Effects of in utero heat stress on postnatal body composition in pigs: I. Growing phase[1,2]

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ABSTRACT: Environmentally induced heat stress (HS) negatively influences production variables in agriculturally important species. However, the extent to which HS experienced in utero affects nutrient partitioning during the rapid lean tissue accretion phase of postnatal growth is unknown. Study objectives were to compare future whole-body tissue accretion rates in pigs exposed to differing in utero and postnatal thermal environments when lean tissue deposition is likely maximized. Pregnant sows were exposed to thermoneutral (TN; cyclical 15°C nighttime and 22°C daytime; $n = 9$) or HS (cyclical 27°C nighttime and 37°C daytime; $n = 12$) conditions during their entire gestation. Twenty-four offspring from in utero TN (IUTN; $n = 6$ gilts and 6 barrows; 30.8 ± 0.2 kg BW) and in utero HS (IUHS; $n = 6$ gilts and 6 barrows; 30.3 ± 0.2 kg BW) were euthanized as an initial slaughter group (ISG). Following the ISG, 48 pigs from IUTN ($n = 12$ gilts and 12 barrows; 34.1 ± 0.5 kg BW) and IUHS ($n = 12$ gilts and 12 barrows; 33.3 ± 0.3 kg BW) were exposed to constant HS (34.1 ± 2.4°C) or TN (21.5 ± 2.0°C) conditions until they reached 61.5 ± 0.8 kg BW, at which point they were sacrificed and their whole-body composition was determined. Homogenized carcasses were analyzed for N, crude fat, ash, water, and GE content. Data were analyzed using PROC MIXED in SAS 9.3. Rectal temperature and respiration rate increased ($P < 0.01$) during postnatal HS compared to TN (39.4 vs. 39.0°C and 94 vs. 49 breaths per minute, respectively). Regardless of in utero environment, postnatal HS reduced ($P < 0.01$) feed intake (2.06 vs. 2.37 kg/d) and ADG (0.86 vs. 0.98 kg/d) compared to TN conditions. Postnatal HS did not alter water, protein, and ash accretion rates but reduced lipid accretion rates (198 vs. 232 g/d; $P < 0.04$) compared to TN-reared pigs. In utero environment had no effect on future tissue deposition rates; however, IUHS pigs from the ISG had reduced liver weight ($P < 0.04$; 17.9%) compared to IUTN controls. In summary, postnatal HS reduced adipose tissue accretion rates, but IUHS did not appear to impact either lean or adipose tissue accretion during this specific growth phase.

Key words: imprinting, in utero heat stress, pigs, tissue accretion

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INTRODUCTION

Heat stress (**HS**) reduces growth, alters carcass quality, and compromises efficiency, thus diminishing efforts by animal agriculture to produce high-quality protein for human consumption (Baumgard et al., 2012; Baumgard and Rhoads, 2013). The negative effects of HS will likely become more pronounced as climate models predict an increase in extreme summer temperatures for most agricultural areas (Luber and McGeehin, 2008). Since basal heat production has markedly increased with genetic selection for

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enhanced lean tissue accretion (Brown-Brandl et al., 2004), some suggest faster growing animals are more sensitive to HS (Nienaber and Hahn, 2007). As a consequence, there is a need to understand the mechanisms by which HS reduces animal performance.

While the detrimental effects of postnatal HS on tissue accretion have been well documented (Kouba et al., 2001; Collin et al., 2001a; Kerr et al., 2003), the extent to which in utero hyperthermia affects future animal performance is relatively unknown. If climate change lengthens summer duration and increases the frequency/intensity of heat waves, the number of animals gestating during the stressful warm times of the year will increase. Previous studies indicate prenatal stressors (nonthermal) can permanently alter growth (Foxcroft et al., 2006, 2009), postabsorptive metabolism (Chen et al., 2010; Pinney and Simmons, 2010), and body composition (Ravelli et al., 1976; Barker et al., 1993; Roseboom et al., 2006); however, the effects of maternal HS on postnatal growth variables in pigs are unknown. Therefore, our study objectives were to determine the subsequent rate and quantity of whole-body tissue accretion in pigs exposed to differing in utero and postnatal thermal environments. We hypothesized that in utero HS exposure would reduce future skeletal muscle accretion during the growing phase (30 to 60 kg BW), a period when lean tissue deposition dominates growth (Wagner et al., 1999; Van Milgen and Noblet, 2003).

MATERIALS AND METHODS

In Utero Environments

The University of Missouri Animal Care and Use Committee approved all procedures involving pregnant sows. Twenty-one first-parity crossbred sows (Large White × Landrace; GPK1 × GPK4; Choice Genetics USA, Des Moines, IA) were exposed to thermoneutral (**TN**; cyclical 15°C nighttime and 22°C daytime and 55% relative humidity [**RH**; %]; $n = 9$), or HS (cyclical 27°C nighttime and 37°C daytime and 67.5% RH; $n = 12$) conditions in the Brody environmental chambers at the University of Missouri (Columbia, MO; Lucy et al., 2012). Thermal treatments began 6 d after insemination (Duroc; Swine Genetics International, Cambridge, IA) and continued until farrowing (116.6 ± 0.5 d gestation; Lucy et al., 2012). Heat stress caused a sustained increase in rectal temperature ($\mathbf{T_{re}}$; $P < 0.05$; 0.3°C) in pregnant sows compared to TN controls (Lucy et al., 2012). Feed intake (**FI**) did not differ between treatments ($P > 0.15$; 2.4 ± 0.1 kg/d for TN and 2.3 ± 0.1 kg/d for HS) as both the HS and TN control pregnant sows were limit fed to prevent excessive maternal weight gain (standard industry practice; Brendemuhl and Myer, 2009). Gestation length was reduced ($P < 0.05$) in HS (115.7 ± 0.5 d) compared to TN (117.4 ± 0.5 d) sows (Lucy et al., 2012). Although piglets born alive were similar ($P > 0.05$; 11.5 ± 0.8), piglet birth weight was reduced ($P < 0.01$) in HS (1.18 ± 0.05 kg) compared to TN sows (1.41 ± 0.05 kg) (Lucy et al., 2012), but piglet BW at weaning did not differ between treatments ($P > 0.10$; 5.04 ± 0.21 kg; Lucy et al., 2012). Between parturition and weaning, all piglets were exposed to the same environmental conditions (26–32°C) as recommended by the Federation of Animal Science Societies (2010). After weaning, all offspring ($n = 253$) were transported to Iowa State University (Ames, IA).

Postnatal Environments

The Iowa State University Institutional Animal Care and Use Committee approved all procedures involving animals. Between weaning and approximately 30 kg BW, all pigs were housed in TN conditions as recommended by the Federation of Animal Science Societies (2010) and allowed to consume water and feed (based primarily on corn and soybean meal) ad libitum. From the 253 pigs transported to Iowa State University, 24 pigs (72.5 ± 1.7 d of age) with no previous postnatal HS exposure from in utero TN (**IUTN**; $n = 6$ gilts and 6 barrows; 30.8 ± 0.2 kg BW) and in utero HS (**IUHS**; $n = 6$ gilts and 6 barrows; 30.3 ± 0.2 kg BW) were selected based on similar BW, balanced by gender, and euthanized as part of an initial slaughter group (**ISG**). Following the ISG, 48 pigs from IUTN ($n = 12$ gilts and 12 barrows; 34.1 ± 0.5 kg initial BW) and IUHS ($n = 12$ gilts and 12 barrows; 33.3 ± 0.3 kg initial BW) were selected based on similar BW, balanced by gender, and then housed in individual pens (0.61 by 2.44 m) in 1 of 2 environmentally controlled rooms. Pigs were evenly distributed within rooms based on treatment and gender. Within each room, ambient temperature ($\mathbf{T_a}$) and RH were continuously recorded by 2 mounted data loggers (EL–WIN–USB; accuracy: ±1.0°C; Lascar Electronics Ltd., Wiltshire, UK) every 30 min. Fans were used to prevent uneven temperature dispersion, and data loggers were positioned at opposite ends of the rooms to confirm uniform environmental conditions. Although Ta was controlled, percent RH was ungoverned throughout the experiment.

Twelve barrows ($n = 6$ IUTN and 6 IUHS) and 12 gilts ($n = 6$ IUTN and 6 IUHS) were housed in constant TN conditions (21.5 ± 2.0°C and 68.1 ± 8.3 % RH), and 12 barrows ($n = 6$ IUTN and 6 IUHS) and 12 gilts ($n = 6$ IUTN and 6 IUHS) were maintained in constant HS conditions (34.1 ± 2.4°C and 48.9 ± 6.7 % RH) until they reached approximately 60 kg. All pigs were given ad libitum access to a standard commercial diet formulated to meet or exceed nutritional requirements (Table

1; NRC, 1998). Feed intake was determined weekly and BW was determined every two weeks. Respiration rate (**RR**; breaths per minute; **bpr**) and T_{re} were obtained twice daily (0800 and 1600 h). Respiration rate was determined by counting flank movement for 15 s and multiplying by 4. Rectal temperature was determined with a calibrated and lubricated thermistor thermometer (Welch Allyn SureTemp Plus; accuracy: ±0.1°C; Welch Allyn, Skaneateles Falls, NY) inserted approximately 10 cm into the rectum of unrestrained pigs.

Blood Sampling and Analysis

Blood (10 mL) was obtained from all pigs via jugular venipuncture (BD vacutainers; Becton Dickinson, Franklin Lakes, NJ; K_3EDTA; lithium heparin; serum) 1 d before sacrifice at 1500 h (in a fed state while still experiencing their respective environmental treatment) and stored on ice until processing; plasma and serum were then harvested by centrifugation at 2,500 × *g* for 15 min at 4ºC, aliquoted, and stored at –80°C. Glucose concentration was immediately determined from whole blood collected in lithium heparin tubes using a Vet Scan iStat C68+ cartridge (Abaxis, Inc., Union City, CA). Plasma insulin concentration was measured using an ELISA kit (Mercodia Porcine Insulin ELISA; Mercodia AB, Uppsala, Sweden), following the manufacturer's instructions. Commercially available kits were used to determine plasma NEFA (Autokit NEFA; Wako Chemicals USA, Richmond, VA), plasma urea N (**PUN**; Urea Nitrogen Reagent; TECO Diagnostics, Anaheim, CA), and serum creatine kinase (**CK**) concentrations (Creatine Kinase-SL Assay; SEKISUI Diagnostics, Charlottetown, PE, Canada). The intra- and interassay coefficients of variation were 3.8, 7.2, 6.4, and 1.9% and 8.0, 4.7, 8.3, and 4.5% for insulin, NEFA, CK, and PUN, respectively. Quantification of insulin resistance was determined by the homeostatic model assessment of insulin resistance ([glucose (mmol/L) × insulin (mg/L)]/450; Haffner et al., 1996), and the insulin to FI ratio ([insulin]/ADFI throughout the entire experiment) was calculated for individual pigs.

Serial Slaughter and Subsample Analysis

All pigs used in the postnatal experiment were harvested after an overnight fast in TN conditions at 30.6 ± 0.2 kg BW (ISG) and 61.5 ± 0.8 kg BW (final slaughter group [**FSG**]). Initial and final BW were selected in an attempt to capture a period of rapid lean accretion in commercially relevant growing pigs (Wagner et al., 1999; Van Milgen and Noblet, 2003). The ISG was euthanized with an intravenous barbiturate overdose (100 mg/kg BW; Nembutol; Ovation Pharmaceuticals Inc., Deerfield, IL). Since a similar BW at sacrifice was an objective, FSG pigs were harvested at different times (32.5 ± 4.5 d on feed).

Table 1. Ingredients and chemical composition of diet for growing pigs (as-fed basis)

Ingredient	Percent
Corn	62.24
Soybean meal	20.04
Distillers dried grains with solubles	15.00
45–30 vitamin and mineral premix[1]	2.15
Monocalcium phosphate	0.14
L-Lys HCl	0.35
L-Thr	0.04
DL-Met	0.04
Calculated chemical composition,[2] %	
DM	90.11
CP	18.9
Crude fat	4.31
NDF	12.95
ADF	5.27
Calcium	0.62
Phosphorus	0.50
SID[3] Lys	1.14
ME, Mcal/kg	3.31
NE, Mcal/kg	2.44

[1]Supplied per kilogram of diet: 9,464 IU vitamin A, 1,735 IU vitamin D_3, 50 IU vitamin E, 2.5 IU vitamin K, 5.8 mg choline, 4.8 mg riboflavin, 24 mg niacin, 18.9 mg pantothenic acid, 30 μg vitamin B_{12}, 1.7 μg biotin, 0.00047 mg folic acid, 4.84 g Ca, 430 mg P, 158 mg Zn, 61 mg Mn, 177 mg Fe, 21 mg Cu, and 0.28 mg Se.

[2]Values were estimated according to the NRC (1998).

[3]SID = standardized ileal digestible.

At sacrifice, FSG pigs were electrically stunned and exsanguinated and blood was collected. For ISG and FSG, stomach, intestinal, gallbladder, and bladder contents were removed and liver, spleen, and total viscera weight (minus contents) was recorded. Whole carcass, head, viscera (minus contents of stomach, intestine, gallbladder, and bladder), and blood were weighed to determine empty BW (**EBW**). Carcass, head, viscera, and blood of individual pigs were frozen, sectioned, passed twice through a mechanical grinder (Buffalo number 66BX Enterprise; Buffalo No. 66BX Enterprise, St. Louis, MO), and then passed 4 times through a Hobart 52 grinder with a 5-mm die (model number 4046; Hobart Corporation, Troy, OH) for homogenization. Ground pigs were subsampled, and subsamples were immediately dried in a convection oven (101°C for 24 h) to a constant weight for determination of DM according to method 950.46 (AOAC, 2002). Dried subsamples were ground through a 1-mm screen (Retsch ZM 100; Glen Mills Inc., Clifton, NJ) and analyzed for ash, crude fat, N, and GE. Ash was determined by drying at 600°C (for 24 h) to a constant weight according to method 923.03 (AOAC, 2002). Crude fat was

determined by Soxhlet extraction according to method 991.36 (AOAC, 2002) using *n*-hexane as the solvent (Fischer Scientific, Fair Lawn, NJ). Nitrogen content was determined by combustion using a LECO TruMac N Nitrogen Determinator (model number 630-300-300; Leco Corporation, St. Joseph, MI) according to method 992.15 (AOAC, 2002). Calibration of the LECO was conducted with an EDTA standard (known N content 9.56 ± 0.03%; N content determined to be 9.58 ± 0.06%), and CP was expressed as N × 6.25. Gross energy was determined using isoperibol bomb calorimetry (Parr 620 calorimeter, model number 6200; Parr Instrument Company, Moline, IL), and benzoic acid was used as the calibration standard (known GE 6,318 ± 18 kcal/kg; GE was determined to be 6,319 ± 8 kcal/kg). All chemical analyses were performed in duplicate and repeated when the intraduplicate CV exceeded 3%.

Based on the subsample chemical content, total body composition was determined for water, ash, lipid, and protein using the EBW (Noblet et al., 1987). The average chemical composition of IUTN and IUHS pigs in the ISG was used to estimate the initial body composition of IUTN and IUHS pigs in the FSG, respectively. Within each individual pig, the accretion of water, lipid, protein, and ash were estimated by [final content (g of tissue) – estimated initial content (g of tissue)]/days between harvest dates.

To determine carcass gain efficiency (**CE**), the average carcass weight of IUTN and IUHS pigs in the ISG was used to estimate the initial carcass weight of IUTN and IUHS pigs in the FSG, respectively. Within each individual pig, the accretion of carcass tissue was estimated by (final carcass weight – estimated initial carcass weight)/days between harvest dates. Carcass gain efficiency was calculated by dividing carcass gain by ADFI in individual pigs (carcass gain:FI).

Statistical Analysis

All data were analyzed using the PROC MIXED procedure in SAS 9.3 (SAS Inst. Inc., Cary, NC). Statistical model components included in utero environment (IUTN or IUHS), postnatal environment (TN or HS), sex (barrow or gilt), and all interactions. Since no significant sex differences were detected, it was removed from the final analysis. All interactions, regardless of significance level, were included in the model and dam was used as a random effect for all analyses. Average daily gain of individual pigs calculated 2 wk before the start of the postnatal treatment was used as a covariate for analysis of all body composition and growth variables. For repeated analysis of T_{re} and RR, each pig's respective parameter was analyzed using repeated measures with an autoregressive covariance structure with day as the repeated effect. Statistical significance was defined as $P \leq 0.05$, and a tendency was defined as $0.05 < P \leq 0.10$.

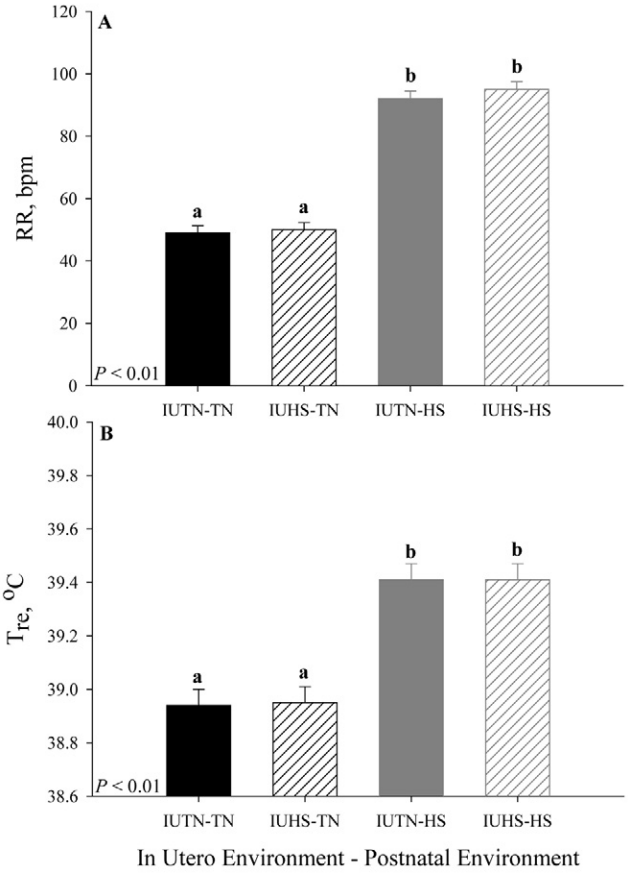

Figure 1. Effects of in utero and postnatal thermal environments on (A) respiration rate (RR; in breaths per minute [bpm]) and (B) rectal temperature (T_{re}) by in utero and postnatal environment in growing pigs. IUTN-TN = in utero thermoneutral pigs in postnatal thermoneutral conditions; IUHS-TN = in utero heat-stressed pigs in postnatal thermoneutral conditions; IUTN-HS = in utero thermoneutral pigs in postnatal heat stress conditions; IUHS-HS = in utero heat-stressed pigs in postnatal heat stress conditions. [a,b]Letters above bars indicate significance ($P < 0.01$).

RESULTS

Thermal Indices

An overall increase ($P < 0.01$) in RR and T_{re} was detected in postnatal HS pigs compared to TN controls (94 ± 2 vs. 49 ± 2 breaths per minute [Fig. 1A] and 39.4 ± 0.1 vs. 39.0 ± 0.1°C [Fig. 1B], respectively). Neither in utero nor in utero × postnatal treatment differences were detected for RR or Tre.

Growth Performance

Although not affected by in utero environments ($P > 0.50$; 2.29 kg/d), postnatal HS reduced ($P < 0.01$) FI by 13% compared to TN conditions (Table 2).

Table 2. Effect of in utero and postnatal environment on growth parameters in growing pigs

	Environment[1]					*P*-value[2]		
Parameter	IUTN-TN	IUHS-TN	IUTN-HS	IUHS-HS	SEM	IE	PE	IE × PE
Initial slaughter group (30 kg)								
Final live BW, kg	30.8	30.3	–	–	0.2	0.15	–	–
EBW,[3] kg	26.2	26.2	–	–	0.8	0.98	–	–
Final slaughter group (60 kg)								
Final live BW, kg	62.1	61.9	62.3	59.5	0.8	0.06	0.23	0.07
EBW, kg	58.8	58.1	58.9	55.8	0.9	0.05	0.32	0.17
ADG, kg	0.99	0.97	0.88	0.84	0.03	0.34	0.01	0.52
Feed intake, kg	2.42	2.31	2.06	2.06	0.08	0.51	0.01	0.47
G:F, kg/kg	0.41	0.42	0.43	0.41	0.01	0.51	0.84	0.12
Carcass gain:feed,[4] kg/kg	0.31	0.33	0.37	0.35	0.02	0.83	0.01	0.16
Gain:ME intake,[5] kg/Mcal	0.12	0.12	0.12	0.12	<0.01	0.49	0.75	0.11

[1]IUTN-TN = in utero thermoneutral pigs in postnatal thermoneutral conditions; IUHS-TN = in utero heat-stressed pigs in postnatal thermoneutral conditions; IUTN-HS = in utero thermoneutral pigs in postnatal heat stress conditions; IUHS-HS = in utero heat-stressed pigs in postnatal heat stress conditions.

[2]IE = in utero environment; PE = postnatal environment.

[3]EBW = empty BW (final live BW minus gastrointestinal contents).

[4]Carcass weight gain per kilogram of feed intake.

[5]Body weight gain per megacalorie of ME intake.

Average daily gain was reduced ($P < 0.01$; 0.12 kg/d) in pigs in postnatal HS compared to TN conditions (Table 2). No postnatal feed efficiency (**FE**) differences were detected (0.42 kg gain/kg feed; Table 2), and neither ADG nor FE were influenced by in utero thermal treatment (Table 2). Regardless of in utero treatment, CE increased ($P < 0.01$; 12.5%) in postnatal HS compared to TN-exposed pigs; however, no in utero or in utero × postnatal treatment differences in CE ($P > 0.15$) were observed (Table 2). No in utero, postnatal, or in utero × postnatal treatment differences ($P > 0.10$) were detected in BW gain per Mcal of ME consumed (0.12 kg/Mcal; Table 2).

Organ and Carcass Weights

In the ISG, absolute liver weight and liver weight as a percent of EBW were reduced ($P < 0.04$; 17.9 and 13.6%, respectively) and total viscera weight tended ($P < 0.10$) to be reduced (6.5%) in IUHS compared to IUTN pigs (Table 3). In the FSG, head weight was reduced ($P \leq 0.05$; 4.9%) in IUHS compared to IUTN pigs (Table 3). A decrease ($P < 0.01$) in total viscera (450 g), liver (80 g), spleen (20 g), and blood (190 g) weight was observed in pigs in postnatal HS compared to TN environments (Table 3). Total viscera, liver, and spleen weight as a percent of EBW were decreased ($P < 0.02$) 5.9, 7.6, and 15.8%, respectively, in postnatal HS compared to TN control pigs (Table 3). No other in utero or postnatal organ and carcass weight differences were detected (Table 3).

Tissue Composition and Accretion

In utero HS pigs in postnatal HS conditions had reduced ($P < 0.03$; 56 g/d) whole-body water accretion compared to IUTN pigs in postnatal HS conditions (**IUTN-HS**; Table 4). Regardless of in utero environment, postnatal HS pigs had reduced whole-body adipose tissue accretion ($P < 0.04$; 14.7%) compared to TN controls (Table 4; Fig. 2). No other in utero or postnatal tissue composition or accretion differences were observed (Table 4).

Blood Analyses

In utero HS pigs in postnatal HS conditions tended ($P < 0.10$) to have reduced glucose concentrations (7.25 mg/dL) compared to IUTN-HS pigs (Table 5). Regardless of in utero treatment, PUN was reduced ($P < 0.04$; 17%) and [insulin]/FI was increased ($P \leq 0.05$; 30%) for pigs in postnatal HS compared to TN conditions (Table 5). No other in utero or postnatal treatment differences in blood variables were detected (Table 5).

DISCUSSION

Despite cooling system advances and improved management practices, HS continues to compromise efficient high-quality animal protein production. Although HS is primarily an animal welfare and economic issue in developed countries, it is a food security and humanitarian concern in regions that lack the resources to afford heat abatement technology (Battisti and Naylor, 2009). While the negative effects of HS on postnatal performance have been well documented

Table 3. Effect of in utero and postnatal environment on carcass and organ weights of growing pigs

	Environment[1]					*P*-value[2]		
Parameter	IUTN-TN	IUHS-TN	IUTN-HS	IUHS-HS	SEM	IE	PE	IE × PE
Initial slaughter group (30 kg)								
Weight								
Carcass, kg	22.3	21.8	–	–	0.7	0.61	–	–
Total viscera, kg	4.15	3.88	–	–	0.11	0.09	–	–
Liver, kg	0.95	0.78	–	–	<0.01	0.03	–	–
Spleen, kg	0.07	0.08	–	–	<0.01	0.28	–	–
Percent of EBW[3]								
Carcass, % of EBW	84.3	84.8	–	–	0.4	0.35	–	–
Total viscera, % of EBW	15.7	15.2	–	–	0.4	0.34	–	–
Liver, % of EBW	3.59	3.10	–	–	0.15	0.03	–	–
Spleen, % of EBW	0.30	0.29	–	–	0.02	0.57	–	–
Final slaughter group (60 kg)								
Weight								
Carcass, kg	45.9	45.3	46.4	43.8	0.9	0.07	0.61	0.21
Head, kg	4.07	3.94	4.09	3.92	0.07	0.05	0.89	0.95
Total viscera, kg	5.85	5.90	5.53	5.33	0.11	0.48	0.01	0.22
Liver, kg	1.00	0.99	0.94	0.89	0.02	0.14	0.01	0.17
Spleen, kg	0.11	0.11	0.09	0.09	0.01	0.62	0.01	0.62
Blood, kg	2.97	2.93	2.85	2.67	0.06	0.14	0.01	0.20
Percent of EBW								
Carcass, % of EBW	78.0	78.0	78.8	78.6	0.3	0.72	0.06	0.78
Head, % of EBW	7.07	6.75	6.96	6.93	0.01	0.29	0.74	0.14
Total viscera, % of EBW	10.0	10.2	9.4	9.6	0.2	0.29	0.01	0.92
Liver, % of EBW	1.71	1.73	1.60	1.59	0.01	0.96	0.02	0.74
Spleen, % of EBW	0.18	0.19	0.16	0.16	0.01	0.78	0.01	0.94
Blood, % of EBW	5.1	5.1	4.8	4.8	0.1	0.93	0.07	0.91

[1]IUTN-TN = in utero thermoneutral pigs in postnatal thermoneutral conditions; IUHS-TN = in utero heat-stressed pigs in postnatal thermoneutral conditions; IUTN-HS = in utero thermoneutral pigs in postnatal heat stress conditions; IUHS-HS = in utero heat-stressed pigs in postnatal heat stress conditions.

[2]IE = in utero environment; PE = postnatal environment.

[3]EBW = empty BW (final live BW minus gastrointestinal contents).

(Renaudeau et al., 2008, 2012; Baumgard and Rhoads, 2013), the impact of IUHS on future progeny performance is ill defined. The uncertainty of environmental influence on future productivity will become more important if climate change intensifies the severity and frequency of heat waves and extends summer length.

Nonthermal prenatal stressors can permanently reduce growth (Foxcroft et al., 2006, 2009), alter postabsorptive metabolism (Chen et al., 2010; Pinney and Simmons, 2010), and influence body composition (Ravelli et al., 1976; Barker et al., 1993; Roseboom et al., 2006). The offspring's altered body composition and metabolism is generally characterized by increased adiposity and a metabolite/endocrine profile that resembles Type II diabetes. However, in the present study, in utero hyperthermia had no measurable impact on key body composition variables during the growing phase of postnatal growth, when protein accretion is ostensibly maximized (Wagner et al., 1999). Reasons for the lack of in utero treatment effects on postnatal lean and adipose tissue accretion are not clear but may be the result of similar maternal FI (i.e., differences in dam nutrient intake may partly explain the aforementioned nonthermal in utero effects) or insufficient maternal hyperthermia (i.e., the heat load applied to the pregnant dams was inadequate to alter fetal programming) or the impact of IUHS may not be expressed until later in life when the rate of skeletal muscle accretion has slowed and adipose accretion is increased.

As reviewed by Graham et al. (1998), in utero hyperthermia caused microcephaly (reduced cranial size) in the present study, independent of postnatal treatment (Table 3). Additionally, liver weight and liver weight as a percent of EBW were reduced in IUHS pigs compared to IUTN pigs in the ISG, which may impact maintenance costs because reduced organ size (particularly liver and intestinal tissue) is correlated with decreased fasting heat production (Koong et al., 1982). Regardless of in utero environment and similar to reports by others (Lefaucheur et al., 1989; Rinaldo and Le Dividich, 1991), pigs maintained in postnatal HS conditions had reduced total viscera mass and liver weight compared to those in postnatal

Table 4. Effect of in utero and postnatal environment on whole-body composition and tissue accretion in growing pigs

	Environment[1]					*P*-value[2]		
Parameter	IUTN-TN	IUHS-TN	IUTN-HS	IUHS-HS	SEM	IE	PE	IE × PE
Initial slaughter group (30 kg)								
Percent of EBW[3]								
Water, % of EBW	71.2	70.9	–	–	3.6	0.53	–	–
Protein, % of EBW	16.7	16.8	–	–	<0.1	0.77	–	–
Lipid, % of EBW	9.6	10.0	–	–	0.4	0.52	–	–
Ash, % of EBW	2.5	2.4	–	–	<0.1	0.79	–	–
GE, kcal/g	6,230	6,221	–	–	36	0.86	–	–
Final slaughter group (60 kg)								
Percent of EBW								
Water, % of EBW	63.1	63.7	65.2	63.8	0.6	0.56	0.13	0.10
Protein, % of EBW	16.9	17.2	17.2	17.3	<0.1	0.36	0.37	0.62
Lipid, % of EBW	17.3	16.8	15.2	16.4	<0.1	0.67	0.14	0.20
Ash, % of EBW	2.5	2.6	2.4	2.7	0.2	0.44	0.98	0.61
Tissue accretion								
Water,[4] g/d	568[a]	592[a]	594[a]	538[b]	18	0.39	0.52	0.03
Protein,[5] g/d	171	177	175	168	7	0.97	0.74	0.23
Lipid,[6] g/d	240	224	192	204	14	0.88	0.04	0.28
Ash,[7] g/d	25	26	23	26	3	0.44	0.77	0.79
Lipid:protein	1.41	1.29	1.13	1.24	0.09	0.99	0.12	0.18
GE, kcal/g	6,796	6,782	6,740	6,720	76	0.85	0.44	0.96

[a–c]Means within a row with different superscripts differ ($P < 0.05$).

[1]IUTN-TN = in utero thermoneutral pigs in postnatal thermoneutral conditions; IUHS-TN = in utero heat-stressed pigs in postnatal thermoneutral conditions; IUTN-HS = in utero thermoneutral pigs in postnatal heat stress conditions; IUHS-HS = in utero heat-stressed pigs in postnatal heat stress conditions.

[2]IE = in utero environment; PE = postnatal environment.

[3]EBW = empty BW (final live BW minus gastrointestinal contents).

[4]Water accretion per day.

[5]Protein accretion per day.

[6]Lipid accretion per day.

[7]Ash accretion per day.

TN conditions. Since the viscera (especially the liver) has a high metabolic activity (Burrin et al., 1988; Van Milgen and Noblet, 2003), decreased splanchnic bed mass likely represents a strategy to minimize basal heat production in response to hyperthermia, possibly due to reduced visceral blood flow as described by others (Lambert et al., 2002; Leon and Helwig, 2010).

Although whole-body lipid accretion was reduced (15%), protein deposition was similar (173 g/d) in postnatal HS pigs compared to TN controls (Fig. 2). Reduced adiposity corroborates results observed by Le Bellego et al. (2002), where both carcass fat content and back fat thickness were decreased in HS pigs compared to ad libitum–fed TN controls. The observed reduction in adipose content was not unexpected, because postnatal HS in the present study negatively impacted FI and ADG (Table 2) as previously described in heat-stressed livestock species (Renaudeau et al., 2008; Baumgard et al., 2012; Johnson et al., 2013a; Pearce et al., 2013). Reduced FI limits energy available for growth, thus decreasing NE available for lipid deposition, but it is unclear why protein accretion was not similarly affected. A likely explanation is that although FI was reduced, it was still adequate to allow for maximum lean tissue deposition during this growth phase (Van Milgen and Noblet, 2003).

No in utero thermal treatment differences were detected in circulating glucose, insulin, NEFA, PUN, and CK (Table 5). The lack of differences in glucose and NEFA agrees with our previous IUHS reports (Boddicker et al., 2014) and were not unexpected, as we did not detect gross differences in body composition. However, we previously reported that IUHS increases future circulating insulin concentration, regardless of postnatal thermal environments (Boddicker et al., 2014). Reasons why the current experiment did not corroborate our previous results are not clear but may be due to infrequent blood sampling in the current study.

Circulating insulin, glucose, NEFA, and CK concentrations were similar in pigs in both postnatal environments. These data are somewhat surprising as insulin is frequently reported to increase while glucose and NEFA concentrations are decreased during HS, especially compared to pair-fed TN controls (Baumgard and

Table 5. Effect of in utero and postnatal environment on blood parameters in growing pigs

	Environment[1]					*P*-value[2]		
Parameter	IUTN-TN	IUHS-TN	IUTN-HS	IUHS-HS	SEM	IE	PE	IE × PE
Glucose, mg/dL	100.7	104.3	101.6	94.3	3.1	0.16	0.57	0.09
Insulin, ng/mL	0.11	0.14	0.16	0.14	0.02	0.56	0.22	0.13
Insulin:glucose	0.10	0.15	0.15	0.15	0.01	0.28	0.21	0.23
[Insulin]:feed intake,[3] kg	4	6	7	6	1	0.56	0.05	0.12
HOMA-IR,[4] AU	1.91	1.95	1.90	1.87	0.32	0.99	0.86	0.90
NEFA, mEq/L	80.7	82.0	81.2	86.8	11.4	0.77	0.81	0.85
Creatine kinase, units/L	198	194	196	225	24	0.61	0.54	0.48
PUN,[5] mg/dL	13.9	14.9	11.2	12.7	1.1	0.30	0.03	0.83

[1]IUTN-TN = in utero thermoneutral pigs in postnatal thermoneutral conditions; IUHS-TN = in utero heat-stressed pigs in postnatal thermoneutral conditions; IUTN-HS = in utero thermoneutral pigs in postnatal heat stress conditions; IUHS-HS = in utero heat-stressed pigs in postnatal heat stress conditions.

[2]IE = in utero environment; PE = postnatal environment.

[3]Insulin concentration per kilogram of feed intake.

[4]HOMA-IR = homeostatic model assessment of insulin resistance (quantification of insulin resistance). AU = Arbitrary Units.

[5]PUN = plasma urea N.

Rhoads, 2013; Pearce et al., 2013). Additionally, HS pigs consumed 13% less feed compared to TN controls, and decreased FI normally reduces glucose and insulin levels and increases circulating NEFA concentrations, because insulin secretion (a potent lipogenic and antilipolytic signal) is sensitive to changes in nutrient intake (Vernon, 1992). To better understand how HS influences the insulin to FI relationship, we calculated the insulin to FI ratio and determined that HS-exposed pigs had increased circulating insulin per unit of FI of 30% compared to TN controls. These data agree with reports demonstrating that HS increases insulin secretion compared to TN environments (Baumgard and Rhoads, 2013; Pearce et al., 2013). Contrary to previous reports indicating that acute HS increases PUN in various species (as reviewed by Baumgard and Rhoads, 2013), chronic postnatal HS in the present study reduced PUN by 17% (Table 5). Discrepancies between data sets may be due to the length and severity of HS, because chronic HS reduces protein turnover and PUN relative to control treatments (Temim et al., 2000) and blood samples in the present study were obtained approximately 5 wk after HS exposure began.

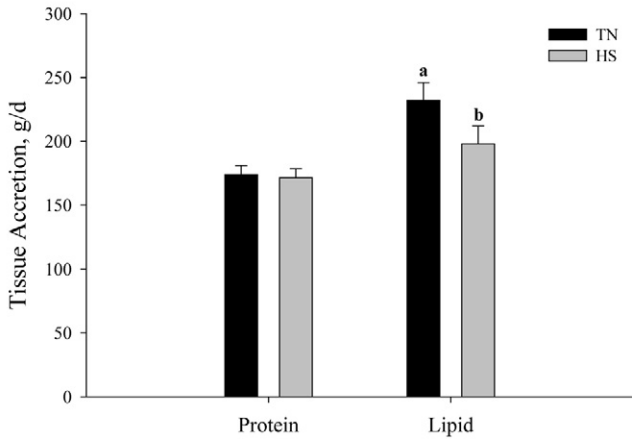

Figure 2. Effect of postnatal thermal environments on protein accretion (g/d) and lipid accretion (g/d) in growing pigs inpostnatal thermoneutral (TN) conditions and postnatal heat stress (HS) conditions. [a,b]Letters above bars indicate significance ($P < 0.05$).

In general, early reports (Kleiber, 1961; NRC, 1981; Curtis, 1983) indicate that an animal's maintenance costs increase when T_a exceeds the upper critical temperature. Increased HS-induced maintenance costs has specifically been reported in multiple species including cattle (McDowell et al., 1969; Beede and Collier, 1986), lambs (Ames and Brink, 1977), rodents (Collins et al., 1980), and pigs (Campos et al., 2014), and this increase is primarily attributed to greater energy costs associated with employing heat mitigating processes (i.e., panting and sweating) and enhanced chemical reaction rates as predicted by the Van't Hoff Arrhenius equation (Kleiber, 1961). However, some reports in heat-stressed pigs (Collin et al., 2001b; Renaudeau et al., 2013) describe reduced total heat and fasting heat production, and this is likely due to reductions in visceral mass caused by both the hyperthermia (Rinaldo and Le Dividich, 1991) and reduced FI (Koong et al., 1982). To gain a better appreciation for how HS alters bioenergetics, ME for maintenance ($\mathbf{ME_{maintenance}}$) was estimated with the following equation (Patience, 2012): $ME_{maintenance} = ME_{intake} - (ME_{protein} + ME_{lipid})$, in which $\mathbf{ME_{intake}}$ = ME in the diet (Mcal/kg) × FI (kg/d), $\mathbf{ME_{protein}}$ = ME used for protein gain (g protein gain/d × 10.03 kcal/g of protein gain), and $\mathbf{ME_{lipid}}$ = ME used for lipid gain (g lipid gain/d × 11.65 kcal/g of lipid gain; as reviewed by Patience, 2012). Assuming that the efficiency of protein and lipid gain and the efficiency of

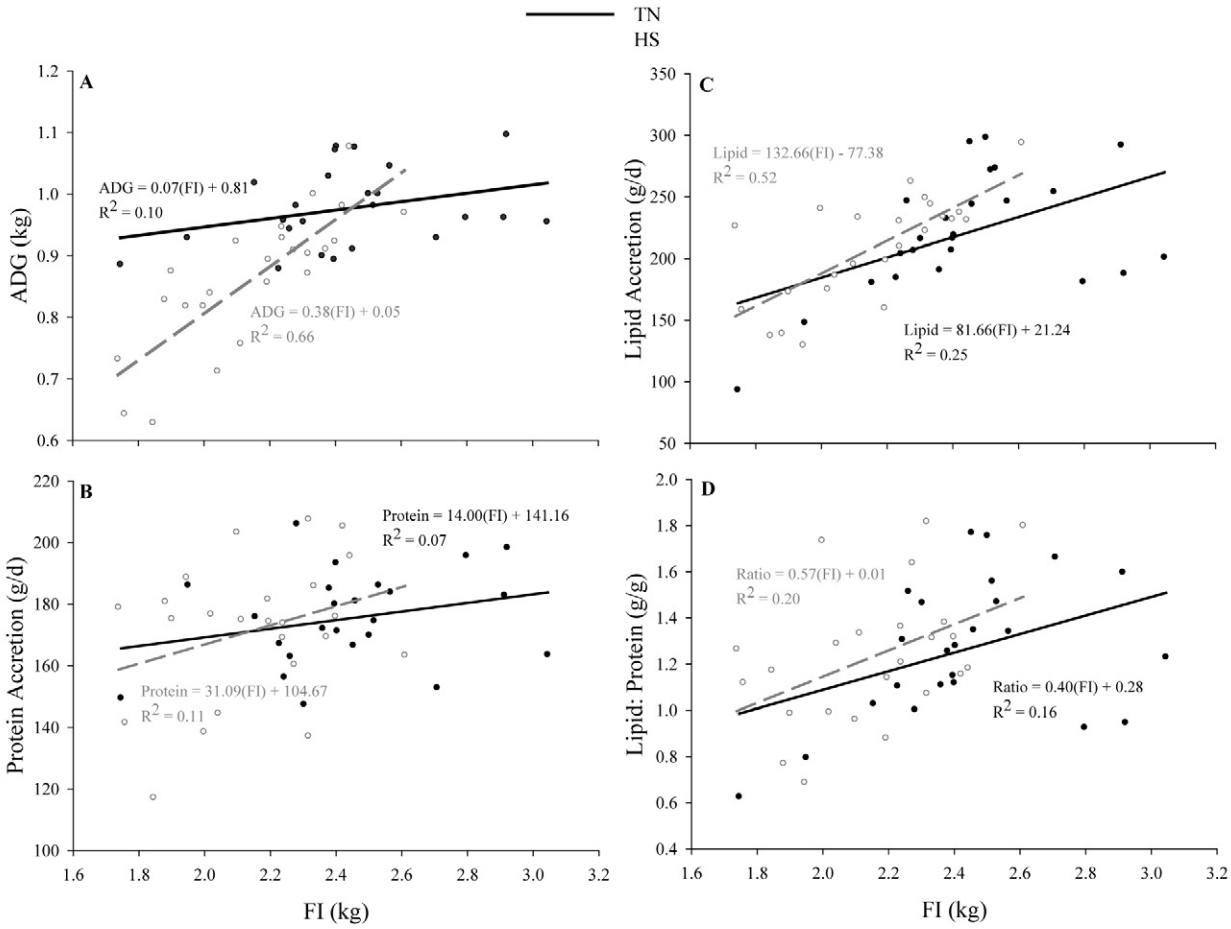

Figure 3. Linear regression ($y = mx + b$) of (A) ADG as a function of feed intake (FI), (B) protein accretion (g/d) as a function of FI, (C) lipid accretion (g/d) as a function of FI, and (D) the ratio of lipid to protein accretion/day as a function of FI in postnatal thermoneutral (TN) pigs and postnatal heat stress (HS) pigs. Coefficient of determination (R^2) and slope (m) is presented for each regression line.

dietary energy use remained unaltered during HS, it appears that pigs exposed to postnatal HS (regardless of in utero environment) required approximately 588 kcal ME/d less energy for $ME_{maintenance}$.

The presumed increase in maintenance costs is the principal reason why HS likely decreases FE as is frequently reported in pig research articles (Kerr et al., 2003; Renaudeau et al., 2008), and reviews (NRC, 1981; Renaudeau et al., 2012). However, the effects of HS on FE are inconsistent as some studies report either no FE differences (Collin et al., 2001a; Johnson et al., 2013a) or actually improved FE (Lefaucheur et al., 1989) in HS compared to TN-exposed pigs. In agreement with the aforementioned reports, gross FE was similar among treatments (0.42 kg gain/kg feed; Table 2). While reasons for this are unclear, it is possible that the reduced maintenance costs observed in the current study increased energy efficiency in HS-exposed pigs. Other indirect evidence also suggests that energy efficiency is enhanced during HS. First, the efficiency of converting dietary energy into body mass was increased during HS (i.e., the relationship between FI and BW gain is steeper in HS compared to TN controls; Fig. 3). Second, despite reduced FI, HS pigs retained 4.0% more MEintake for growth ($ME_{protein}$ + ME_{lipid}) compared to TN controls. These data suggest that although HS pigs consume less feed, a greater percentage of energy is partitioned toward growth, possibly due to reduced maintenance costs and overall heat production compared to TN controls. There are inconsistencies within the literature regarding how environmental HS affects FE and one explanation for this variability may be differences between measurements of FE and CE. For example, in the current experiment, although carcass and head weights were similar, total viscera weight was reduced (7.7%) in HS-exposed pigs compared to TN controls (Table 3), and this confirms reports by others (Lefaucheur et al., 1989; Rinaldo and Le Dividich, 1991). Therefore, although gross FE was similar in the current experiment, CE was actually increased ($P < 0.01$; 12.5%) in postnatal HS compared to TN-exposed pigs (Table 2). These data suggest that reductions in gross FE due to HS (NRC, 1981; Kerr et al., 2003; Renaudeau et al., 2008, 2012) may be a result of decreased visceral mass and not due to a reduced rate

of converting dietary nutrients into skeletal muscle and adipose tissue.

Conclusion

While in utero programming can permanently modify future offspring development, the present study indicates that IUHS had little effect on growth during the period of life primarily characterized by rapid lean tissue accretion. In contrast to in utero effects, postnatal HS reduced whole-body adipose accretion but had no effect on protein accretion rates. When considering that postnatal HS reduced visceral weight, HS pigs had increased efficiency of converting dietary nutrients into carcass tissue, and this has obvious energetic implications for production agriculture. Although it is possible that IUHS may alter future nutrient partitioning later in life (i.e., when skeletal muscle deposition plateaus and adipose accretion is increased), it does not appear to influence tissue accretion during this particular growth phase.

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