# Chronic prenatal heat stress alters growth, carcass composition, and physiological response of growing pigs subjected to postnatal heat stress

Aira Maye Serviento, Bénédicte Lebret, David Renaudeau

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**A. M. Serviento*, B. Lebret*, and D. Renaudeau*, [2]**

*** INRAE, Agrocampus Ouest, PEGASE, 35590 Saint-Gilles, France**

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[2]Corresponding author: david.renaudeau@inrae.fr

## ABSTRACT

Postnatal heat stress (**HS**) effects on pig physiology and performance are widely studied but prenatal HS studies, albeit increasing, are still limited. The objective of this study was to evaluate chronic prenatal HS effects in growing pigs raised in thermoneutral (**TN**) or in HS environment. For prenatal environment (**PE**), mixed-parity pregnant sows were exposed to either TN (**PTN**; cyclic 18 to 24°C; n = 12) or HS (**PHS**; cyclic 28 to 34°C; n = 12) conditions from d 9 to 109 of gestation. Two female offspring per sow were selected at 10 weeks of age and allotted to one of two postnatal growing environments (**GE): GTN** (cyclic 18 to 24°C; n = 24) and **GHS** (cyclic 28 to 34°C; n = 24). From 75 to 140 d of age, GTN pigs remained in GTN conditions, while GHS pigs were in GTN conditions from 75 to 81 d of age and in GHS conditions from 82 to 140 d of age. Regardless of PE, postnatal HS increased rectal and skin temperatures (+0.30 and +1.61 °C on average, respectively; $P < 0.01$), and decreased ADFI (-332 g/d; $P < 0.01$), resulting in lower ADG and final BW (-127 g/d and -7.9 kg, respectively; $P < 0.01$). The GHS pigs exhibited thicker backfat ($P < 0.01$), lower carcass loin percentage ($P < 0.01$), increased plasma creatinine levels ($P < 0.01$), and decreased plasma glucose, nonesterified fatty acids, T3, and T4 levels ($P < 0.05$). Prenatal HS increased feed intake in an age-dependent manner (+10 g•kg $BW^{-0.60}$•$d^{-1}$ for PHS pigs in the last 2 weeks of the trial; $P = 0.02$) but did not influence BW gain ($P > 0.10$). Prenatal HS decreased plasma levels of superoxide dismutase on d 3 of GHS (trend at $P = 0.08$) and of T4 on d 49 ($P < 0.01$) but did not affect T3 on d 3 nor 49 ($P > 0.10$). Prenatal HS increased rectal and skin temperatures, and decreased temperature gradient between skin and rectal temperatures in GTN pigs (+0.10°C, +0.33 and -0.22°C, respectively; $P < 0.05$) but not in GHS pigs ($P > 0.10$). There were also PE×GE interactions found with lower BW ($P = 0.06$) and higher backfat ($P <$

0.01) and perirenal adiposity ($P < 0.05$) for GHS-PHS pigs than the other groups. Overall, increased body temperature, and altered thyroid functions and physiological stress responses suggest decreased heat tolerance and dissipation ability of pigs submitted to a whole-gestation chronic prenatal HS. Postnatal HS decreased growth performance, increased carcass adiposity, and affected metabolic traits and thyroid functions especially in pigs previously submitted to prenatal HS.

**Key words:** carcass adiposity, growth, pig, prenatal heat stress, postnatal heat stress, thermoregulation

**ABBREVIATIONS**

BAP: biological antioxidant potential

CK: creatine kinase

BFT: backfat thickness

FCR: feed conversion ratio

FI: feed intake

GE: growing environment

GHS: growing heat stress

GTN: growing thermoneutral

HP: heat production

HPA hypothalamic-pituitary-adrenal

HS: heat stress

LDH: lactate dehydrogenase

NEFA: nonesterified fatty acids

PE: prenatal environment

PHS: prenatal heat stress

PTN: prenatal thermoneutral

SOD: superoxide dismutase

TN: thermoneutral

## INTRODUCTION

As extreme heat events become longer, more frequent, and more intense, the impact on swine production also increases (Luber and McGeehin, 2008; IPCC, 2014). During postnatal heat stress (**HS**), pigs decrease their feed intake (**FI**) as an adaptive response to reduce heat production (**HP**) (Renaudeau et al., 2008; Renaudeau et al., 2013). These negative effects are heightened with the selection for higher lean percentage as HP increases with higher lean tissue accretion rate (Brown-Brandl et al., 2014) resulting to less heat-tolerant pigs. In female mammals like the sow, hyperthermia can induce physiological changes that can impair oocyte development, early embryonic development, fetal and placental growth, and nursing performance (as reviewed by Hansen, 2009) which can all affect the subsequent growth of the offspring. It is thus, important to understand how prenatal HS can affect the pig's postnatal performance.

Prenatal HS can be defined as the in-utero exposure of the offspring to maternal hyperthermia (Edwards, 1969; Lary, 1986). There are many studies in mice suggesting that prenatal HS can depress brain and body growth (Shiota and Kayamura, 1989; Hinoue et al., 2001), although effects of prenatal stress in general can also depend on stress duration: acute prenatal stress enhances protection of fetus from maternal corticosteroids but chronic prenatal stress weakens it (Welberg et al., 2005). In farm animals, thermal conditioning during incubation have been reported to influence adaptive HS response of poultry species (Loyau et al., 2015) while in calves, prenatal HS can alter DNA methylation profile and reduce growth performance (Monteiro et al., 2016; Skibiel et al., 2018). Recent studies in pigs suggest that sows exposed to gestational HS produced pigs with increased core body temperature and altered metabolic processes, body composition, and thermal responses (Boddicker et al., 2014; Cruzen et al., 2015; Johnson et al.,

2015a; Johnson et al., 2015b; Johnson et al., 2015c; Chapel et al., 2017). The objective of this study was to evaluate the effects of chronic prenatal HS on the growth performance, body composition, and physiological responses of growing pigs in postnatal thermoneutral (**TN**) or HS environment.

## MATERIALS AND METHODS

The experiment was conducted in accordance with the French legislation on animal experimentation and was approved by the French National Committee for Consideration of Ethics in Animal Experimentation (Authorization: APAFiS #11016-2017080718212019 delivered on September 26, 2017).

### *Experimental design and animal management*

The study was conducted in the INRAE experimental facilities at the Unité Expérimentale Porcs de Rennes (UEPR) located in Saint-Gilles, France from October 2017 to June 2018. The general framework of the experimental study is presented in Fig. 1.

***Gestating and lactating sows*.** A total of 16 gilts and 16 multiparous sows (16 blocks of 2 sisters) were initially blocked according to parity and litter origin with additional blocking factors for the multiparous sows, i.e. BW and backfat thickness (**BFT**) at weaning of their respective litters. The animals were kept in one of two identical rooms during their pregnancy: one thermoneutral (**PTN**) room and one heat-stressed (**PHS**) room. Each room was equipped with two pens. In both rooms, gilts and sows were placed in separate pens (8 animals per pen of 4.5 × 4.8 m). The gilts were moved to the gestation rooms 3 weeks prior the expected date of breeding while the sows were blocked and transferred on the day of weaning immediately after weighing and BFT

measurement. Sows were artificially inseminated with 4 different sire origins, with one sire used to inseminate 4 females (2 multiparous and 2 primiparous sows) per treatment. One PHS primiparous sow was removed prior to insemination because of urogenital infection. From d 0 to 6 of gestation, the ambient temperature was kept under cyclic TN conditions (18 to 24°C) in both experimental gestation rooms. The PTN sows were maintained at this environmental temperature regimen until d 109 of gestation. In the PHS room, the ambient temperature was gradually increased from d 6 to 9 and thereafter maintained under cyclic HS conditions (28 to 34°C) from d 9 to 109 of gestation. Whatever the temperature treatment, the minimum and maximum temperatures were reached at 0600 h and 1800 h, respectively (Fig. 2a). All animals were given a commercial gestation feed (13.6% CP; 2,300 kcal/kg NE) following a daily individual feed allowance calculated according to Dourmad et al. (1997). The daily ration was distributed in two meals at 0830 h and at 1600 h and water was provided *ad libitum.*

On d 110 of gestation, 12 sows (6 primiparous and 6 multiparous) from each temperature treatment were selected based on litter origin, BW, and BFT and were distributed equally to one of two identical farrowing rooms equipped with 12 pens and maintained in constant TN conditions of 25°C. Cross-fostering was done within each pregnancy treatment (PTN or PHS) and within the parity, e.g., piglet of a primiparous PTN sow can only be fostered to another primiparous PTN sow. Sows were placed in individual pens (1.79 × 2.38 m) until weaning (d 28 of lactation). From d 1 to 5 of lactation, sows were given individual rations of 0.5, 1.5, 3.0, 5.0, 6.0 kg/d, respectively. From d 6 to 28 of lactation, feed and water were provided *ad libitum*. The commercial lactation feed (16.5% CP; 2,343 kcal/kg NE) was distributed 3 times per day (0800 h, 13h00 h, and 1600 h).

130 ***Weanlings and growing pigs***. At weaning, 10 littermate piglets (3 entire males, 4 castrated
131 males, and 3 females) were selected from each of the 24 sows (total of 240 piglets). The pigs
132 selected were those closest to average weaning BW of pigs from PTN and PHS groups (i.e., 9.7
133 kg and 9.5 kg, respectively). During post-weaning, the 10 selected littermates were housed in one
134 pen (1.45 × 2.72 m), hence one litter per pen. The 24 litters were allocated to two rooms, with 6
135 PTN and 6 PHS litters in each room. The piglets were given a standard pre-starter diet (18.9% CP;
136 2,498 kcal/kg NE) for one week and a standard starter diet (18.0% CP; 2,262 kcal/kg NE) until the
137 end of post-weaning. Water and feed were provided *ad libitum*, and the rooms were maintained in
138 constant TN conditions (25°C) throughout the 6 weeks of post-weaning.

139 At 10 weeks of age, 48 females were randomly selected from these 240 piglets (choice of
140 2 out of the 3 females per litter). Considering the prenatal environment (**PE**: PTN vs. PHS), the 2
141 selected females per litter were allotted to one of two similar growing (G) rooms with two different
142 thermal environments (**GE**: GTN or GHS). Each room was equipped with 8 pens (2 × 2 m)
143 designed for housing 3 pigs each. To balance out the experimental design, the pigs were blocked
144 according to litter origin so that in one pen, all pigs were half-sisters (same sire). Moreover, each
145 pig of one pen in the GTN room has a full sister in the corresponding pen of the GHS room.

146 The experiment, which started after 5 days of adaptation, was divided into two main
147 periods. The first main period was from 75 to 81 d of age where all pigs were kept under cyclic
148 TN conditions (18 to 24°C). The second main period started at 82 d of age (d 0; transition day for
149 the GHS pigs from TN to HS conditions) when temperature in the GHS room was gradually
150 changed at a rate of 1°C/hour from 0600h to 1800h and was thereafter maintained in a cyclic HS
151 conditions of 28 to 34°C until 140 d of age (Fig. 2b). The GTN room was maintained in cyclic TN

conditions from 82 to 140 d of age. *Ad libitum* access to a standard growing-finishing feed (16.3% CP; 2,495 kcal/kg NE) and to water was provided throughout the growing period. Meals were distributed three times daily (0900 h, 1300 h, and 1600 h). The pigs were slaughtered at 140 d of age.

***Measurements***

***Growth and slaughter performance.*** For the overall growth performance, the two main periods previously described (i.e. 75 to 81 and 82 to 140 d of age) were considered. The pigs were individually weighed at the beginning of each main period, every one or two weeks during the experimental period, and on the day before slaughter. The daily feed intake was measured for each pen as the difference between offered and refused feed. Refusals and spillages were collected daily at 0800 h before the first feed distribution of the day; their DM (103°C for 24 h) were also determined daily.

Pigs were slaughtered in the experimental slaughterhouse of INRAE-UEPR after 24 h fasting. Pigs were slaughtered by electrical stunning and exsanguination in compliance with the current national regulations applied in slaughterhouses. Hot carcass, perirenal fat, and head were weighed just after slaughter. Weights of the hypothalamus and pituitary gland were also recorded. Backfat (G2) and muscle (M2) depths were measured on one dorsal spot between the 3rd and 4th last ribs at 6 cm of spinal canal axis, using a CGM device (Fives Syleps, Lorient, France). Backfat thickness was measured on carcass split at three different locations: on the first and last ribs, and on the Gluteus muscle (minimum fat). Length of the left side of the carcass was also measured. The day after slaughter, cold carcass and wholesale cuts from the right carcass side (ham, loin, shoulder, belly and backfat) were weighed.

174 ***Physiological parameters***. Rectal temperature was measured using a digital thermometer
175 (Microlife Corpororation, Paris, France; accuracy ±0.1°C) and skin temperature by a Type K
176 thermocouple probe (HH-21 model, Omega, Stamford, CT, USA; accuracy ±0.1°C). These
177 measurements were done on the sows at d 4, 9, 12, 29, 60, 106, 110 of gestation and d 2, 6, 13, 20
178 and 26 of lactation. In the growing pigs, rectal and skin temperatures were measured at 1300 h on
179 d -5, 0, 2, 3, 7, 29, 43, and 53 of second main experimental period. Skin temperature was not
180 measured on d 3. On all pigs, at 1330 h on d -4, 3, and 49, blood was collected at the jugular vein
181 in heparin tubes, centrifuged (3,000 g; 10 min; 4ºC), and plasma was stored at -20°C until analysis.
182 Commercially available kits were used to measure plasma levels of creatinine [Creatinine (Jaffe),
183 Thermo Fisher Scientific Oy, Vantaa, Finland], glucose [Glucose (HK), Thermo Fisher Scientific
184 Oy, Vantaa, Finland], nonesterified fatty acids or **NEFA** (FUJIFILM Wako Chemicals Europe
185 GmbH, Neuss, Germany), and biological antioxidant potential or **BAP** (Diacron Labs srl,
186 Grosseto, Italy). Intra-assay CV were 4.9%, 1.7%, 0.4%, and 3.5%, respectively. Inter-assay CV
187 were 7.0%, 8.4%, 2.4%, and 5.1%, respectively. For the enzymatic activities, creatine kinase or
188 **CK** [CK (IFCC), Thermo Fisher Scientific Oy, Vantaa, Finland], lactate dehydrogenase or **LDH**
189 [LDH (IFCC), Thermo Fisher Scientific Oy, Vantaa, Finland], and superoxide dismutase or **SOD**
190 (Sigma-Aldrich, St Louis, MO) were measured. Intra-assay CV 11.1%, 1.1%, and 4.1%,
191 respectively. Inter-assay CV were 16.8% and 17.2% for CK and LDH, respectively. Plasma levels
192 of thyroid hormones of T3 and T4 (ST AIA-PACK TT3 and ST AIA-PACK T4, Tosoh
193 Corporation, Tokyo, Japan) were also determined. Intra-assay CV were 3.8% and 3.9%,
194 respectively.

195 ***Calculations***

196 Live BW measured on 75, 82, and 140 d of age were considered. Growth performance was
197 calculated in two ways. First, for the overall growth performance, average performance of the two
198 main periods were considered (75 to 82 and 82 to 140 d of age). Since pen was the experimental
199 unit, the ADG for a given period corresponded to the mean of the three individual ADG. The ADFI
200 (measured per pen and divided by 3) was expressed in two ways: as the classical ADFI (g/d) and
201 as the ADFI per metabolic BW ($g \cdot kg^{-0.60} \cdot d^{-1}$). The feed conversion ratio (**FCR**) was calculated as
202 the feed intake divided by the BW gain for a given pen and for a given period. For data collected
203 only from 82 to 140 d of age, this second main period was split into 5 sub-periods: sub-period 1
204 (d 0 to 6; with d 0 as the transition of the GHS room to cyclic HS conditions), and sub-periods 2,
205 3, 4, and 5 (d 7 to 15, d 16 to 29, d 30 to 43, and d 44 to 58, respectively). Growth performance
206 (ADFI per metabolic BW and ADG) was calculated for each sub-period.

207 For the carcass traits, carcass dressing was calculated as percentage of hot carcass to
208 slaughter BW. Wholesale cut weights were expressed as percentage of the cold right carcass side.
209 Carcass lean meat content was calculated using the CGM measurements (G2 and M2) according
210 to the equation by Daumas et al. (2010): Lean meat content (%) = 62.19 − 0.729 G2 + 0.144 M2.
211 Average BFT was calculated as the mean of the measurements from the 3 different locations
212 previously described. For the thermoregulation parameters, temperature gradient was calculated
213 as the difference between rectal and skin temperatures. Data of enzymes CK and LDH were log-
214 transformed to follow normal distribution.

215 ***Statistical Analyses***

216 According to the factorial design based on 2 postnatal growing environments (**GE; GTN**
217 and **GHS**) and 2 prenatal environments (**PE; PTN** and **PHS**), there were 4 treatments (i.e., GTN-
218 PTN, GTN-PHS, GHS-PTN, and GHS-PHS) with 4 pens per treatment, for a total of 12 pigs per
219 treatment.

220 For the overall growth performance, the pen (n=16) was considered as the experimental
221 unit and data were analyzed using a repeated measure of the PROC MIXED procedure (SAS Inst.
222 Inc., Cary, NC) considering PE (n=2), GE (n=2), the two main periods (n=2; n=3 for live BW),
223 their interactions, and sire (n=4) as fixed effects. The average growth performance during the
224 second main period was also analyzed using PROC MIXED model with the PE (n=2), GE (n=2),
225 their interaction, and sire (n=4) as fixed effects, and including the growth performance during the
226 first main period as covariates. For growth performance calculated per sub-period (ADG and ADFI
227 per metabolic BW), data were analyzed using a repeated measure of the PROC MIXED procedure
228 considering the PE (n=2), GE (n=2), sub-period (n=5), their interactions, and sire (n=4) as fixed
229 effects and with performance measured during the first main period as covariates.

230 For carcass and physiological parameters, the pig (n=48) was used as the experimental unit.
231 Individual pig data were analyzed using the PROC MIXED procedure with the PE (n=2), GE
232 (n=2), their interaction, pen (n=16), and sire (n=4) as fixed effects. Slaughter BW was included in
233 the model as covariate for data analysis of carcass traits. Thermoregulation and blood parameters
234 were subjected to a repeated measurement PROC MIXED procedure based on the days of
235 measurement (n=8 for rectal temperature, n=7 for skin temperature and temperature gradient, and
236 n=3 for blood parameters), and the interactions with PE and with GE. Thermoregulation responses

were also subjected to another repeated measurement analysis but only considering measurements during the second main period (d 2, 3, 7, 29, 43, and 53 measurements).

## RESULTS

The average hourly temperature of the rooms (Fig. 2) indicates that the actual average minimal temperatures (20 to 25°C for PTN; 29 to 34°C for PHS; 19 to 24°C for GTN; 29 to 34°C for GHS) were slightly higher than the targeted temperatures.

***Thermoregulatory responses.*** Results for the skin and rectal temperatures of the sows during gestation and lactation are presented in Fig. 3. For the overall duration of HS exposure, gestating PHS sows had higher skin temperature (36.40 vs. 33.96°C on average; $P < 0.001$) and rectal temperature (38.51 vs 38.35°C on average; $P = 0.020$) than PTN sows. For growing pigs, thermoregulation data of 2 pigs from GTN-PHS group were removed because they were sick from d 0 to 2 of the second main period. The skin and rectal of the growing pigs are presented in Fig. 4. Considering only the second main period, regardless of the PE, GHS pigs had higher skin temperature (37.55 vs. 35.94°C on average; $P < 0.001$) and rectal temperature (39.57 vs. 39.27°C on average; $P < 0.001$) compared to their GTN counterparts. On d 2 of the second main period, GHS pigs had higher skin temperature (37.89 vs 36.27°C on average; $P < 0.001$) and rectal temperature (39.96 vs 39.43°C on average; $P < 0.001$) compared to GTN pigs. Thereafter, skin temperature remained significantly and constantly higher in GHS than in GTN pigs until d 53 (37.45 vs. 36.11°C on average; $P < 0.001$), whereas rectal temperature gradually decreased and by d 53, the difference was less pronounced although still significant (39.30 vs. 39.15°C on average; $P = 0.011$).

Regarding the interaction between PE × GE, the rectal temperature of the 2 GHS groups was not different from those of GTN-PHS pigs ($P > 0.100$) but was higher than those of GTN-PTN pigs on d 7, 43, and 53 of HS ($P < 0.050$). Considering all repeated measures during the second main period, an overall significant PE × GE interaction was also observed for skin temperature ($P = 0.005$) and for temperature gradient ($P = 0.018$; data not shown). In GTN conditions, PHS pigs had higher skin temperature (36.10 vs. 35.77°C on average; $P < 0.001$) and narrower temperature gradient (3.20 vs. 3.42°C on average; $P = 0.015$) than PTN pigs, but not in GHS conditions (37.55°C on average, $P = 0.979$ for skin temperature, and 1.96°C on average $P = 0.997$ for temperature gradient). In GTN conditions, the rectal temperature of PHS pigs were also higher than those of PTN pigs (39.32 vs. 39.22°C on average; $P = 0.038$), but not in GHS conditions (39.57°C on average; $P = 0.735$).

***Growth performance***. Table 1 shows the summary of the growth performance of the growing pigs starting at 35.9±0.8 kg BW. The interaction between PE and GE treatments was not significant except a trend ($P = 0.059$) for the live BW, with PHS pigs tending to be lighter at final BW than PTN pigs when raised in GHS conditions, but not in GTN conditions. Regardless of PE treatment, GHS pigs had lower final BW than GTN pigs (97.2 vs 105.1 kg; $P < 0.001$). Neither GE nor PE had significant effect on the FCR ($P = 0.221$ and $P = 0.549$, respectively). In the second main period (82 to 140 d of age), the GHS pigs had overall lower performance than GTN pigs in terms of ADG (1,003 vs. 1,130 g/d on average; $P = 0.017$), ADFI (2,327 vs. 2,659 g/d on average; $P = 0.008$), and ADFI per metabolic BW (199 vs. 181 $g \bullet kg^{-0.60} \bullet d^{-1}$ on average; $P = 0.045$). When corrected for the same performance during the first main period, PHS tended to have higher ADFI

per metabolic BW than PTN pigs in the second main period (190 vs. 185 g•kg$^{-0.60}$•d$^{-1}$ on average; $P = 0.097$).

Looking at the sub-periods during the second main period (Fig. 5), there were no significant interaction between GE and sub-period for ADFI per metabolic BW ($P = 0.819$) and ADG ($P = 0.467$) since GHS pigs performed consistently lower than GTN pigs ($P < 0.050$). Meanwhile, there was a significant interaction between PE and sub-period for ADFI per metabolic BW ($P = 0.012$). Regardless of the GE group, PHS pigs ate more than PTN pigs starting only from the sub-period 3 with this difference being significant on sub-period 5 (186 vs. 175 g•kg$^{-0.60}$•d$^{-1}$ on average for PTN and PHS pigs, respectively; $P = 0.022$). There was also a tendency for a PE and sub-period interaction ($P = 0.061$) for ADG but no significant difference was seen between PHS and PTN pigs in any of the sub-periods.

***Carcass and organ traits.*** The slaughter performance of the pigs is presented in Table 2. The slaughter BW of GHS pigs was lower than those of GTN pigs ($P < 0.001$), while the hot carcass weight of GHS-PHS pigs was lower than those of the 2 GTN groups ($P < 0.006$) but not different from those of the GHS-PTN pigs ($P = 0.431$). Postnatal HS decreased carcass length ($P < 0.001$) and tended to increase carcass dressing ($P = 0.068$) but was not affected by the prenatal treatment. Expressed as a percentage of the slaughter BW, PHS pigs tended to have lower head weight ($P = 0.066$) than PTN pigs, regardless of the GE.

Looking at carcass traits adjusted for the same slaughter BW, lean meat content was decreased both by postnatal HS ($P = 0.028$) and prenatal HS ($P = 0.029$), with GHS-PHS pigs having the lowest lean meat content compared to the three other groups ($P < 0.040$). A PE × GE interaction was also observed for average BFT ($P = 0.003$) and for cold carcass proportions of

backfat ($P$ = 0.016) and of perirenal fat ($P$ = 0.017), with PHS pigs being fatter than their PTN counterparts when they were submitted to postnatal HS. Irrespective of the prenatal treatment, postnatal HS also increased average BFT ($P$ < 0.001) and ham percentage ($P$ = 0.001), and decreased loin percentage ($P$ < 0.001). Meanwhile, prenatal HS decreased ham percentage ($P$ = 0.007) regardless of the GE treatment. Neither postnatal nor prenatal HS affected belly percentage ($P$ = 0.171 and $P$ = 0.514, respectively).

For the brain parts, a PE and GE interaction was observed for the weight of the hypothalamus ($P$ = 0.022) and of the pituitary gland ($P$ = 0.038) expressed per kg BW. Hypothalamus of GTN-PTN pigs were heavier ($P$ = 0.027) than that of GTN-PHS pigs but were not different to the 2 GHS groups ($P$ > 0.050). Meanwhile, pituitary gland of GHS-PHS pigs were heavier ($P$ < 0.050) than those of GTN-PHS and GHS-PTN pigs but not different ($P$ = 0.223) to that of GTN-PTN pigs.

***Plasma parameters.*** Whatever the parameter, plasma concentration was similar among all 4 treatments on d -4 (Fig. 6). The PE × GE interaction was significant for some blood parameters at some specific days. Plasma creatinine concentration was higher in GHS pigs (GHS-PTN and GHS-PHS) compared to GTN-PHS pigs on d 3 ($P$ = 0.049 and $P$ = 0.026, respectively), and was highest in GHS-PTN pigs among all groups on d 49 ($P$ < 0.030). Plasma glucose of the GTN pigs (GTN-PTN and GTN-PHS) were higher than the GHS-PTN pigs on d 49 ($P$ = 0.008 and $P$ = 0.036, respectively). Plasma NEFA of the GHS pigs (GHS-PTN and GHS-PHS) were lower only when compared to GTN-PHS pigs ($P$ = 0.001 and $P$ = 0.008, respectively) and only on d 49. The GTN groups (GTN-PTN and GTN-PHS) had higher SOD plasma levels on d 3 ($P$ = 0.004 and $P$ = 0.031, respectively) and higher plasma T3 levels on d 49 ($P$ = 0.006 and $P$ = 0.025, respectively)

compared to GHS-PHS pigs. And on d 49, plasma T4 was higher in GTN-PTN pigs than in GHS-PHS pigs ($P = 0.003$).

Regardless of PE treatment, acute and chronic postnatal HS (on d 3 and d 49, respectively) increased creatinine ($P = 0.012$ on d 3; $P < 0.001$ on d 49), and decreased glucose ($P < 0.001$ on d 3; $P = 0.002$ on d 49), T3 ($P < 0.001$ on d 3 and d 49), and T4 ($P < 0.001$ on d 3; $P = 0.020$ on d 49) plasma concentrations. Compared to GTN pigs, plasma NEFA levels of GHS pigs were lower on d 3 (trend at $P = 0.074$) and on d 49 ($P < 0.001$) Higher activities of CK and LDH ($P < 0.001$) were observed in GHS pigs but only on d 3. Postnatal HS also decreased plasma SOD levels on d 3 ($P = 0.004$) and BAP levels on d 49 ($P < 0.001$). Regardless of the postnatal treatment, plasma SOD level tended to be lower ($P = 0.082$) in PHS pigs than in PTN pigs on d 3. Prenatal HS also decreased plasma T4 on d 49 ($P = 0.007$) without a significant decrease in plasma T3 ($P = 0.305$).

## DISCUSSION

In this study, we investigated the effects of both prenatal and postnatal thermal environments on performance and physiological responses in growing pigs. The cyclic HS treatments applied in the experiment were enough to elicit responses from both the PHS sows and GHS pigs as they exhibited biphasic acclimation HS responses similar to previous studies (Black et al., 1993; Renaudeau et al., 2010). This response, as described by Renaudeau et al. (2010), is characterized by a short-term heat acclimation where pigs experience rapid physiological changes as shown by the spike in rectal temperature and followed by a long-term heat acclimation where animals show improved heat tolerance by increasing ability to dissipate heat and to decrease HP. The higher skin

345 temperature of PHS sows and GHS pigs throughout the thermal challenge can be related to the
346 shift of the blood flow more toward the peripheral tissues and away from internal tissues (Collin
347 et al., 2001a). The GHS pigs in our study were able to adapt to chronic HS as reflected in their
348 decreasing rectal temperatures. Exposure to HS has been shown to decrease FI and to decrease
349 metabolic HP due to a reduction in maintenance requirements as adaptation responses (Quiniou
350 et al., 2000; Renaudeau et al., 2013). The FI reduction was not observed in gestating sows
351 submitted to HS (data not shown) because a large part of the ration was given at 0800 h when the
352 ambient temperature was low and because they had a strict daily feed allowance, thus leaving no
353 room for refusals.

354 ***Effects of postnatal HS on growth performance and metabolism***

355 In the present study, postnatal HS decreased ADFI per metabolic BW of growing pigs by 39
356 $g \bullet kg^{-0.60} \bullet d^{-1}$ on average, which is slightly lower than the values reported by Renaudeau et al.
357 (2008) in younger pigs (-40, -65, and -104 $g \bullet kg^{-0.60} \bullet d^{-1}$ at constant temperatures of 28, 32 and
358 36°C, respectively, compared with controls). It is possible that implementing cyclic rather than
359 constant heat stress conditions allowed the pigs in the present study to compensate by eating more
360 feed during the colder parts of the day (decrease in ADFI is lower at 28°C than at 32°C).
361 Nevertheless, the reduction in ADFI had negative consequences on growth rate due to reduction
362 in nutrient intake similar to a previous study (Collin et al., 2001b).

363 Carcass traits were adjusted for the same slaughter BW in our study to evaluate the strict
364 effect of HS on carcass composition and to take into account study limitations of not having the
365 pigs slaughtered at the same live BW. Based on the fact that maximum protein deposition can be
366 reached at 80 kg and maximum fat deposition is not reached even in the range of 110 to 130 kg

BW (Van Milgen and Noblet, 2003), it can be assumed that GHS pigs (average of 95.3 kg at slaughter) would deposit more fat than lean if they had been slaughtered at the same BW (but older age) as GTN pigs (average of 103.0 kg at slaughter). In our study, the GHS pigs were fatter than GTN pigs which is in contrast to other studies with *ad libitum*-fed pigs subjected to constant high ambient temperature (32 to 33°C) being leaner and having less lipid deposition than pigs in TN conditions (Collin et al., 2001b; Cruzen et al., 2015). Differences in results could be related to the previously discussed higher level of ADFI reduction in pigs under constant HS conditions compared to the cyclic HS conditions implemented in our study. Kouba et al. (2001) reported increased triglyceride uptake and storage in HS pigs compared to their pair-fed TN counterparts and the "additional" FI in our study could have been deposited as fat since pigs decrease their metabolic HP during chronic postnatal HS and it is more energy-efficient to deposit fat (Van Milgen and Noblet, 2003). The fatter carcasses of GHS pigs are logically associated to lower loin (lean cut) percentage, whereas the increased ham percentage of GHS pigs may be explained by changes in conformation as shown by their shorter carcass.

The drop in plasma thyroid hormone levels of GHS pigs can be linked to the reduction of their ADFI, and to the lower growth and HP (Collin et al., 2002). Since thyroid hormones stimulate dietary and endogenous fat breakdown (Sinha et al., 2018), the lower T3 and T4 levels found in postnatal HS pigs in our study and in agreement with Sanz Fernandez et al. (2015) could contribute to the increased body fat content of GHS compared to GTN pigs, as also shown in rats (Iossa et al., 2001). Plasma NEFA of GHS pigs were also lower which can be due to lower amounts of lipid ingested because of the reduced FI, or to a limited ability of HS pigs to mobilize fat as demonstrated by Pearce et al. (2013). Indeed, it has been previously suggested that pigs subjected

389 to postnatal HS had lower metabolic flexibility with a lower fatty acid oxidation in their skeletal
390 muscles than pair-fed TN pigs (Baumgard and Rhoads, 2013; Zhao et al., 2018). The elevated
391 plasma creatinine level and CK activity in GHS pigs can also suggest increased muscle protein
392 catabolism instead of fat catabolism for energy production (Clarkson et al., 2006). Lower plasma
393 SOD (in acute HS) and plasma BAP (in chronic HS) levels measured in GHS pigs could be
394 indications of reduced antioxidant capacities similar to previous reports in pigs (Yang et al., 2014;
395 Liu et al., 2018) which, in humans, could be markers of metabolic syndrome such as lower
396 adiponectin and increased insulin (Kim et al., 2014). Altogether, results of our study thus validates
397 effects of postnatal HS as reported in existing literature such as reduction in growth performance,
398 increased carcass adiposity, and altered physiology and metabolism.

399 ***Effects of prenatal HS on growth performance and metabolism in growing pigs raised in***
400 ***postnatal TN conditions.***

401 The effects of prenatal HS on growth performance, metabolism and physiology in swine have
402 been studied in the past few years as reviewed by Johnson and Baumgard (2018). According to
403 Johnson et al. (2015c), prenatal HS increased core temperature of growing pigs which is
404 comparable to our results. Prenatal stress and increased cytokines in the fetal environment have
405 both been reported to alter set point in the hypothalamic-pituitary-adrenal (HPA) axis (Glover et
406 al., 2010; Dreier et al., 2014), which plays a role in the body temperature control of mammals
407 including pigs (Bligh, 1966; Baldwin and Ingram, 1968). In our study, there were indeed
408 differences in the relative weight of hypothalamus and pituitary gland among the treatments. The
409 anterior and posterior parts of these glands have different functions which could explain why the
410 glands did not have the same pattern of increase or decrease in weight. However, the gland parts

411 were not weighed separately in our study, so this remains to be theoretical. Nevertheless, pituitary
412 gland size itself does not signify a lower or higher hormone activity; it can only be suggested that
413 these observed changes could be related to morphological changes and/or abnormality which, in
414 humans, is related to altered pituitary function (Maghnie et al., 1991; Cooper et al., 2017). These
415 changes could also be related to smaller head size of GHS pigs observed in our and in other studies
416 (Cruzen et al., 2015; Johnson et al., 2015b). It can thus be hypothesized that the higher rectal
417 temperature of prenatal heat-stressed pigs observed at many stages during growing phase could be
418 a consequence of an altered HPA set point due to their fetal environment.

419 Chapel et al. (2017) suggested that the elevated core temperature of pigs submitted to in-utero
420 HS pigs during mid-gestation could be associated to higher fasting HP and circulating T3 levels.
421 Meanwhile, Johnson et al. (2015c) found that although in-utero HS pigs in their study also
422 exhibited increased body temperature, they found no effect in plasma levels of T4 (a pro-hormone)
423 nor of T3 (active form of T4) regardless of the timing (first or second half of gestation) and the
424 length of gestational HS. In our study, however, PHS pigs subjected to whole-gestation HS had
425 lower levels of plasma T4 with no significant change in plasma T3 levels. Nevertheless, these
426 results suggest that prenatal HS, depending on the timing and duration, can alter thyroid functions
427 which to some extent could also be associated to the previously discussed hypothesis of an altered
428 HPA set point. Between the two GTN groups in our study, those subjected to prenatal HS also had
429 higher skin temperature denoting increased heat dissipation activity (possibly from the increased
430 rectal temperature), and had narrower temperature gradient implying a lower capacity to further
431 dissipate heat (Cuddy et al., 2014).

The increased core temperature could also be partly attributed to a higher thermic effect of feeding linked to the age-dependent ADFI increase of PHS pigs in our study. This higher ADFI of prenatally heat-stressed pigs, however, failed to translate to a significant ADG increase in agreement to previous studies (Cruzen et al., 2015; Safranski et al., 2015; Wilmoth et al., 2015). This could somewhat be related to what happened during lactation since the weaning BW difference between PTN and PHS pigs could have affected the, although insignificant, small differences in initial BW among groups. There is also an age-dependent decrease in feed efficiency in in-utero HS pigs as it was observed during the finishing phase (Johnson et al., 2015b) but not in the growing phase (Johnson et al., 2015a). These results suggest limited ability of PHS pigs to convert increased intake to gain which can be an effect of intrauterine growth restriction due to fetal undernutrition (Ji et al., 2017). This can be related to reports of prenatal HS causing placental insufficiency or inefficiency (Galan et al., 1999; Zhao et al., 2019) perhaps due to blood redirection away from the placenta in heat-stressed sows. According to Boddicker et al. (2014), the positive effect of prenatal HS on subsequent pig FI is dependent on the HS timing, pigs from sows heat-stressed in the first half of gestation ate more than pigs from sows heat-stressed in the second half of gestation. The differences Altogether, this suggests that prenatal HS can indeed affect postnatal pig performance. Whether this effect is neutral, positive, or negative seem to depend, among other things, on age of the offspring, and the timing and duration of the prenatal HS.

In-utero heat stressed pigs reared in thermoneutral conditions have been described to have increased fat deposition in some studies (Boddicker et al., 2014; Johnson et al., 2015b); however, other studies found no effect (Cruzen et al., 2015; Johnson et al., 2015a) comparable to our results. Differences between studies could be due to several factors, such as pig physiological stage

454 considered or sex : fatter carcasses of prenatal HS in TN-reared pigs have been observed in mixed-
455 sex (barrows and females) finishing pigs (Johnson et al., 2015b), but not in mixed-sex young and
456 growing pigs (Boddicker et al., 2014; Johnson et al., 2015a) nor in finishing barrows (Cruzen et
457 al., 2015). These discrepancies may be at least partly explained by differential effects of prenatal
458 HS on pig FI between studies, as the increased FI generally observed with increasing pig age and
459 BW is associated to increased fat and decreased protein deposition (Van Milgen and Noblet, 2003).
460 In our study the increased FI of PHS pigs was significant only at the end of the experimental
461 growth period, which was probably insufficient to significantly influence body fatness of PHS pigs
462 in postnatal TN conditions. Finally, as mentioned previously, prenatal HS effect can depend on
463 the timing and duration. For example, pigs exposed to prenatal HS only in the first half of gestation
464 had thicker subcutaneous than control pigs, but pigs exposed during the entire gestation exhibited
465 no significant response (Boddicker et al., 2014), in agreement with present results. Overall, results
466 from our study and from existing literature show that in pigs in postnatal TN conditions, exposure
467 to prenatal HS can increase body temperature attributed to a possible alteration in the HPA axis
468 and in thyroid functions. Depending on the timing and duration, prenatal HS can also increase
469 carcass adiposity but this is not proven for TN-raised pigs exposed to a whole-gestation prenatal
470 HS.

471 ***Effects of prenatal HS on growth performance and metabolism in growing pigs subjected to***
472 ***postnatal HS***

473 Despite the increasing literature in prenatal HS in pigs, there is still little known on the
474 effect of prenatal heat stress on ability of pigs to cope with postnatal HS. In our study, prenatal HS
475 tended to reduce plasma SOD during acute postnatal HS. This result is in agreement with previous

476 studies (Lista et al., 2010; Yin et al., 2018) which suggests a decreased anti-oxidative capacity
477 previously reported in offspring of stressed mammals. This decreased oxidative stress tolerance
478 and other prenatal HS effects discussed so far can influence the pig's postnatal life and production
479 efficiency. Indeed, prenatal HS seemed to aggravate production performance of GHS pigs in our
480 study as GHS-PHS pigs had slightly lower ADG and final BW than GHS-PTN pigs even with
481 similar overall ADFI. Under postnatal HS, both GHS groups still had higher rectal temperature
482 than GTN-PTN pigs even after 53 d of chronic HS suggesting they were unable to dissipate all
483 heat necessary to lower body temperature to a similar level with pigs kept in pre- and postnatal
484 thermoneutrality. In the previous section, the decreased ability of pigs to dissipate heat as
485 consequence of prenatal HS was discussed. At 28 to 34°C, pigs in our study may have already
486 reached the maximum heat dissipation level through the skin as GHS-PHS and GHS-PTN had
487 similar skin temperature and temperature gradient. With a reduced anti-oxidative capacity, one
488 could hypothesize a decreased heat tolerance in PHS pigs as they were subjected to postnatal HS
489 wherein heat dissipation has already been maximized.

490 In the present study, the GHS-PHS pigs had lower carcass lean meat content than GHS-
491 PTN pigs and tented to exhibit higher fatness at both subcutaneous and internal levels. Reasons
492 for this decrease in leanness are still unclear but one theory could be related to the hypothesized
493 fetal growth restriction which, in other mammals, is linked to impaired glucose and insulin
494 metabolism (Thureen et al., 1992; Lesage et al., 2004). Indeed, Johnson et al. (2015b) also reported
495 that insulin per unit of glucose or per kg of FI was higher in in-utero HS pigs but only if raised in
496 postnatal HS environment. Boddicker et al. (2014) reported that it is the exposure to prenatal HS
497 during the first half of the gestation that increases circulation insulin levels regardless of the

498 environment during the second half of gestation. A whole-gestation prenatal HS has also been
499 reported to increase postnatal stress response of pigs (Merlot et al., 2018) possibly linked to the
500 hypothesized altered HPA set point. Hypersecretion of chronic stress hormones in humans is
501 attributed to increased visceral fat accumulation and muscle loss (Pervanidou and Chrousos, 2012)
502 and could be related to the increased visceral adiposity of pigs subjected to both prenatal and
503 postnatal HS in our study.

504 With effects such as heightened postnatal stress responses, altered thyroid functions,
505 decreased heat dissipation ability, and reduced anti-oxidative capacity, chronic prenatal HS seems
506 to impair long-term heat tolerance of pigs in our study, as indicated by their reduced growth,
507 increased body adiposity, and overall decreased productive efficiency. However, it must be noted
508 that the present study considered only a chronic whole gestation HS effect. Existing literature in
509 pigs and other mammals suggests that impacts on pig performance and carcass composition could
510 differ depending on the timing, duration, and intensity of the prenatal HS applied, and this still
511 remains to be further investigated.

512 **CONCLUSION**

513 Studies about prenatal heat stress on growing pigs are still limited. Chronic prenatal heat
514 stress in our study decreased heat tolerance of growing pigs due to increased body temperature,
515 and altered thyroid functions and physiological stress responses. With the increasing impact of
516 climate change, this implies a decreased global swine production efficiency. Existing literature and
517 present data suggests that multiple factors can influence prenatal heat stress effects such as duration
518 and timing during gestation, postnatal thermal environment, and physiological stage of pigs. More
519 studies are needed to further elucidate its effects and biological mechanism in pigs.

## CONFLICT OF INTEREST STATEMENT

The authors declare that there is no conflict of interest

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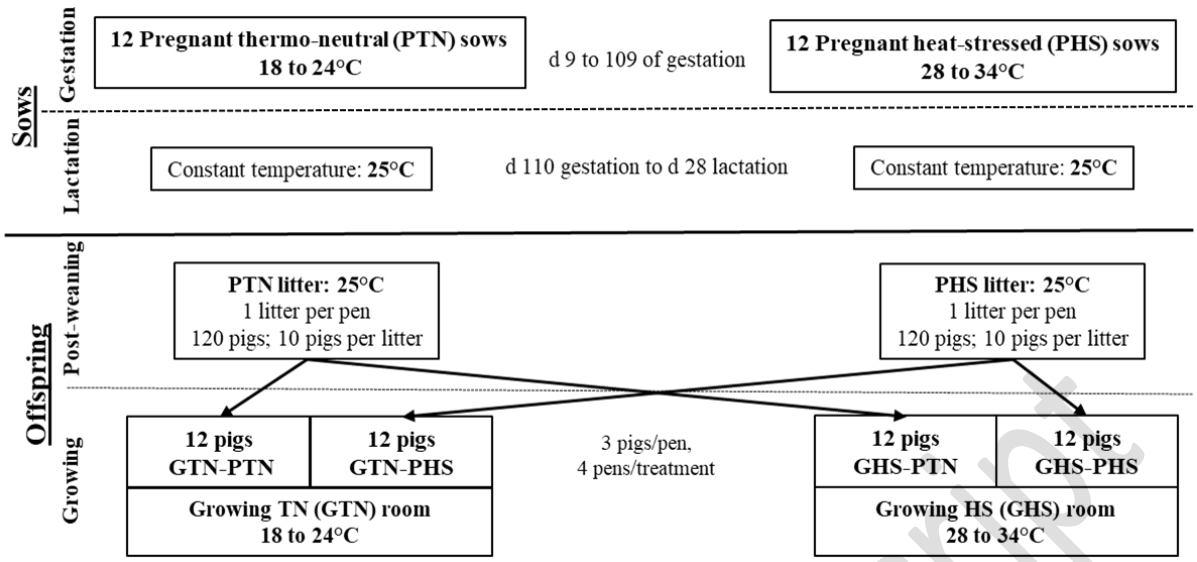


**Figure 1. Framework of the experimental study from gestation to growing period.**

Pregnant sows (12 primiparous and 12 multiparous) were housed under either thermoneutral (PTN) or heat-stressed (PHS) conditions from 9 d to 109 d of gestation. Their female offspring were subjected to thermoneutral (GTN) or heat-stressed (GHS) conditions from 82 to 140 d of age. Sows during lactation and piglets during post-weaning were housed under thermoneutral conditions.

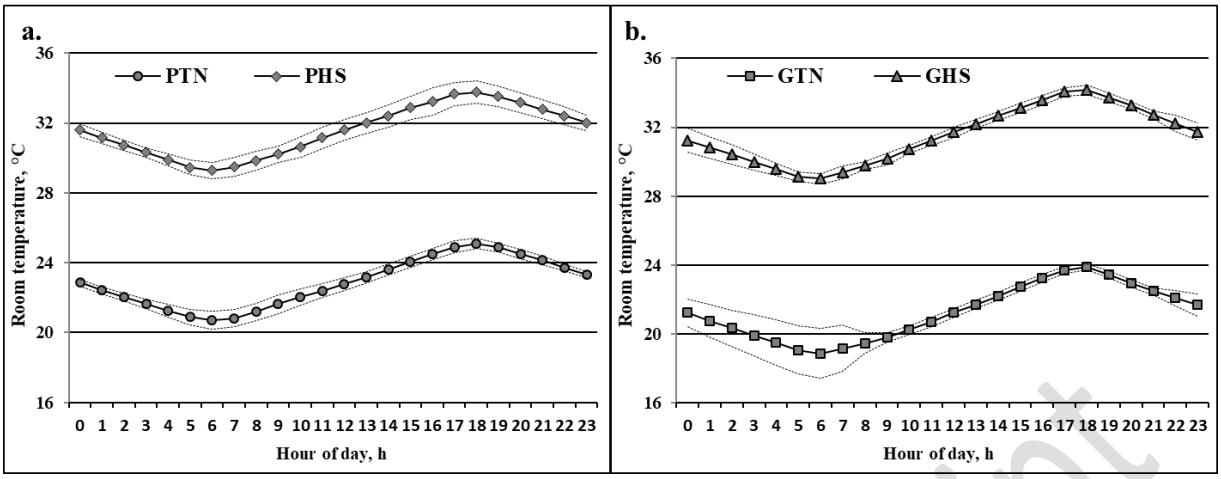


**Figure 2.** Actual average hourly ambient temperature (mean±SE) of the gestation rooms (2a) from d 9 to 109 of gestation and of the growing rooms (2b) from 83 to 140 d of age during the growth period. PTN: prenatal thermoneutral; PHS: prenatal heat-stressed; GTN: growing thermoneutral; GHS: growing heat-stressed.

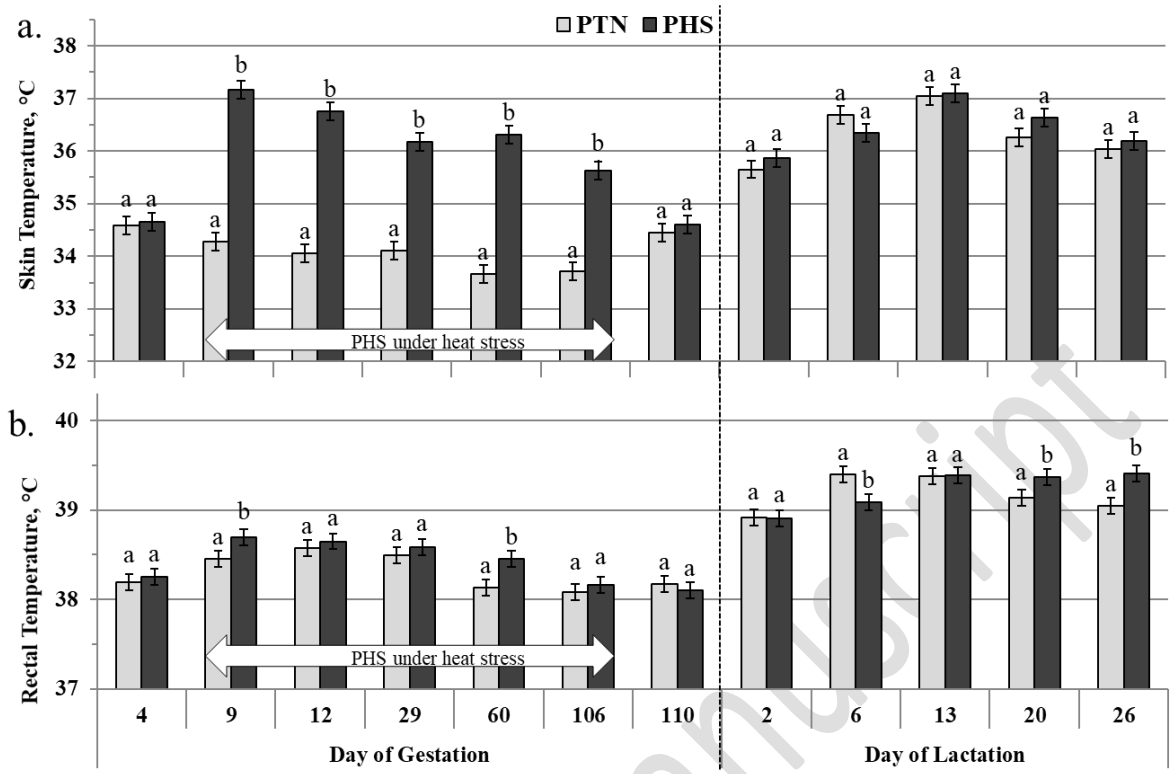


**Figure 3.** Effect of the climatic environment on the skin (3a) and rectal (3b) temperatures of mixed parity sows from gestation to lactation (LSmeans ± SEM). From d 9 to 109 of pregnancy, sows (n=24) were subjected to one of two environments: thermoneutral (PTN; 18 to 24°C) and heat-stressed (PHS; 28 to 34°C). During the whole lactation period, sows were kept at 25°C. [a,b]Within each day, LSmeans with different superscript letters differ according to the experimental group ($P$<0.05).

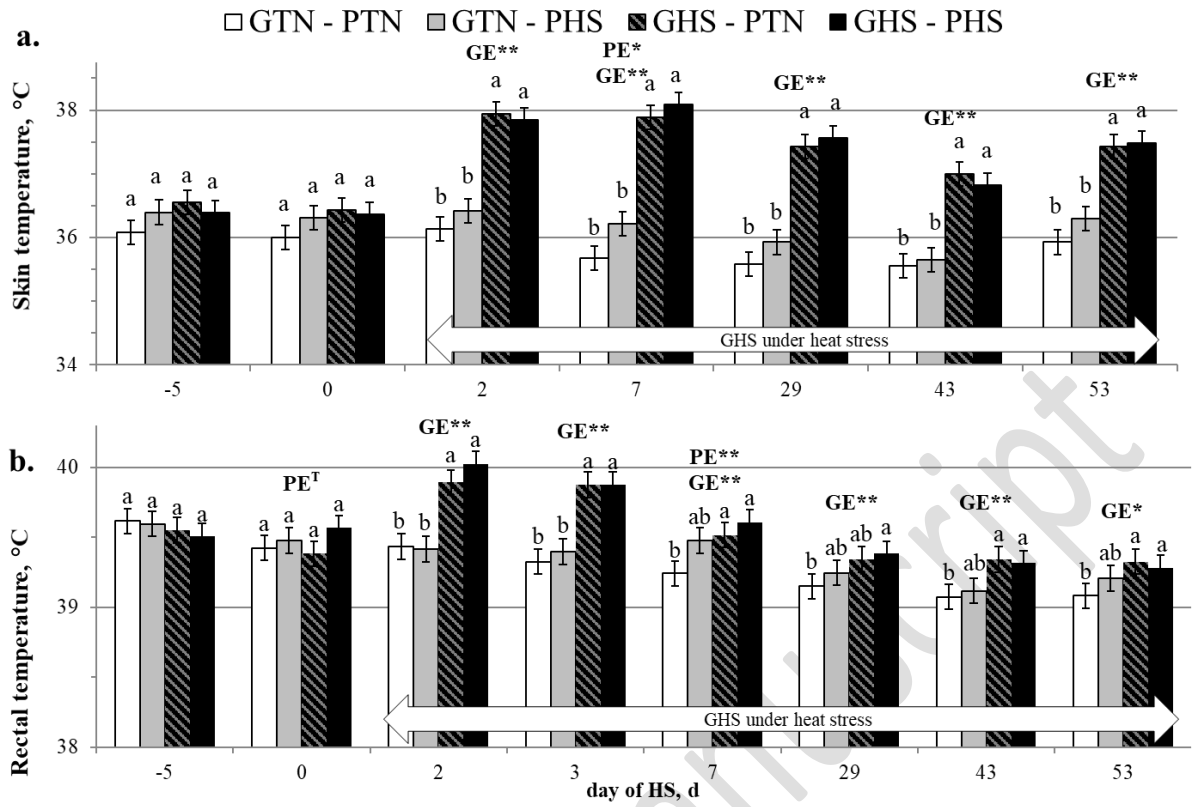


**Figure 4.** Effect of climatic environment (thermoneutral [TN] or heat-stressed [HS]) during prenatal development (PE; PTN vs. PHS) and during growing (GE; GTN vs. GHS) on the skin (5a) and rectal (5b) temperatures of growing pigs (LSMeans±SEM). [a,b]Within each day, LSmeans with different superscript letters differ according to the experimental group ($P < 0.05$). PE = effect of the prenatal environment, regardless of the growing environment. GE = effect of the growing environment regardless of the prenatal environment. *$P$<0.05, **$P$<0.01.

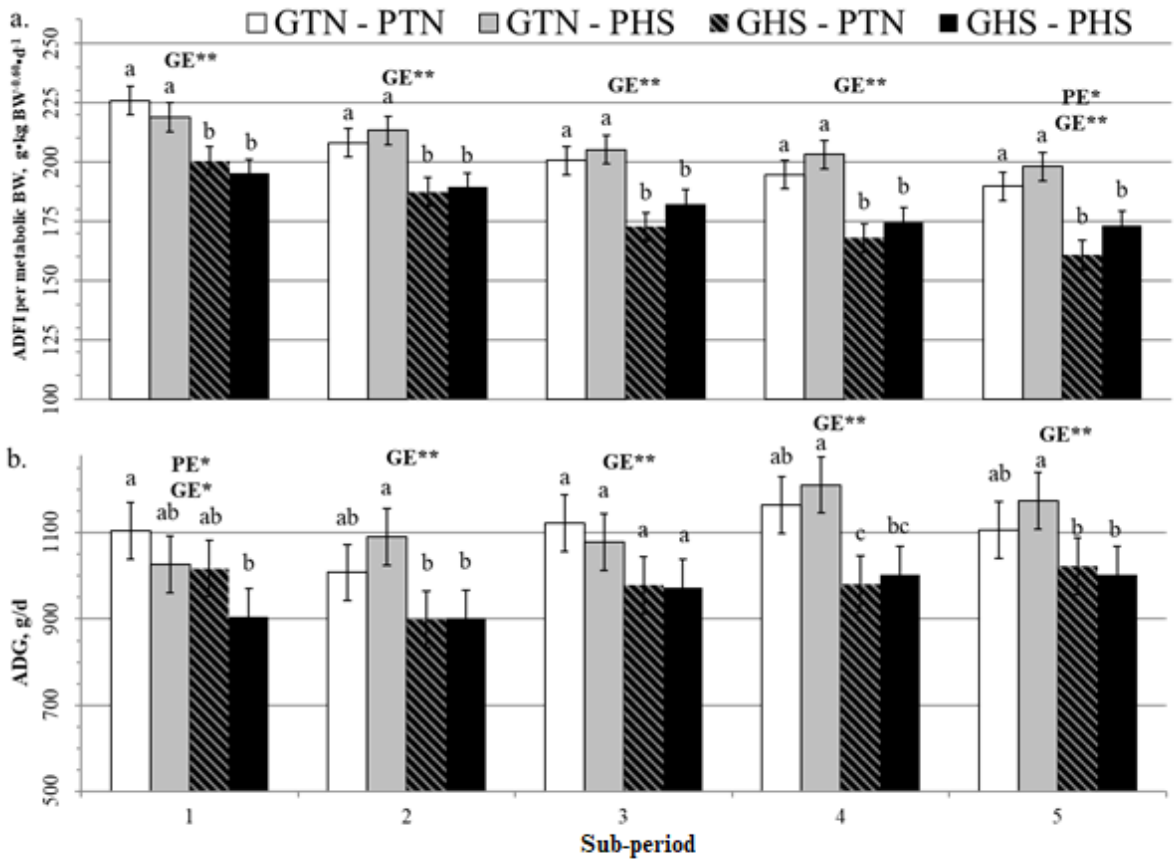


**Figure 5.** Effect of climatic environment (thermoneutral [TN] or heat-stressed [HS]) during prenatal development (PE; PTN vs. PHS) and during growing (GE; GTN vs. GHS) on ADFI per metabolic BW (4a) and ADG (4b) in growing pigs using TN performance as covariate (LSMeans±SEM). Sub-period 1: d 0 to 6 of GHS; Sub-period 2: d 7 to 15; Sub-period 3: d 16 to 29; Sub-period 4: d 30 to 43; and Sub-period 5: d 44 to 58. [a,b,c]Within each sub-period, LSmeans with different superscript letters differ according to the experimental group ($P$<0.05). PE = effect of the prenatal environment, regardless of the growing environment. GE = effect of the growing environment regardless of the prenatal environment. *$P$<0.05, **$P$<0.01.

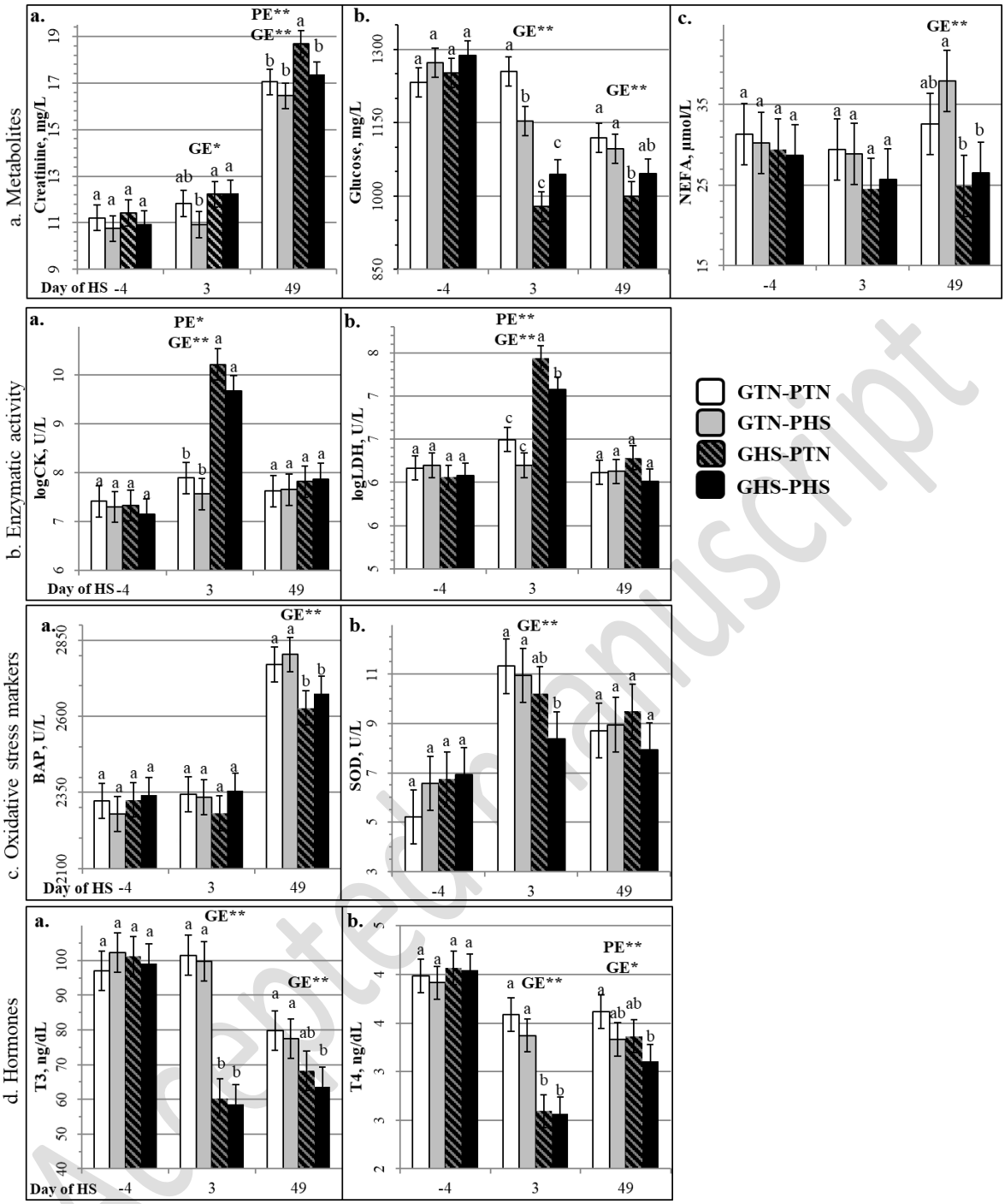


**Figure 6.** Effect of climatic environment (thermoneutral [TN] or heat-stressed [HS]) during prenatal development (PE; PTN vs. PHS) and during growing (GE; GTN vs. GHS) on plasma parameters of growing pigs (LSMeans±SEM). [a,b]Within each day, LSmeans with different superscript letters differ according to the experimental group ($P<0.05$). PE = effect of the prenatal environment, regardless of the growing environment. GE = effect of the growing environment regardless of the prenatal environment. *$P<0.05$, **$P<0.01$.

Table 1. Effect of the prenatal and postnatal (growing) climatic environment on the growth performance in growing pigs.[1]

| Items | GTN-PTN | GTN-PHS | GHS-PTN | GHS-PHS | RSD[2] | Statistics[3] |
|---|---|---|---|---|---|---|
| Live BW, kg | | | | | | |
| 75 d | 35.8 | 36.1 | 36.6 | 35.2 | 1.69 | GE**, P**, S* PE×GE$^{T}$, GE×P** |
| 82 d | 42.3 | 42.6 | 43.7 | 41.6 | | |
| 140 d | 104.4$^{a}$ | 105.8$^{a}$ | 98.8$^{b}$ | 95.6$^{b}$ | | |
| ADG, g · d$^{-1}$ | | | | | | |
| 75 to 81 d | 859 | 888 | 997 | 943 | 70 | P**, GE×P** |
| 82 to 140 d | 1,147 | 1,114 | 1,017 | 989 | | |
| 82 to 140 d† | 1,110$^{a}$ | 1,129$^{a}$ | 983$^{b}$ | 967$^{b}$ | 52 | GE** |
| ADFI, g · d$^{-1}$ | | | | | | |
| 75 to 81 d | 1,729 | 1,884 | 1,996 | 1,901 | 168 | P**, GE×P** |
| 82 to 140 d | 2,635 | 2,683 | 2,347 | 2,307 | | |
| 82 to 140 d† | 2,602$^{a}$ | 2,680$^{a}$ | 2,215$^{b}$ | 2,255$^{b}$ | 84 | GE**, S* |
| ADFI per metabolic BW, g· kg$^{-0.60}$·d$^{-1}$ | | | | | | |
| 75 to 81 d | 198 | 208 | 218 | 210 | 11 | P**, GE×P* |
| 82 to 140 d | 196 | 202 | 180 | 181 | | |
| 82 to 140 d† | 198$^{a}$ | 202$^{a}$ | 172$^{b}$ | 178$^{b}$ | 6 | PE$^{T}$, GE**, S* |
| FCR | | | | | | |
| 75 to 81 d | 2.02 | 2.12 | 2.01 | 2.02 | 0.09 | P** |
| 82 to 140 d | 2.30 | 2.41 | 2.31 | 2.33 | | |
| 82 to 140 d† | 2.34 | 2.37 | 2.29 | 2.31 | 0.08 | Ns |

[1]A total of 48 female pigs (housed 3 pigs per pen) were distributed to a 2 x 2 factorial design based on their prenatal environment (PE) and their growing environment (GE): TN – thermoneutral (18 to 24˚C), HS = heat-stressed (28 to 34˚C). First main period: Pigs were maintained in TN conditions from 75 to 81 d of age. Second main period: The GTN room was maintained in TN conditions until 140 d of age; in GHS room, temperature transition started at 82 d of age and full-blown thermal challenge was from 84 to 140 d of age.

[2] Residual standard deviation.

[3]The pen was considered as the experimental unit. Data were analyzed using PROC MIXED model with prenatal environment (PE), growing environment (GE), period (P), their interactions, and sire (S) as fixed effects. LSmeans with different superscript letters differ according to the experimental group $^{T}$ $P < 0.10$, $^{*}P < 0.05$, $^{**}P < 0.01$.

†Adjusted performance based on the average value measured during first main period (944 g/d, 1915g/d, 212 g•kg BW$^{-0.60}$•d$^{-1}$, and 2.03 for ADG, ADFI, ADFI per metabolic BW, and FCR, respectively).

Table 2. Effect of the prenatal and postnatal (growing) climatic environments on the carcass and organ traits of growing pigs.[1]

| Items | GTN-PTN | GTN-PHS | GHS-PTN | GHS-PHS | RSD[2] | Statistics[3] |
|---|---|---|---|---|---|---|
| No. of pigs | 12 | 12 | 12 | 12 | | |
| Slaughter BW (sBW), kg | 102.3$^{ab}$ | 103.6$^{a}$ | 96.8$^{bc}$ | 93.7$^{c}$ | 5.6 | GE** |
| Hot carcass weight, kg | 80.8$^{a}$ | 81.7$^{a}$ | 77.0$^{ab}$ | 74.2$^{b}$ | 4.5 | GE** |
| Carcass dressing, % | 79.1 | 78.8 | 79.6 | 79.2 | 0.9 | GE$^{T}$ |
| Carcass length, cm | 95.7$^{ab}$ | 97.4$^{a}$ | 94.9$^{bc}$ | 93.4$^{c}$ | 1.9 | GE**, PE×GE** |
| Head, % sBW | 4.47$^{ab}$ | 4.31$^{b}$ | 4.59$^{a}$ | 4.49$^{ab}$ | 0.24 | PE$^{T}$, GE* |
| Carcass composition[4] | | | | | | |
| Lean meat, % | 63.5$^{a}$ | 63.4$^{a}$ | 63.2$^{a}$ | 61.8$^{b}$ | 1.2 | PE*, GE*, PE×GE$^{T}$, sBW** |
| Average BFT, mm | 18.7$^{bc}$ | 17.0$^{c}$ | 19.3$^{ab}$ | 20.9$^{a}$ | 1.7 | GE**, PE×GE**, sBW** |
| Perirenal fat, g/kg sBW | 7.6$^{ab}$ | 7.0$^{b}$ | 7.6$^{ab}$ | 9.2$^{a}$ | 1.5 | PE×GE$^{*}$ |
| Carcass cuts[4], % cold carcass wt | | | | | | |
| Loin | 29.0$^{a}$ | 29.2$^{a}$ | 27.8$^{b}$ | 27.9$^{b}$ | 0.9 | GE** |
| Ham | 26.8$^{bc}$ | 26.3$^{c}$ | 27.5$^{a}$ | 27.1$^{ab}$ | 0.6 | PE*, GE** |
| Belly | 12.1 | 12.0 | 12.2 | 12.5 | 0.6 | sBW** |
| Backfat | 4.9$^{ab}$ | 4.6$^{b}$ | 4.9$^{ab}$ | 5.4$^{a}$ | 0.6 | GE$^{T}$, PE×GE*, sBW** |
| Organ wt., mg/kg BW | | | | | | |
| Hypothalamus | 11.2$^{a}$ | 9.8$^{b}$ | 10.1$^{ab}$ | 10.8$^{ab}$ | 1.5 | PE×GE* |
| Pituitary gland | 2.8$^{ab}$ | 2.7$^{b}$ | 2.7$^{b}$ | 3.0$^{a}$ | 0.3 | PE×GE* |

[1]A total of 48 female pigs (housed 3 pigs per pen) were distributed to a 2 x 2 factorial design based on their prenatal environment (PE) and their growing environment (GE): TN – thermoneutral (18 to 24˚C), HS = heat-stressed (28 to 34˚C).
[2] Residual standard deviation.
[3]The pig was considered as the experimental unit. Data were analyzed using PROC MIXED model with prenatal environment (PE), growing environment (GE), their interaction, pen, and sire as fixed effects. $^{T}P < 0.10$, $^{*}P < 0.05$, $^{**}P < 0.01$
[4]Slaughter BW (sBW) was used as covariate (adjusted slaughter BW = 99.1 kg).