RESEARCH ABSTRACT

PORK QUALITY

Title: Impact of Seasonal Heat Stress on Fatty Acid Composition and Pork Fat Quality – **NPB #13-080**

Investigator: Lance H. Baumgard, N. Jacobson Professor of Nutritional Physiology

Co-Investigators: John F. Patience, ISU
Kenneth Prusa, ISU
Tim Goodmann, JBS, Greely CO
Roger Johnson, Farmland Foods, Denison, IA

Institution: Iowa State University

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Scientific Abstracts:

Experiment 1

Heat stress results in major losses to the pork industry through reduced growth performance and possibly carcass fat quality. The objective was to investigate the effect of heat stress on the pig's response to dietary fat in terms of growth performance, dietary lipid digestion, and pork fat quality over a 35 d finishing period. A total of 96 barrows (PIC 337 × C22/29) with an initial BW of 100.4 ± 1.2 kg were randomly allotted to 1 of 9 treatments arranged as a 3 × 3 factorial: [TN (thermonetural: constant 24°C; ad libitum access to feed), PFTN (pair-fed thermoneutral: constant 24°C; limit-fed based on previous HS daily feed intake), or HS (heat stress: cyclical 28°C nighttime, 33°C-35°C daytime; ab libitum access to feed)] and diet [a corn-soybean meal based diet with 0% added fat (CNTR), 3% added tallow (3%TAL; iodine value = 41.8), or 3% added corn oil (3%CO; iodine value = 123.0)]. Pigs were individually housed to track intake and to allow for jowl fat biopsies. Titanium dioxide was included as an indigestible marker at 0.4%. Fecal samples were collected on d 17 (~ 114 kg). True total track digestibility (TTTD) (%) of acid hydrolyzed ether extract (AEE) was calculated by correcting apparent total track digestibility (ATTD) of AEE for endogenous fat losses at 20 g of AEE/kg of dry matter intake. Data were analyzed using PROC MIXED with environment and dietary treatment as fixed effects, and replicate (2 replicates of 48 barrows) as a random effect. Rectal temperature (HS = 39.0, TN = 38.1, PFTN = 38.2°C) and respiration rates (HS = 78, TN = 36, PFTN = 34 bpm) increased due to HS ($P < 0.001$). HS decreased ADFI (27.8%; $P < 0.001$). ADG (HS = 0.72, TN = 1.03, PFTN = 0.78 kg/d; $P < 0.001$), and G:F (HS = 0.290, TN = 0.301, PFTN = 0.319; $P < 0.01$) also reduced by HS. G:F but not ADG or ADFI tended to be influenced by dietary treatment (CNTR = 0.292, 3%TAL = 0.303, 3%CO = 0.314 g/100 g; $P \leq 0.07$). Exposure to HS did not impact IV at market (HS = 69.2, TN = 69.3, PFTN = 69.8 g/100 g; $P = 0.62$). Carcass IV increased with increasing degree of unsaturation of the dietary fat (CNTR = 68.5, 3%TAL = 68.2, 3%CO = 71.5 g/100 g; $P < 0.001$). HS tended to have the lowest ATTD of AEE (HS = 59.0, TN = 60.2, PFTN = 61.4%, $P = 0.055$). Inclusion of dietary fat, and a source that was unsaturated increased ATTD of AEE (CNTR = 41.6, 3%TAL = 67.9, 3%CO = 71.2%, $P < 0.001$). TTTD of AEE

of 3%CO-based diets was higher (99.3%) than that of CNTR (97.3%) and 3%TAL-based diets (96.3%; $P = 0.01$). Environment had no impact of TTTD of AEE ($P = 0.12$). There was no interaction between HS and diet ($P \geq 0.19$) on carcass IV. In conclusion, HS impaired growth performance but not carcass IV or the response to dietary fat. IV was affected by dietary fat source.

Experiment 2

Heat stress (HS) negatively impacts several production variables in swine, including carcass fat quality. Study objectives were to evaluate insulin's role (via feeding insulin sensitizing compounds) in decreasing fat quality during HS. Forty crossbred barrows (113±9 kg BW) were randomly assigned to 1 of 5 treatments during 2 experimental periods: 1) thermoneutral (TN) *ad libitum* feed intake control (TNAL), 2) TN pair-fed control (TNPF), 3) HS *ad libitum* (HSAL), 4) HS *ad libitum* with dietary sterculic oil (HSSO; 13 g/d), and 5) HS *ad libitum* with dietary chromium propionate (HSCr; 0.5 mg/d; Kemin Industries, Des Moines, IA). During period 1 (7 d), all pigs were exposed to TN conditions (23±3°C, 68±10% RH) and fed *ad libitum*. During period 2 (21 d), HSAL, HSSO, and HSCr pigs were fed *ad libitum* and exposed to cyclical (HS) conditions (28 to 33°C, 58±10% RH). The TNAL and TNPF pigs remained in TN conditions and were fed *ad libitum* or pair-fed to their HSAL counterparts (to eliminate the confounding effect of dissimilar nutrient intake), respectively. Rectal temperature (T_r), respiration rate, and skin temperature were increased (0.9°C, 37 bpm, and 2.5°C, respectively) in HS pigs. Interestingly, HSSO increased T_r relative to HSAL and HSCr (0.40 and 0.42°C, respectively) during the last week of period 2 ($P < 0.05$). HS decreased feed intake and ADG compared to TNAL (2.43 vs. 3.26 kg/d and 0.74 vs. 1.09 kg/d, respectively; $P < 0.01$) and neither variable was affected by SO or Cr supplementation. Moisture content tended to be increased in pigs from all HS treatments compared to TNAL controls in abdominal (7.7 vs. 5.9%; $P < 0.09$) and inner subcutaneous adipose tissue (11.4 vs. 9.8%; $P < 0.06$) depots. Interestingly, TNPF pigs had or tended to have increased adipose tissue moisture content in abdominal (7.3 vs. 5.9%; $P < 0.01$), inner subcutaneous (11.0 vs. 9.8%; $P < 0.07$), and outer subcutaneous (14.9 vs. 12.1%; $P < 0.01$) depots compared to TNAL pigs. HS had little or no effect on fatty acid (FA) composition of abdominal, inner, and outer subcutaneous adipose tissue depots. Feeding SO decreased the desaturase index in the abdominal (0.36 vs. 0.43; $P < 0.01$), inner subcutaneous (0.46 vs. 0.52; $P < 0.01$), and outer subcutaneous (0.51 vs. 0.55; $P < 0.01$) adipose tissue. In summary, HS did not alter FA composition in any adipose depot, but both TNPF and HS markedly increased adipose tissue moisture content.