.90	1.94	1.92	0.04	0.659	1.88	1.97	1.90	0.04	0.063	0.812
IV, g/100g	68.3	69.2	69.3	0.7	0.259	68.3^{y}	70.6^{x}	67.8^{y}	0.7	<0.001	0.960
n-3:n-6[10]	0.048	0.048	0.048	0.004	0.860	0.050^{x}	0.045^{y}	0.049^{x}	0.004	<0.001	0.146

[a-c]Within a row, least squares means lacking a common superscript letter differ due to effect of environment, $P < 0.05$.

[x-z]Within a row, least squares means lacking a common superscript letter differ due to effect of dietary fat, $P < 0.05$.

[1]Refer to Footnote 1 in Table 2.

[2]Refer to Footnote 2 in Table 2.

[3]Refer to Footnote 3 in Table 2.

[4]Iodine value was calculated by: [C16:1] × 0.95 + [C18:1] × 0.86 + [C18:2] × 1.732 + [C18:3] × 2.616 + [C20:1] × 0.785 + [C22:1] × 0.723; brackets indicate percentage concentration (AOCS, 1998).

[5]Probability value for environment × dietary fat interaction (E × DF).

[6]Lauric acid (C12:0), tridecanoic acid (C13:0), myristic acid (C14:0), pentadecanoic acid (C15:0), palmitic acid (C16:0), palmitoleic acid (C16:1), margaric acid (C17:0), heptadecenoic acid (C17:1), stearic acid (C18:0), oleic acid (C18:1), linoleic acid (C18:2), linolenic acid (C18:3), arachidic acid (C20:0), gadoleic acid (C20:1), eicosadienoic acid (C20:2), eicosatrienoic acid (C20:3), docosenoic acid (C22:1).

[7]Saturated fatty acids.

[8]Unsaturated fatty acids.

[9]Unsaturated to saturated fatty acid ratio.

[10]Omega-3 fatty acid to Omega-6 fatty acid ratio.

Table 8. Effects of ad libitum feed intake in thermal neutral conditions (TN)[1], pair feeding in thermal neutral conditions (PFTN)[1,2], or heat stress (HS)[3], additional inclusion of no dietary fat (CNTR), 3% tallow (TAL), or 3% corn oil (CO) on fatty acid profile and calculated iodine value (IV)[4] of jowl fat on d 35

	Environment					Dietary fat					E × DF[5]
	Treatment					Treatment					
Item	TN	PFTN	HS	SEM	*P*-value	CNTR	CO	TAL	SEM	*P*-value	*P*-value
Fatty acid[6], %											
C12:0, %	0.04	0.04	0.04	0.01	0.315	0.04	0.04	0.04	0.01	0.913	0.710
C14:0, %	1.11	1.04	1.08	0.03	0.257	1.08	1.07	1.08	0.03	0.902	0.955
C15:0, %	0.04	0.04	0.03	0.01	0.294	0.03	0.04	0.04	0.01	0.054	0.168
C16:0, %	21.88	21.36	21.72	0.19	0.211	21.72	21.47	21.78	0.19	0.508	0.580
C16:1, %	2.39	2.24	2.36	0.08	0.338	2.41	2.26	2.31	0.08	0.327	0.477
C17:0, %	0.38	0.41	0.40	0.04	0.427	0.38	0.39	0.42	0.04	0.089	0.129
C17:1, %	0.36	0.38	0.36	0.03	0.492	0.36	0.36	0.38	0.03	0.302	0.162
C18:0, %	10.51	10.43	10.70	0.41	0.529	10.53	10.29	10.82	0.41	0.162	0.138
C18:1, %	45.88	46.59	45.99	0.45	0.497	47.14^{x}	44.65^{y}	46.67^{x}	0.46	<0.001	0.178
C18:2, %	14.40	14.41	14.30	0.37	0.961	13.73^{y}	16.30^{x}	13.44^{y}	0.37	<0.001	0.116
C18:3, %	0.62	0.64	0.64	0.02	0.707	0.60^{y}	0.68^{x}	0.61^{y}	0.03	0.003	0.533
C20:0, %	0.15	0.15	0.14	0.01	0.600	0.14	0.15	0.15	0.01	0.705	0.167
C20:1, %	0.94	0.96	0.94	0.03	0.767	0.97	0.92	0.95	0.03	0.351	0.245
C20:2, %	0.76	0.78	0.76	0.02	0.658	0.73^{y}	0.84^{x}	0.73^{y}	0.02	<0.001	0.494
C20:3, %	0.11	0.11	0.11	0.01	0.872	0.10	0.11	0.11	0.01	0.127	0.882
C22:1, %	0.27	0.27	0.28	0.01	0.649	0.26	0.29	0.27	0.01	0.082	0.304
Other SFA[7], %	0.11	0.11	0.11	0.01	0.839	0.11	0.11	0.11	0.01	0.965	0.269
Other UFA[8], %	0.03	0.05	0.03	0.01	0.260	0.03	0.03	0.05	0.01	0.068	0.645
U:S[9]	1.93	1.98	1.93	0.05	0.370	1.95	1.99	1.91	0.05	0.164	0.185
IV, g/100g	69.3	69.8	69.2	0.7	0.624	68.5^{y}	71.5^{x}	68.2^{y}	0.7	<0.001	0.197
n-3:n-6[10]	0.050	0.051	0.051	0.003	0.216	0.051^{y}	0.047^{z}	0.053^{x}	0.003	<0.001	0.115

[a-c]Within a row, least squares means lacking a common superscript letter differ due to effect of environment, $P < 0.05$.

[x-z]Within a row, least squares means lacking a common superscript letter differ due to effect of dietary fat, $P < 0.05$.

[1]Refer to Footnote 1 in Table 2.

[2]Refer to Footnote 2 in Table 2.

[3]Refer to Footnote 3 in Table 2.

[4]Iodine value was calculated by: [C16:1] × 0.95 + [C18:1] × 0.86 + [C18:2] × 1.732 + [C18:3] × 2.616 + [C20:1] × 0.785 + [C22:1] × 0.723; brackets indicate percentage concentration (AOCS, 1998).

[5]Probability value for environment × dietary fat interaction (E × DF).

[6]Lauric acid (C12:0), tridecanoic acid (C13:0), myristic acid (C14:0), pentadecanoic acid (C15:0), palmitic acid (C16:0), palmitoleic acid (C16:1), margaric acid (C17:0), heptadecenoic acid (C17:1), stearic acid (C18:0), oleic acid (C18:1), linoleic acid (C18:2), linolenic acid (C18:3), arachidic acid (C20:0), gadoleic acid (C20:1), eicosadienoic acid (C20:2), eicosatrienoic acid (C20:3), docosenoic acid (C22:1).

[7]Saturated fatty acids.

[8]Unsaturated fatty acids.

[9]Unsaturated to saturated fatty acid ratio.

[10]Omega-3 fatty acid to Omega-6 fatty acid ratio.

Table 9. Effects of ad-libitum feed intake in thermal neutral conditions (TN)[1], pair-feeding in thermal neutral conditions (PFTN)[1,2], or heat stress (HS)[3] on mRNA abundance in adipose tissue on d 7[4]

			Environment, $\Delta\Delta C_t$[5]				Fdiff[6]			
Gene	Description	Primers, 5`-3`	TN	PFTN	HS	SEM	TN vs. PFTN	HS vs. TN	HS vs. PFTN	*P*-value[7]
ACLY	ATP citrate lyase	F:AGGAGGAGTTCTATGTCTGC[8] R:CAACAGGTGTTTCTTGATGGCC[9]	0.30	-0.64	0.21	0.63	-1.91	1.06	-1.80	0.537
ACSS2	Acyl-CoA synthetase short-chain family member 2	F:TGTGAACCTGAAGGAGCTGG R:ACAATGCAGCATCTCACTGG	0.23	-0.38	-0.45	0.72	-1.52	1.60	1.05	0.633
ACACA	Acetyl CoA carboxylase	F:ATGGATGAACCGTCTCCC R:TGTAAGGCCAAGCCATCC	-0.20	-0.56	0.27	1.25	-1.28	-1.39	-1.78	0.517
FASN	Fatty acid synthase	F:CACAACTCCAAAGACACG R:AGGAACTCGGACATAGCG	-0.42	-0.23	-1.15	0.81	1.14	1.66	1.89	0.249
SCD	Stearoyl CoA desaturase (delta-9-desaturase)	F:TACTATCTGCTGAGTGCTGTGG R:CTGGAATGCCATCGTGTTGG	0.48[a]	-0.29[ab]	-2.13[b]	1.19	-1.71	6.11	3.58	0.047
FADS2	Fatty acid desaturase 2 (delta-6-desaturase)	F:GCCTTCATCCTTGCTACC R:AGATGGCCGTAATCGTGC	0.89	-1.02	0.33	1.35	-3.76	1.47	-2.55	0.295
EVOLV6	Fatty acid elongase 6	F:CTGGTTTCTGCTCTGTATGC R:ACCTGAACACTGCAAGGC	0.63	-0.31	0.80	0.81	-1.91	-1.13	-2.16	0.542
PRKAG1	Protein kinase, AMP-activated, gamma 1 non-catalytic subunit	F:TTGGTGACTAATGGTGTCCG R:TGAAATCAGTGATGGTCAGC	0.36	0.02	0.30	1.84	-1.27	1.04	-1.21	0.889
PLIN1	Perilipin 1	F:GAGTGCTTCCAGAAGACC R:GATGCCCTTCTCGTAAGC	0.35	0.45	-0.85	1.60	1.07	2.30	2.46	0.418
ATGL (PNPLA2)	Adipose triglyceride lipase (Patatin-like phospholipase domain containing 2)	F:ATCATAACCCACTTCGCC R:ACACGGGAATGAAGGTGC	0.08[a]	-1.80[b]	1.15[a]	0.88	-3.68	-2.10	-7.73	<0.001
HSL	Hormone sensitive lipase	F:AACGCAATGAAACAGGCC R:TGTATGATCCGCTCAACTCG	-0.01[b]	-0.36[b]	1.54[a]	1.53	-1.27	-2.93	-3.73	0.041
INSR	Insulin receptor	F:CGACCATCTGTAAGTCGC R:GTCTTGGAAGTGGTAGTAGG	-0.39	0.40	-0.02	0.81	1.73	-1.29	1.33	0.823

[a-c]Within a row, least squares means lacking a common superscript differ, $P < 0.05$.

[1]Refer to Footnote 1 in Table 2.

[2]Refer to Footnote 2 in Table 2.

[3]Refer to Footnote 3 in Table 2.

[4]No interaction between environment and dietary fat was evident ($P \geq 0.15$).
[5]Delta delta C_t.
[6]Fold difference: positive/negative values indicate increase/decrease mRNA abundance.
[7]Probability value for main effect of environment.
[8]Forward sequence.
[9]Reverse sequence.

Table 10. Effects of dietary fat (CNTR), 3% tallow (TAL), or 3% corn oil (CO) on mRNA abundance in adipose tissue on d 7[1]

			Dietary fat, $\Delta\Delta C_t$[2]				Fdiff[3]			
Gene	Description	Primers, 5`-3`	CNTR	TAL	CO	SEM	CNTR vs. TAL	CO vs. CNTR	CO vs. TAL	*P*-value[4]
ACLY	ATP citrate lyase	F:AGGAGGAGTTCTATGTCTGC[5] R:CAACAGGTGTTTCTT GATGGCC[6]	-0.04	0.78	-0.85	0.63	1.76	1.75	3.10	0.201
ACSS2	Acyl-CoA synthetase short-chain family member 2	F:TGTGAACCTGAAGGAGCTGG R:ACAATGCAGCATCTCACTGG	-0.81	0.52	-0.33	0.72	2.51	-1.39	1.80	0.215
ACACA	Acetyl CoA carboxylase	F:ATGGATGAACCGTCTCCC R:TGTAAGGCCAAGCCATCC	0.15	0.02	-0.66	1.25	-1.09	1.75	1.60	0.566
FASN	Fatty acid synthase	F:CACAACTCCAAAGACACG R:AGGAACTCGGACATAGCG	-0.36[a]	0.20[a]	-1.64[b]	0.81	1.47	2.43	3.58	0.011
SCD	Stearoyl CoA desaturase (delta-9-desaturase)	F:TACTATCTGCTGAGTGCTGTGG R:CTGGAATGCCATCGTGTTGG	0.11[a]	0.90[a]	-2.94[b]	1.18	1.72	8.28	14.32	0.002
FADS2	Fatty acid desaturase 2 (delta-6-desaturase)	F:GCCTTCATCCTTGCTACC R:AGATGGCCGTAATCGTGC	0.83	-0.49	-0.14	1.34	-2.50	1.96	-1.27	0.474
EVOLV6	Fatty acid elongase 6	F:CTGGTTTCTGCTCTGTATGC R:ACCTGAACACTGCAAGGC	1.16	0.45	-0.48	0.82	-1.63	3.11	1.91	0.309
PRKAG1	Protein kinase, AMP-activated, gamma 1 non-catalytic subunit	F:TTGGTGACTAATGGTGTCCG R:TGAAATCAGTGATGGTCAGC	0.69	0.21	-0.22	1.84	-1.39	1.88	1.35	0.444
PLIN1	Perilipin 1	F:GAGTGCTTCCAGAAGACC R:GATGCCCTTCTCGTAAGC	0.51	0.90	-1.46	1.60	1.31	3.92	5.13	0.101
ATGL (PNPLA2)	Adipose triglyceride lipase (Patatin-like phospholipase domain containing 2)	F:ATCATAACCCACTTCGCC R:ACACGGGAATGAAGGTGC	0.04	0.31	-0.92	0.88	1.21	1.95	2.35	0.258
HSL	Hormone sensitive lipase	F:AACGCAATGAAACAGGCC R:TGTATGATCCGCTCAACTCG	0.13	0.36	0.68	1.53	1.17	-1.46	-1.25	0.807
INSR	Insulin receptor	F:CGACCATCTGTAAGTCGC R:GTCTTGGAAGTGGTAGTAGG	0.91	-0.04	-0.88	0.81	-1.93	3.46	1.79	0.313

[a-c]Within a row, least squares means lacking a common superscript differ, $P < 0.05$.

[1]No interaction between environment and dietary fat was evident ($P \geq 0.15$).

[2]Delta delta C_t.

[3]Fold difference: positive/negative values indicate increase/decrease mRNA abundance.

[4]Probability value for main effect of dietary fat.

[5]Forward sequence.
[6]Reverse sequence.

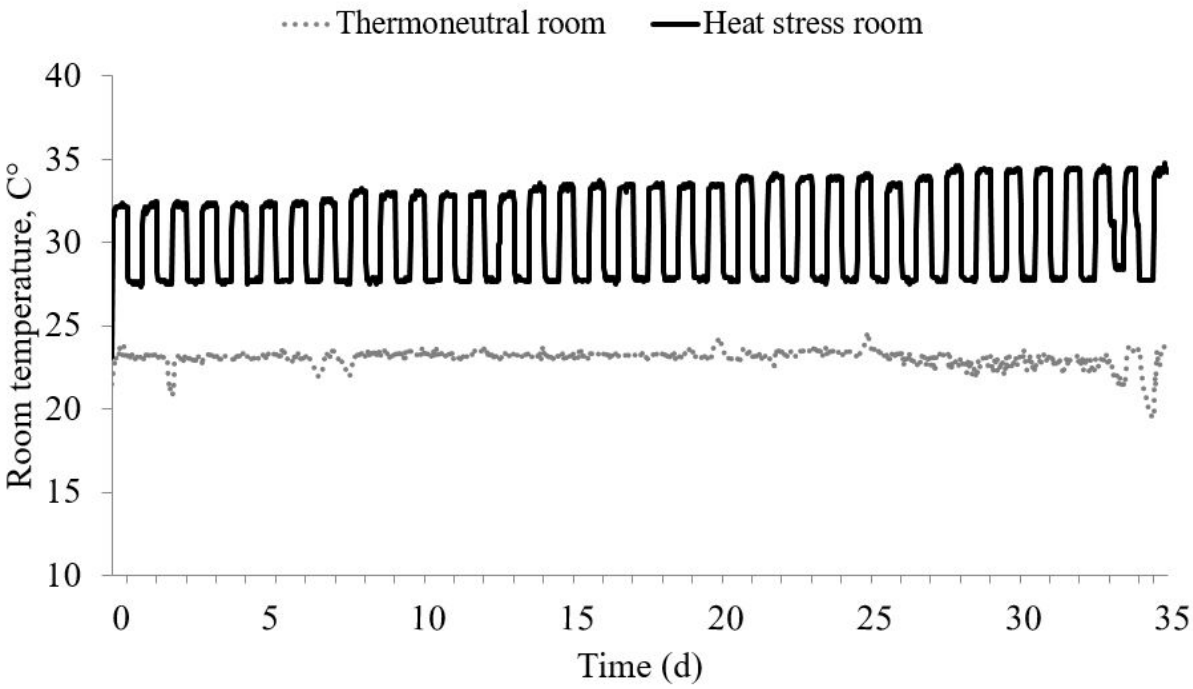
Thermoneutral room
Heat stress room
Room temperature, C°
40
35
30
25
20
15
10
0
5
10
15
20
25
30
35
Time (d)

Figure 1.

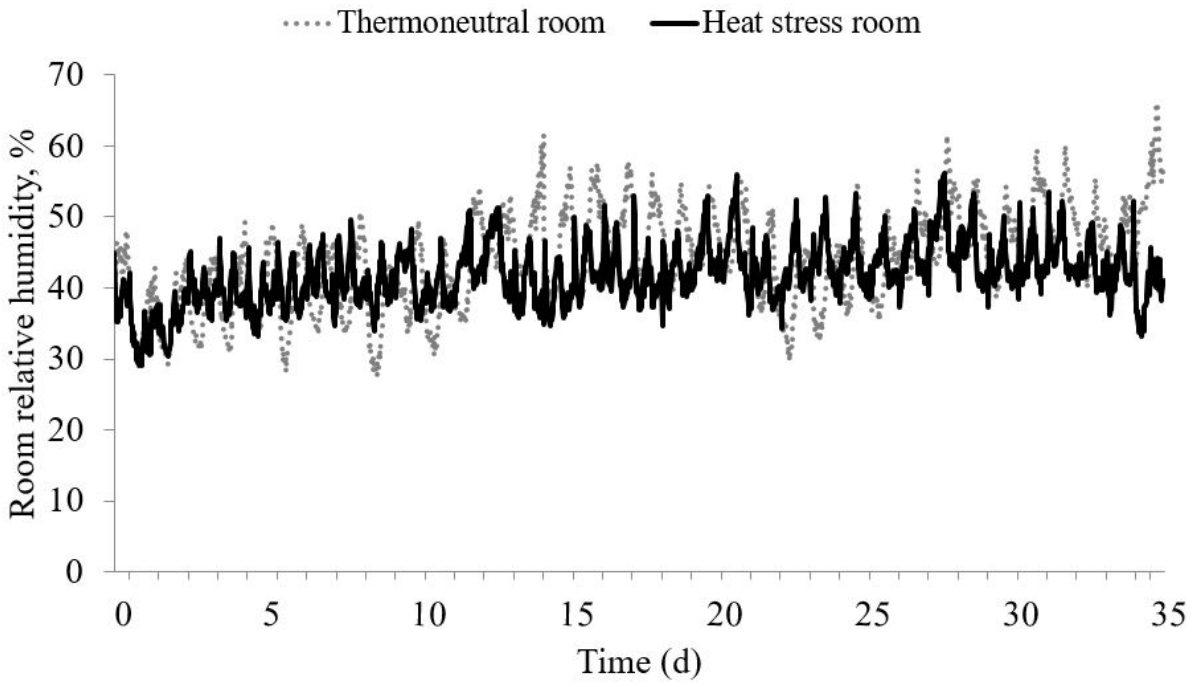
Thermoneutral room
Heat stress room
Room relative humidity, %
70
60
50
40
30
20
10
0
0
5
10
15
20
25
30
35
Time (d)

Figure 2.

Figure 1. Ambient room temperature (°C) by d during the 35 d experiment. Temperature was controlled to achieve a constant 24°C in the thermoneutral room which housed thermoneutral (TN) and pair-fed thermoneutral (PFTN) barrows. The heat stress room which housed the heat stress (HS) barrows was controlled to heat in a diurnal pattern at 28°C from 2000 h to 800 h and at 33°C d 0 to 7, 33.5°C d 7 to 14, 34°C d 14 to 21, 34.5°C d 21 to 28, 35°C d 28 to 35 from 800 h to 2000 h.

Figure 2. Relative humidity (%) of the room by d during the 35 d experiment. Humidity was not governed during the 35 d experiment. Thermoneutral room housed thermoneutral (TN) and pair-fed thermoneutral (PFTN) barrows, and the heat stress room housed heat stress (HS) barrows.