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Effect of temperature on thermal acclimation in growing pigs estimated using a nonlinear function

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ABSTRACT: Ninety-six Large White growing barrows were used to determine the effect of temperature on thermoregulatory responses during acclimation to increased ambient temperature. Pigs were exposed to 24°C for 10 d and thereafter to a constant temperature of 24, 28, 32, or 36°C for 20 d. The study was conducted in a climate-controlled room at the INRA experimental facilities in Guadeloupe, French West Indies. Relative humidity was kept constant at 80% throughout the experimental period. Rectal temperature, cutaneous temperature, and respiratory rate were measured [breaths per minute (bpm)] 3 times daily (0700, 1200, and 1800 h) every 2 or 3 d during the experiment. The thermal circulation index (TCI) was determined from rectal, cutaneous, and ambient temperature measurements. Changes in rectal temperature, respiratory rate, TCI, and ADFI over the duration of exposure to hot temperatures were modeled using nonlinear responses curves. Within 1 h of exposure to increased temperature, rectal temperature and respiratory rate increased by 0.46°C/d and +29.3 bpm/d, respectively, and ADFI and TCI decreased linearly by 44.7 g·d−2·kg−0.60 and 1.32°C/d, respectively until a first breakpoint time (td1). This point marked the end of the short-term heat acclimation phase and the beginning of the long-term heat acclimation period. The td1 value for ADFI was greater at 28°C than at 32 and 36°C (2.33 vs. 0.31 and 0.26 d, respectively, P < 0.05), whereas td1 for the TCI increase was greater at 36°C than at 28 and 32°C (1.02 vs. 0.78 and 0.67 d, respectively; P < 0.05). For rectal temperature and respiratory rate responses, td1 was not influenced by temperature (P > 0.05) and averaged 1.1 and 0.89 d, respectively. For respiratory rate and rectal temperature, the long-term heat acclimation period was divided in 2 phases, with a rapid decline for both variables followed by a slight decrease (P < 0.05). These 2 phases were separated by a second threshold day (td2). For rectal temperature, td2 increased significantly with temperature (1.60 vs. 5.16 d from 28 to 36°C; P < 0.05). After td2, the decline in rectal temperature during the exposure to thermal challenge was not influenced by temperature, suggesting that the magnitude of heat stress would affect thermoregulatory responses only at the beginning of the long-term heat acclimation period. The inclusion of random effects in the nonlinear model showed that whatever the temperature considered, interindividual variability of thermoregulatory responses would exist.

Key words: acclimation, heat stress, nonlinear mixed model, swine, temperature

INTRODUCTION

Heat stress is an important factor contributing to production losses in the swine industry (St-Pierre et al., 2003). Although heat stress is an occasional challenge only during summer in temperate countries, it is a constant problem in many tropical areas. In these regions, the negative effects of thermal stress can be accentuated by a high relative humidity (RH). Above the upper limit of the thermal neutral zone (i.e., 24 to 25°C in growing pigs; Quiniou et al., 2001), feed consumption is reduced to limit the metabolic heat production (HP; Le Dividich et al., 1998). This decreased feed intake results in a reduction in growth performance.

A better understanding of the acclimation of pigs to thermal environments is a key factor in improving thermotolerance without adversely affecting production, especially in the context of genetic selection of heat stress-resistant genotypes. There is evidence for a biphasic pattern of heat acclimation divided in 2 pe-
periods based on time. The short-term heat acclimation (STHA) phase is characterized by rapid physiological adaptations to cope with heat stress before more permanent adjustments can be made. When all changes made during STHA are completed, the long-term heat acclimation (LTHA) begins (Horowitz, 2001). In pigs, rectal temperature (RT) increases within 24 h of exposure and declines steadily thereafter (Morrison and Mount, 1971; Renaudeau et al., 2007). In most of these studies, only 1 high ambient temperature has been tested. In practice, the physiological responses of pigs to a continuous and prolonged heat stress will vary with the intensity of the heat challenge.

The aim of the present study was to investigate the effect of the intensity of heat stress on performance and thermal acclimation in growing pigs. Results on growth performance and metabolism have been reported previously (Renaudeau et al., 2008). The present study focuses on the effect of temperature on the thermal acclimation responses.

**MATERIALS AND METHODS**

Care and use of animals were performed according to the certificate of authorization for experiments involving living animals (issued by the French Ministry of Agriculture).

**Experimental Design and Animal Management**

The effect of temperature (24, 28, 32, and 36°C) was tested on physiological thermal responses in 96 Large White barrows, which were used in an experiment with 8 successive replicates (2 replicates/treatment), conducted at the experimental facilities of INRA in Guadeloupe, French West Indies. Within each replicate, 12 pigs were randomly selected at 11 wk of age (37.9 ± 4.0 kg of BW) and moved to a climate-controlled room. A total of 7 sire origins were used in this experiment. Pigs were adapted to experimental conditions (housing, diet) for 10 d. During this period, ambient temperature was maintained at 24°C. The experimental period was divided in 2 phases: pigs were kept at 24°C for 10 d (P0), and were kept thereafter at a constant temperature of 24, 28, 32 or 36°C for 20 d (P1; Figure 1). Between P0 and P1, the temperature was changed on day 0 from 24°C to the experimental temperature at a constant rate of 2°C/h beginning at 0900 h. The RH was kept constant at 80% over the total duration of the experimental period. Pigs were offered, for ad libitum intake, a diet that was formulated with corn, soybean meal, and wheat middlings to meet the NRC (1998) requirement for growing pigs (16.0% CP, 11.0 MJ of NE/kg). Pigs were individually housed in an 800-m³ climate-controlled room equipped with 12 metal slatted pens (1.50 × 0.85 m). Each pen was equipped with a feed dispenser and a nipple drinker designed to avoid water spillage. In the climate-controlled room, ambient temperature and RH were maintained within ±0.2°C and ±3%, respectively. The photoperiod was fixed at 12.5 h of artificial light (from 0600 to 1830 h), and the ventilation rate was set at 50 m³/h per pig. Air speed was not controlled, but periodical spot measurements at the level of the animal indicated that it did not exceed 0.15 m/s.

**Measurements**

Every morning, feed refusals were manually collected between 0700 and 0800 h, weighed, and subsampled for DM determination. Subsamples of feed offered to the
animals were collected weekly for DM determination, and subsamples were pooled at the end of each replicate for chemical analysis.

Rectal and cutaneous (CT) body temperatures and respiration rate (RR) were measured 3 times daily (0700, 1200, 1800 h) on d −10, −7, −5, −3, −1, 0, 1, 2, 4, 7, 9, 11, 14, 16, 18, and 20 of experiment. For each recording period, the following protocol was applied: first, RR rate was interpreted as breaths per minute (bpm) and determined by counting flank movements in resting animals only for a period of 1 min. Variation in RR under heat stress conditions is considered a good indicator of the latent heat loss (Kamada and Notsuki, 1987). After RR measurements were completed, RT was measured using a digital thermometer (Microlife Corporation, Paris, France). Cutaneous body temperature was measured on the backs and flanks by using a digital thermometer (HH-21 model, Omega, Stamford, CT) with a K probe. Variation in CT under heat stress conditions is an indicator of increased blood flow to the skin (Mount, 1975).

Calculations and Statistical Analysis

Cutaneous body temperature was calculated as the average of CT measurements on the back and flank locations. Because CT is affected by both internal and ambient temperature, it was difficult to analyze the significance of a change in CT without taking into account these other values. According to Curtis (1983), the 3 temperatures can be incorporated into a single index, namely, the thermal circulation index (TCI). The TCI is used as an indicator of blood and heat transfer to a particular area of skin under steady-state thermal conditions. It can be calculated from the core-to-skin and skin-to-environment temperature gradients with the following formula:

\[
TCI = \frac{(CT - Ta)}{(RT - CT)},
\]

where Ta is the mean actual ambient temperature. For all the criteria studied, data were averaged by pig on a daily basis.

According to Morrison and Mount (1971) and Renaudeau et al. (2007), the thermoregulatory response in pigs has a biphasic profile consisting of an initial hyperthermia within the first 24 h of exposure to heat stress and a subsequent recovery period characterized by a gradual decrease in body temperature. To make a clear distinction between changes in ADFI, RT, TCI, or RR during the STHA and LTHA periods, we investigated a model adapted from Koops and Grossman (1991) with 2 “threshold days” (i.e., days that marked the beginning or intermediate phases of the acclimation response):

\[
Y_{ij} = y_0 + v_1(d_{ij} - td_1) + \ln(1 + \exp((d_{ij} - td_1)/r_1)) - r_2(v_{2i} - v_{3i}) \ln(1 + \exp((d_{ij} - td_2)/r_2)) + \varepsilon_{ij},
\]

where Y is the response variable (g·d\(^{-1}\)·kg\(^{-0.60}\), °C, or bpm) between d −1 and 20; i is 1 to n pigs, j is 1 to 4 temperature levels; \(y_0\) (g·d\(^{-1}\)·kg\(^{-0.60}\), °C, or bpm) is the value of Y at d 0; d is the day of exposure to the experimental temperature; \(td_1\) and \(td_2\) (day of exposure) are the threshold days; and \(v_1\), \(v_2\), and \(v_3\) (g·d\(^{-2}\)·kg\(^{-0.60}\), °C/d, or bpm/d) are the linear variations of Y before and after \(td_1\) and after \(td_2\), respectively (Figure 2). In the approach of Koops and Grossman (1991), \(r_1\) and \(r_2\) determine the smoothness of the transition around \(td_1\) and \(td_2\), respectively. In the present study, \(r_1\) and \(r_2\) were determined for each studied variable with the assumption that it was not influenced by temperature.

The components of each parameter of the model were modeled as

\[
\begin{align*}
y_0i &= y_0 + y_{0T} + \alpha_i, \\
v_1i &= v_1 + v_{1T} + \beta_i, \\
v_2i &= v_2 + v_{2T} + \gamma_i, \\
v_3i &= v_2 + v_{2T} + \delta_i, \\
td_1i &= td + tdT + \zeta_i, \text{ and } \\
td_2i &= td + tdT + \eta_i,
\end{align*}
\]

where \(y_0\) is the mean asymptote, \(y_{0T}\) is the effect of temperature on the asymptote, and \(\alpha_i\) is the random effect of the asymptote of the ith individual pig. A similar description could be given for \(v_{1i}, v_{2i}, v_{3i}, td_{1i}\), and \(td_{2i}\). We assumed that the 6 random effects (\(\alpha_i, \beta_i, \gamma_i, \delta_i, \zeta_i, \eta_i\)) were distributed normally and were independent. These random effects reflect how much the subject-specific profile deviates from the overall average profile. The nonlinear mixed model (NLMM) was fitted using the NLMIXED procedure (SAS Inst. Inc., Cary, NC). In a previous analysis, the model was first fitted without any random effects and thereafter was fitted with 6 random effects. Because NLMIXED does not provide adjusted \(R^2\) values, we used the following formula for its estimation (Robbins et al., 2006):

\[
\text{adjusted } R^2 = 1 - \frac{\text{SSE}/(n - p - q - 1)}{\text{CTSS}/(n - 1)},
\]

where SSE is the sum of squares for error (calculated from the estimation of residual values), CTSS is the corrected total sum of squares, n is the number of observations, p is the number of parameters, and q is the number of random effects. Correlations within individual random effect estimates adjusted for the fixed effects of temperature and replicate, and between random effect estimates and residual estimates of ADFI or ADG measured during P0 (ADFI\(_0\) and ADG\(_0\)) were calculated using the CORR procedure of SAS.
A total of 10 pigs were removed from the experiment because of leg problems (n = 3), rectal prolapse (n = 4), or diarrhea (n = 3). In addition, all the data obtained from pigs kept at 24°C during P1 were removed from the database for the modeling approach because of a lack of change in physiological measures between d 0 and 20. In all, data for only 63 pigs, with 12 successive recordings each, were used in the data analysis.

The estimates of the curve coefficients for ADFI, RT, TCI, and RR as affected by temperature are given in Table 1. The individual fitted acclimation responses for all temperatures are shown in Figure 3.

Immediately after the change between 24°C and the experimental temperature, RT increased (P < 0.05) until the time point in acclimation when the maximal RT occurred (i.e., td1; this first threshold day marked the end of the STHA; Figure 2). The v1 value increased with the ambient temperature (0.29 vs. 0.66°C/d from 28 to 36°C; P < 0.05), whereas td1 remained unaffected by temperature (1.10 d on average; P > 0.05). According to our results, the LTHA was divided in 2 phases, with a rapid decline (P < 0.05) in RT between td1 and td2 (v2) and thereafter a slight but significant (P < 0.05) change in RT (v3; Figures 3 and 4). The v2 and v3 estimates for RT were not influenced by temperature (−0.18 and −0.012°C/d, respectively; P > 0.05), but td2 increased significantly (P < 0.05) from 1.60 to 5.16 d between 28 and 36°C; an intermediate value was reported at 32°C (i.e., 2.98 d). The variance in random effects associated with the v1, v3, and td1 parameter esti-
The decrease in feed consumption during STHA increased gradually with ambient temperature, from -20.2 to -69.6 g·d⁻¹·kg⁻⁰.₆⁰ between 28 and 36°C (P < 0.05). The td₁ estimates for ADFI at 32 and 36°C were not different from zero (P > 0.10). The TCI ended 2.33 d later at 28°C, rather than at 32 and 36°C. After td₁, the increase in ADFI was less (P < 0.05) as the temperature increased (9.25, 3.07, and 1.07 g·d⁻¹·kg⁻⁰.₆⁰ at 28, 32, and 36°C, respectively). The time point in the acclimation response, which marked the second phase of the LTHA (i.e., after td₂), was greater at 32 than at 28°C (11.55 vs. 5.93 d on average at 28 and 36°C) (Figure 4). During the STHA period, the magnitude of decrease in TCI was greater as the temperature increased (9.25, 3.07, and 1.07 g·d⁻¹·kg⁻⁰.₆⁰ at 28, 32, and 36°C, respectively). The time point in the acclimation response, which marked the second phase of the LTHA (i.e., after td₂), was greater at 32 than at 28°C (11.55 vs. 5.93 d; P < 0.001), but the increase in ADFI after this threshold day was not different from zero regardless of the temperature considered. The random effects associated with the y₀, v₁, and v₂ parameter estimates were significantly different from zero (P < 0.05), and σ²ᵣᵣ is the individual variance for each parameter within the studied population [an asterisk (*) indicates σ²ᵣᵣ different from zero, P < 0.05], and σ²ₑ is the residual variance of the model.
32°C; \( P < 0.05 \)). Irrespective of the ambient temperature, the increase in TCI after \( t_d_1 \) was not significantly different from zero.

Residual correlation coefficients within random estimates of parameters obtained with the NLMM showed positive relationships between the \( v_2 \) value for TCI and the \( v_1 \) or \( v_2 \) value for ADFI (\( r = 0.33 \) and \( 0.25 \), respectively; \( P < 0.05 \)). The ADFI changes before and after \( t_d_1 \) were inversely related (\( r = -0.25; \ P < 0.05 \)). A positive relationship between \( t_d_1 \) for ADFI and a decline in RT after \( t_d_1 \) were found (\( r = 0.28; \ P = 0.03 \)). The ADFI during P0 was positively correlated with the \( v_1 \) value for ADFI or the \( v_2 \) value for RT (\( r = 0.43 \) and \( 0.30 \), respectively; \( P < 0.05 \)). A positive correlation was reported between ADG recorded during P0 (ADG\(_0\)) and the \( v_1 \) value for ADFI (\( r = 0.27; \ P < 0.05 \)).

Figure 3. Observed and predicted of individual profiles of ADFI (g·d\(^{-1}\)·kg\(^{-0.60}\); panel A) and rectal temperature (°C; panel B) over the acclimation period at the experimental temperature. Each symbol (circles, 28°C; squares, 32°C; and triangles, 36°C) represents the observed data, and continuous lines are the predicted responses.
DISCUSSION

In the present study, the use of a nonlinear function allowed us to model the thermoregulatory responses with biologically meaningful variables and to compare the effect of temperature on these parameter estimates (Renaudeau et al., 2007). According to previous calculations (D. Renaudeau, unpublished data), the NLMM increased the accuracy of prediction of the thermal acclimation models when compared with a traditional nonlinear model (NLM). For example, the goodness of fit between actual and predicted RT from the model increased from 0.56 to 0.78 when random effects were added in the NLMM. The residual variance in the NLMM was reduced by approximately 73% compared with the NLM (0.03 vs. 0.11°C²). According to these data and Akaike and Bayesian information criteria (for RT analysis, Akaike information criterion values were 510 and −104 and Bayesian information criterion values were 512 and −102 for NLM and NLMM, respectively), it was concluded that an NLMM fit better than an NLM, mainly because the variance-covariance partitioning associated with the random effects allows for the separation of between-pig variation \( \left( \sigma^2_{\alpha} + \sigma^2_{\beta} + \sigma^2_{\gamma} + \sigma^2_{\delta} + \sigma^2_{\zeta} + \sigma^2_{\eta} \right) \) from within-pig variation \( \sigma^2_{\varepsilon} \). In consequence, each variable estimated can be represented by the fixed effect, with a mean value of each variable for each temperature, as well as the random effect, which refers to differences between the value of a variable fitted for each individual pig and the mean value of the variable.

Mean Thermal Acclimation Responses in Pigs

In contrast to other farm species (ruminants or poultry), the acclimation responses to increased ambient temperature are poorly documented in pigs. According to Horowitz (2001), the thermoregulatory response during acclimation to heat stress is biphasic, with an “inefficient” phase (STHA) followed by an “efficient” state (LTHA) after homeostasis has been reached in acclimation. In the present study, RT was considered to be the most meaningful single criterion for judging the heat tolerance of the animal because this criterion indicates the efficiency of the animal in maintaining homeothermy during thermal stress. The RT variation measured in the present study (i.e., a strong increase within 24 h of exposure, followed by a gradual decline with successive days of exposure) was described previously in pigs (Morrison and Mount, 1971; Giles et al., 1991; Collin et al., 2002; Renaudeau et al., 2006). During the STHA phase, the TCI was reduced, which emphasizes the inability of the pig to lose heat by the sensible pathway under heat stress. Thus, evaporative heat loss accounts for practically all the total heat loss within the first hour of exposure to increased temperature. In the present experiment, regardless of temperature, an increase in RR was measured within 24 h of exposure to heat stress. In contrast to ruminant species, pigs cannot sweat; thus, they rely mostly on respiratory evaporation to lose heat via the latent way (Renaudeau et al., 2006). During STHA, the ADFI decreased in response to increased temperature. This reduction was inter-
produced feed intake on viscera mass. However, mechanisms for increasing heat loss and decreasing HP could not compensate fully for the excessive heat load, which explains the strong increase in RT within the first hour of exposure to increased ambient temperature.

After td₁, the LTHA was described as a gradual reduction in RT over the time of exposure to increased temperature. According to the present study, this response was curvilinear, with a strong decrease from td₁ to td₂, followed by gradual variation. In contrast, previous studies have described a linear decline in RT after the td₁ (Giles et al., 1991; Renaudeau et al., 2007). For the later study, the discrepancy between the results is mainly due to the different mathematical models used to fit thermoregulatory responses during thermal acclimation. Our results suggest that mechanisms involved in heat acclimation include a continuum of processes, varying temporally and differing in their efficiency. According to Horowitz (2001), enhanced heat tolerance in the LTHA phase is achieved by increasing the capacity of thermal effectors for heat dissipation and decreasing HP. According to the RT change and some results obtained in other species, some speculation can be made on the mechanisms predominantly involved in LTHA.

A reduction in HP over the acclimation period was reported by Giles et al. (1991), who showed a gradual decline in O₂ consumption in pigs maintained throughout an 11-d exposure to 31°C. In pigs, the total HP can be partitioned into 3 main components: the fasting HP (FHP), HP related to physical activity (HPact), and the thermic effect of feed (van Milgen et al., 1997). Irrespective of the temperature, the gradual recovery trend measured for ADFI indicates that reduced HP in LTHA was not related to a decrease in the thermic effect of feed. Even though no behavioral observations were recorded in the present study, we can assume that HPact would decrease to favor heat exchange between the body and floor. In addition, Quiniou et al. (2001) suggested that a nonnegligible part of HPact at increased ambient temperature can be explained by the intense panting in heat-exposed pigs. This suggests that a reduction in RR would also contribute to the decrease in HP during the thermal acclimation period. Collin et al. (2001) also reported a reduced FHP under hot conditions. According to Koong et al. (1982) and van Milgen et al. (1998), this reduced FHP is generally explained by an indirect effect of reduced feed intake on viscera mass.

In the present study, the TCI change in LTHA was small. In consequence, thermal acclimation was apparently not due to increased sensible heat loss. Similar results were reported in pigs by Morrison and Mount (1971) and Renaudeau et al. (2007). In calves, Bianca (1959) suggested that reduced evaporative heat loss subsequent to the RR decrease would be a consequence, rather than a cause, of heat acclimation. In other words, as the demand for body cooling becomes reduced during thermal acclimation, the respiratory activity declines. However, one can also hypothesize that evaporative heat loss per breath becomes more efficient with time of exposure, resulting in a decrease in RR. It seems that the reduction in metabolic HP may be a major factor involved in the LTHA in pigs, rather than increased heat losses.

**Effect of Temperature on Thermal Acclimation in Pigs**

Even though thermal acclimation responses have been described previously in the literature (Morrison and Mount, 1971; Giles et al., 1991; Collin et al., 2002; Renaudeau et al., 2007), no other study has assessed the effect of temperature on the physiological responses of pigs to a continuous and prolonged heat challenge. According to our data, the STHA response (i.e., v₁), was significantly affected by the heat stress intensity. Based on RT determination, the onset of the LTHA phase was not affected by temperature, whereas mechanisms developed to compensate for the perturbation due to heat stress during this period differed with temperature. At 28°C, when ADFI and RT responses were compared, the td₁ value for ADFI was 1.4 d later than the corresponding value for RT. The opposite result was found at 32 and 36°C. In fact, it appears that below 32°C, the reduction in ADFI within the first days of exposure to increased temperature would also contribute to the long-term acclimation responses, especially between td₁ and td₂. In other words, we hypothesize that at 32 or 36°C, the decrease in ADFI before td₁ is too great, and thereafter, the animals cannot rely on this mechanism to reduce metabolic HP during long-term thermal response to heat.

In contrast to 32 and 36°C, the TCI remained constant from P0 to P1 at 28°C. In fact, the increase in skin temperature at 28°C appeared to be sufficient to maintain the temperature gradient between the body core and surface. As such, we suggest that for moderate heat stress, pigs also could rely on sensible heat exchange to lose heat. When RT measurements were considered, the lag time between the td₁ and td₂ increased by 0.4 d/°C from 28 and 36°C, whereas the slope of the RT decline after td₂ was not affected by temperature. This indicates that the magnitude of heat stress would affect thermoregulatory responses only at the beginning of the LTHA phase. In consequence, mechanisms involved in LTHA would not change with the intensity of heat stress when the duration of exposure to elevated temperature is greater than td₂.

Verhagen et al. (1988) indicated that most of the acclimation in 20-kg pigs occurred within the first 5 d of exposure when temperature increased from 20 to 25°C. At 28°C, the RT value on d 20 was similar to the average RT measured on P0. This result shows that a 20-d period is required to complete acclimation for a
4°C increase in ambient temperature. Assuming that the rate of decline in RT was constant, more than 60 d would be necessary for the thermal acclimation to be completed at 32 or 36°C. These results suggest that the time course for a complete acquisition of thermal acclimation in pigs varies with the magnitude of heat challenge, but this relationship is not linear. With RT measurements, Morrison and Mount (1971) reported that the process of thermal acclimation in 60-kg pigs kept at 33°C and 50% RH was completed within 10 d. The discrepancy between this later study and the present work could be explained by the difference in pig genotype and in RH (50 vs. 80% RH).

**Interindividual Variability on Thermal Acclimation**

By including additional random effects (besides the fixed effect), the NLMIXED procedure takes into account the interindividual variability in the variables of the fitted curves. In the present study, variance estimates of the random effects of the \( y_0 \), \( v_1 \), \( v_3 \), and \( td_1 \) parameters for RT were significantly different from zero. Similar to RT, variance estimates of the \( y_0 \), \( v_1 \), \( v_2 \), and \( v_3 \) parameters for ADFI were significantly different from zero. Calculation of individual correlations between actual and predicted values of RT and ADFI showed a great variability in the quality of fit (0.52 to 0.99 and 0.60 to 0.98, respectively). Hence, this interindividual variability in RT parameters could be explained by the fact that the same mathematical model could not adjust the individual thermal acclimation responses with the same accuracy. However, results of the current study would also indicate that an interindividual variation may exist for the ability to maintain homeothermy during thermal acclimation. In the present study, all the pigs were measured for their growth performance on P0 at thermoneutrality to estimate the maximum production. From these data, residual correlations with random estimates of the model parameters were calculated. The decrease in ADFI before \( td_1 \) was positively related to the average ADFI during P0. From that, it can be suggested that between-pig differences in production would explain differences in body temperature regulation. Moreover, when ANOVA was used to test the sire effect on the individual random effects adjusted for differences attributable to the fixed effects of temperature and replicate, the part of the variance explained by the sire effect for the random estimates of the \( v_1 \) and \( td_1 \) parameters for RT and the \( v_1 \) parameter for ADFI represented approximately 23, 15, and 25% of the total variance, respectively. These results indicate that the interindividual variability for these parameters would be partly genetically determined. However, this hypothesis needs to be tested with an increased size of sire and progeny × sire observations.

In conclusion, we confirmed that the thermoregulatory responses of pigs over time of exposure to increased temperatures have a biphasic pattern, with an initial phase (STHA) followed by an LTHA phase. These responses were affected by the magnitude of heat stress. The NLM used in the present study allowed us to fit the thermal acclimation responses with biological meaningful variables, which were determined with increased accuracy when the appropriate random effects were included. During the LTHA phase, pigs developed adaptive changes, which resulted in an increase in the ability to cope with a hot environment. It is suggested that a decrease in HP plays a central role in acclimation. Changes in the components of HP during LTHA warrant future research.

**LITERATURE CITED**


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