Prolactin's Role During Acute and Chronic Heat Stress in Growing Pigs

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Summary and Implications

We determined the differential effects of environmental hyperthermia and reduced feed intake on circulating prolactin in growing pigs. Furthermore, we evaluated the correlation between prolactin concentration and other metabolic, productive and intestinal parameters during heat stress. Our results indicate that 1) hyperthermia elevates prolactin, and 2) prolactin participates in both the acute heat stress response and the acclimation to environmental hyperthermia. A better understanding of the physiological alterations produced by heat stress is essential in order to develop alleviating strategies to prevent the decrease in production and lost revenue during the warm summer months.

Introduction

Heat stress (HS) is one of the costliest issues for US animal agriculture and it compromises the global competitiveness of the livestock industry. Despite advances in heat abatement systems, the warm summer months are still a financial burden for producers. In the pork industry, reasons for the economic losses derived from HS are reduced growth, poor sow performance and decrease in carcass quality, in addition to increased veterinary costs. Heat stress will be more of a concern if, as some have predicted, the earth's temperatures keep rising as a consequence of global warming. In addition to environmental heat, genetic selection for leaner phenotypes decreases the pigs' tolerance to elevated temperatures. This has been suggested based on the fact that selection for enhancing protein accretion results in increased basal heat production. Consequently, characterizing the physiological and metabolic aspects of hyperthermia is essential in order to develop management, nutritional, and pharmaceutical strategies to overcome the negative impact of HS.

Blood prolactin (PRL) levels increase in a variety of HS animal models, but whether or not HS elevates PRL in growing pigs is ill-defined. Furthermore, the reasons why PRL goes up during HS remain unknown. Our objectives were to evaluate PRL relationship with metabolic and intestinal permeability variables during HS.

Materials and Methods

Crossbred gilts (n=29) averaging 77±2 kg BW were housed individually at the Iowa State University Swine Nutrition Farm. After an 11 d acclimation, pigs were exposed to 1 of 2 environmental treatments: 1) HS conditions (36°C; 50% humidity) and ad libitum feeding (HS, n=13) or 2) thermo-neutral conditions (19°C; 61% humidity) and pair-feeding (PFTN, n=16). The use of a PFTN group allowed for discriminating between the direct or indirect (i.e. via reduced feed intake) effects of HS on animal physiology/metabolism. Rectal temperature (Tr) and respiratory rates (RR) were obtained 4X daily and feed intake (FI) was recorded once a day. Pigs were sacrificed at d1 or 7 of environmental exposure; blood and intestinal tissue were harvested at death.

Results and Discussion

Pigs exposed to HS had an immediate increase ($P<0.01$) in Tr (40.1 vs. 38.4°C) and RR (111 vs. 36 bpm). Heat-stressed pigs reduced ($P<0.05$) FI by 66% (2.62 vs. 0.90 kg) on d1 and 2 of environmental exposure. By d3 and throughout the rest of the experiment, FI moderately recovered to 53% (2.62 vs. 1.23 kg) of the initial FI (i.e. prior to HS). By experimental design, PFTN pigs' intake mirrored the HS pattern (Table 1). Heat stress increased ($P<0.05$) circulating PRL (0.97 vs. 0.64 ng/ml), tended to decrease ($P<0.10$) ileal transepithelial resistance (TER; 80.4 vs. 106.8 Ω/cm^2) and had no effect on colonic TER (Table 2). There was a treatment by time effect on blood glucose as there was acute hyperglycemia in HS pigs but no differences on day 7. In acute HS (24 h; Table 3), PRL was negatively correlated with feed intake ($r = -0.71$; $P<0.05$), ileal TER ($r = -0.85$; $P<0.01$) and tended to be negatively correlated with colonic TER ($r = -0.69$; $P=0.06$). In chronic HS (d7; Table 4), PRL tended to be only inversely correlated with glucose ($r = -0.81$; $P<0.10$). In PFTN pigs, PRL was not correlated with any of the measured variables.

During heat stress there is a diversion of blood flow from viscera to the skin in an attempt to dissipate heat and avoid hyperthermia. In the case of the gastro-intestinal tract, a lack of adequate blood irrigation can jeopardize epithelial tight junction integrity, and this may lead to increased permeability or "leaky gut". The tightness of the intestinal junctions can be assessed by the TER, where lower resistance indicates higher permeability.

Prolactin is elevated in several animal models of heat stress: rats (Siegel et al., 1979), ewes (Hill and Alliston, 1981), dairy cows (Igono et al, 1987), etc. Our data

demonstrates this is also true for growing pigs; however PRL's role in HS remains unknown. The variables correlated with PRL differ between d1 and d7 of HS. Moreover, PRL was not correlated with any of these variables in the PFTN group. Together, this data suggests that PRL appears to participate in both the acute HS response and the acclimation to chronic environmental hyperthermia.

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Table 1. Effects of heat stress on feed intake, rectal temperature and respiratory rate in growing pigs.

	Period 1*		Period 2**			*P*		
Parameter	PFTN	HS	PFTN	HS	SE	Trt	Day	Trt x Day
Feed Intake, kg	2.5	2.7	1.2	1.1	0.07	0.7	<0.01	0.3
Rectal Temperature, °C	38.91	38.91	38.37	40.06	0.08	<0.01	<0.01	0.24
Respiratory Rate, bpm	35	37	36	111	2.23	<0.01	<0.01	<0.01

*Period 1 refers to pre-environmental exposure.

**Period 2 refers to during the environmental exposure.

Table 2. Effects of heat stress on blood and intestinal parameters in growing pigs.

	d1*		d7*			*P*		
Parameter	PFTN	HS	PFTN	HS	SE	Trt	Day	Trt x Day
Blood parameters:								
Prolactin, ng/ml	0.66	0.83	0.62	1.1	0.10	<0.01	0.27	0.15
Glucose, mg/dl	112.33[a]	138.17[b]	134.12[b]	122.92[ab]	7.84	0.36	0.68	0.03
Intestinal parameters:								
Ileal TER**, Ω/cm2	122.31	87.45	91.29	73.28	14.04	0.07	0.12	0.56
Colonic TER**, Ω/cm2	109.3	114.78	105.61	82.75	10.97	0.44	0.12	0.21

*d1 and 7 relative to initiation of environmental exposure.

** Transepithelial resistance

[a,b] Indicate significant differences in Trt x Day

Table 3. Effects of acute (24 h) heat stress on blood intestinal parameters correlations in growing pigs.

Pearson Correlation Coefficients, N = 8 Prob > |r| under H0: Rho=0					
	Glucose	**PRL**	**TER_ colon**	**TER_ ileum**	**FI**
Glucose	1.00	-0.17 0.69	0.41 0.31	0.04 0.93	0.14 0.74
PRL	-0.17 0.69	1.00	-0.69 0.06	-0.85 <0.01	-0.71 0.05
TER_ colon	0.41 0.31	-0.69 0.06	1.00	0.51 0.20	0.19 0.65
TER_ ileum	0.04 0.93	-0.85 <0.01	0.51 0.20	1.00	0.49 0.21
FI	0.14 0.74	-0.71 0.05	0.19 0.65	0.49 0.21	1.00

Table 4. Effects of chronic (7 d) of heat stress on blood and parameters correlations in growing pigs.

Pearson Correlation Coefficients, N = 5 Prob > |r| under H0: Rho=0					
	Glucose	**PRL**	**TER_ Colon**	**TER_ ileum**	**FI**
Glucose	1.00	-0.81 <0.10	<0.01 1.00	0.31 0.61	-0.02 0.97
PRL	-0.81 <0.10	1.00	-0.30 0.62	-0.59 0.29	-0.05 0.93
TER_ colon	<0.01 1.00	-0.30 0.62	1.00	-0.15 0.81	0.17 0.79
TER_ ileum	0.31 0.61	-0.59 0.29	-0.15 0.81	1.00	0.54 0.35
FI	-0.02 0.97	-0.05 0.93	0.17 0.79	0.54 0.35	1.00