Effects of Somatotropin and Salbutamol in Three Genotypes of Finishing Barrows: Growth, Carcass, and Calorimeter Criteria[1,2,3]

J. A. Hansen*,[4], J. T. Yen†, J. L. Nelssen*, J. A. Nienaber†, R. D. Goodband*,[5], and T. L. Wheeler†

*Department of Animal Sciences and Industry, Kansas State University, Manhattan 66506-0201 and †Roman L. Hruska U.S. Meat Animal Research Center, Agricultural Research Service, U.S. Department of Agriculture, Clay Center, NE 68933

ABSTRACT: We evaluated the combined use of 0 or 4 mg/d of recombinant porcine somatotropin (pST) and 0 or 2.75 ppm salbutamol in three genotypes of growing barrows (139 d old) differing in lean and lipid accretion potential. Treatments were in a 2 × 2 × 3 factorial arrangement, and the three genotypes tested were Meishan (M, n = 32, 49 kg), Duroc × White composite (D × Wc, n = 31, 62 kg), and Meishan × White composite (M × Wc, n = 31, 64 kg) pigs. Growth performance was evaluated over 28 d for individual pigs, and 20-h feed-deprived heat production was measured before slaughter (d 34). Daily pST injection increased ADG (+70 g/d) and reduced ADFI (–.61 kg/d) across genotypes ($P < .05$). Salbutamol increased ($P < .05$) ADG in M × Wc pigs (+146 g/d) but not in M pigs (–60 g/d) or D × Wc (+80 g/d) pigs. However, M pigs had the lowest ADG and ADFI, and M × Wc pigs not treated with salbutamol grew slower than D × Wc pigs ($P < .05$). Carcass protein and moisture accretion were additively ($P < .05$) increased by pST and salbutamol for D × Wc and M × Wc pigs. Meishan pigs had increased carcass protein and moisture accretion from pST, whereas only moisture accretion was increased by salbutamol ($P < .05$). The longissimus muscle area and semitendinosus weight increased as the percentage of M in the genotype decreased ($P < .05$), and both were increased by pST or salbutamol treatment ($P < .001$). Leaf fat was decreased more ($P < .05$) in M pigs than in D × Wc or M × Wc pigs with pST injection. The similar magnitude of leaf fat reduction between D × Wc and M × Wc pigs resulted in a mean genotype difference ($P < .05$), and salbutamol decreased leaf fat across genotypes. Oxygen consumption and heat production were increased by pST in M pigs more than in the crossbred genotypes, but CO_2 production was reduced by similar magnitudes across genotypes, and salbutamol only tended to reduce CO_2 production in D × Wc pigs. In general, these data indicate that pST and salbutamol result in additive increases in carcass lean composition; however, growth rate, carcass accretion, and various organ weights may vary among genotypes with salbutamol and pST treatment.

Key Words: Pigs, Somatotropin, β-Adrenergic Agonists, Performance, Calorimetry, Carcasses

J. Anim. Sci. 1997. 75:1798–1809

Introduction

Administration of exogenous porcine somatotropin (**pST**) greatly alters carcass composition and visceral organ mass of growing swine (Boyd and Bauman, 1989; Brier and Gluckman, 1991). The response to pST treatment is dose related (Etherton et al., 1987; Evock et al., 1988; McLaren et al., 1990) and seems to be limited by dietary nutrient intake (Campbell et al,

[1]Contribution no. 93-469-J from the Kansas Agric. Exp. Sta., Manhattan. Experiment no. 5438-31000-024-0-2, ARS, Roman L. Hruska U.S. Meat Anim. Res. Center.

[2]The authors thank B. R. Shricker of Mallinckrodt Veterinary (formerly Pittman-Moore, Inc.), Mundelein, IL, for providing the recombinant pST used in this research. Appreciation is expressed to T. W. Acton, J. A. Woods, R. J. Pooschke, and T. C. Ziegler for care and feeding of animals, to P. J. Reiman and associates for feed preparation; to D. A. Kohmetscher and associates for slaughter, and to S. S. Cummins, E. L. Shetler, J. A. Waechter, and associates for technical assistance.

[3]Mention of a trade name, proprietary product, or specific equipment does not constitute a guarantee or warranty by the USDA or Kansas State University and does not imply approval to the exclusion of other products that may be suitable.

[4]Present address: Murphy Family Farms, Rose Hill, NC 28458.

[5]To whom correspondence should be addressed.

Received June 27, 1996.

Accepted March 13, 1997.

1990, 1991; Goodband et al., 1990). With respect to genetic potential, pST elicits more dramatic improvements in genotypes that have a greater propensity for lipid deposition (Bidanel et al., 1991; Klindt et al., 1992; Noblet et al., 1992a), although others have suggested that it can accentuate breed differences (Nossaman et al., 1991).

Salbutamol (hemisulfate salt; α-[(t-butylamino)methyl]4-hydroxy-m-xylene-α,α′-diol), a β-adrenergic agonist, has been evaluated to a limited extent in swine (Oksbjerg et al., 1990; Warriss et al., 1990a,b). Nevertheless, increases in carcass muscling with salbutamol are similar to those reported with ractopamine (Yen et al., 1990a, 1991). Differences among genotypes in responses of growth or carcass traits to either salbutamol or ractopamine are less pronounced (Warriss et al., 1990a,b; Yen et al., 1990a, 1991) than responses to pST administration. Experiments evaluating the combined use of somatotropin and a β-adrenergic agonist have shown additive increases in carcass muscle criteria for pigs (Jones et al., 1989), rodents (Bates and Pell, 1991), and cattle (Maltin et al., 1990). It is not known whether differential responses to the combined use of pST and a β-adrenergic agonist exist among different genotypes of animals.

Thus, the objective of our research was to evaluate the effects of singular and combined use of pST and salbutamol on growth and carcass traits, heat production during feed deprivation, and visceral organ mass in swine with different capacities for lean and lipid accretion.

Materials and Methods

Animals and Environment. The experiment was conducted from December 1991 through January 1992. Because the compounds used in this research are not presently approved for use in swine by the U.S. Food and Drug Administration, all remaining feed and animal tissues not kept for analyses were buried at the conclusion of this research. The genotypes evaluated were as follows: 1/4 Duroc, 3/4 MARC White Composite (**D** × **Wc**); 1/4 Meishan, 3/4 MARC White Composite (**M** × **Wc**); and purebred Meishan (**M**). The MARC White Composite was 1/4 Chester White, 1/4 Landrace, 1/4 Large White, and 1/4 Yorkshire. Because of the slow growth rate of M pigs, a similar age rather than initial weight was used for comparing these genotypes. Eight barrows from each genotype were slaughtered at approximately 136 d of age for determination of initial carcass chemical composition. Because the pigs were born during a 5-wk period and to ensure that pigs had similar initial ages, eight pigs from each genotype were placed on test each week for four consecutive weeks (n = 96). Within genotype, pigs were randomly assigned on the basis of ancestry to one of four treatments.

Table 1. Diet composition (as fed)[a]

Ingredient	Amount
Corn	71.49
Soybean meal (48% CP)	13.46
Select menhaden fish meal	5.00
Spray-dried porcine plasma	5.00
Soybean oil	2.00
Dicalcium phosphate (18.5% P, 21% Ca)	1.96
Limestone	.29
Salt	.25
Premix[b]	.40
L-Lysine·HCl	.12
DL-Methionine	.03

[a]Salbutamol was included at 0 or 2.75 ppm of the complete diet.
[b]Premix provided the following per kilogram of complete diet): vitamin A, 5,512 IU; vitamin D_3, 551 IU; vitamin E, 22 IU; menadione, 2.2 mg; riboflavin, 5.5 mg; d-pantothenic acid, 13.8 mg; niacin, 30.3 mg; choline, 551 mg; vitamin B_{12}, 27.6 μg; 150 mg Mn; Fe, 150 mg; Zn, 150 mg; Ca, 60 mg; Cu, 15 mg; K, 6 mg; I, 4.5 mg; Na, 3 mg; Co, 1.5 mg.

Treatments were arranged according to a 2 × 2 × 3 factorial evaluating daily injections of buffer or 4 mg of recombinant pST (Mallinckodt Veterinary [formerly Pitman-Moore, Inc.], Mundelein, IL), and diets containing 0 or 2.75 ppm salbutamol (Sigma Chemical, St. Louis, MO). Composition of the basal diet is described in Table 1 and was selected based on an initial investigation (Hansen et al., 1994). The basal diet was formulated to contain 1.2% lysine, .9% Ca,

Table 2. Chemical composition of experimental diets (as fed)[a]

Item	Amount
GE, kcal/kg	4,080
CP, %	17.78
Ca, %	.90
P, %	.80
Indispensable amino acids, %	
Arginine	1.01
Histidine	.48
Isoleucine	.67
Leucine	1.67
Lysine	1.09
Methionine	.31
Phenylalanine	.85
Threonine	.71
Tryptophan	.23
Valine	.89
Dispensable amino acids, %	
Cystine	.38
Alanine	1.02
Aspartic acid	1.57
Glutamic acid	2.87
Glycine	.79
Proline	1.17

[a]Values are means of the basal and salbutamol-treated diets and represent analyzed contents.

Table 3. Chemical composition (percentage) of the initial slaughter group carcasses[a,b]

Item	Genotype			SD	Significance[c]
	D × Wc	M	M × Wc		
Moisture	54.9	52.0	52.6	2.9	G1
CP	14.8	14.3	14.5	.6	—
Lipid	27.2	30.6	29.9	3.4	G1
Ash	2.9	2.9	2.8	.3	—
Total	99.8	99.8	99.8	.2	—

[a]Values are means of eight pigs for each genotype.
[b]D × Wc = Duroc × White composite crossbred; M = Meishan; M × Wc = Meishan × White composite crossbred.
[c]Comparisons for genotype: (G [1 = D × Wc vs M; 2 = M vs M × Wc; 3 = D × Wc vs M × Wc]; $P < .15$).

and .8% P, and amounts of indispensable amino acids were adjusted relative to lysine to supply at least the minimum requirement based on the ratio described for 50- to 110-kg pigs by NRC (1988). Chemical composition of the basal diet was determined using AOAC (1984) procedures, and amino acids were quantified by HPLC following acid hydrolysis (Table 2). Sulfur-containing amino acids were quantified after performic acid oxidation (Moore, 1963). Pigs were housed in individual pens (1.22 × 1.22 m) equipped with feeders and an automatic nipple waterer in an enclosed, temperature-controlled, slotted-floor building. Recombinant somatotropin was dissolved in bicarbonate buffer (25 m*M* $NaHCO_3$, 25 m*M* Na_2CO_3, .154 *M* NaCl; pH 9.4) in quantities to last 3 d, and injections were administered between 0700 and 0800 daily. Pigs and feeders were weighed on d 0 and 14 and 28 and 24 h prior to slaughter, at which time the feeder was removed from the pen. Average daily gain, ADFI, and gain/feed (**G/F**) are reported from d 0 to 28. One D × Wc pig (4 mg pST/d, 0 ppm salbutamol) was deemed unsuitable to complete the trial because of loss of appetite and weight loss, and one M × Wc pig (4 mg pST/d, 0 ppm salbutamol) died after 18 d on test. Necropsy of the dead pig indicated gastroenteritis with gastric ulcers near the cardia of the stomach.

Indirect Calorimetry. Because only three calorimeters were available each day (4 d/wk), three pigs from each genotype on the same pST and salbutamol treatment combination were placed into the calorimeters at 0800. The pig that died was scheduled to enter the calorimeter; therefore, only 47 pigs were used for criteria measurements. Oxygen consumption and CO_2 production were measured for 20 h (1130 to 0730 the next day) and were obtained as described previously (Nienaber and Maddy, 1985). While in the calorimeters, pigs were allowed ad libitum access to water but were denied access to feed. Differences in total number of days from initiation to termination for all treatment groups were minimized by rotating the day of week treatment groups entered the calorimeter over the 4-wk data collection period. After calorimetric measurements were taken, pigs were transported to an abattoir and slaughtered with three other pigs (one pig per genotype) that were on the same pST and salbutamol treatment but were not used for calorimetry measurement.

Slaughter and Carcass Data Collection. At slaughter, pigs were weighed, killed by electric stunning and exanguination, skinned, eviscerated, and split prior to weighing of the hot carcasses. Fresh weights of heart, liver, lungs, spleen, pancreas, kidney, and leaf fat were recorded. Gastrointestinal tracts were dissected into small intestine, cecum, large intestine, and rectum; tissues were emptied of contents, cleansed, blotted dry, and weighed. Weights of spleen, pancreas, stomach, and gastrointestinal tract were obtained individually and collectively represent the portal vein-drained organs (**PVDO**). Carcasses were allowed to

Table 4. Regression coefficients used to estimate initial carcass composition[a,b]

Item	Genotype		
	D × Wc	M	M × Wc
Moisture			
Intercept	–2,001.3	–130.2	3,275.3
Slope	379.5	267.8	263.3
R^2	.96	.98	.81
CP			
Intercept	–1,661.9	–129.9	239.5
Slope	123.4	75.7	84.4
R^2	.93	.95	.91
Lipid			
Intercept	–1,187.1	398.9	–8,376.5*
Slope*	191.7	149.1	328.9
R^2	.75	.83	.85
Ash			
Intercept	–348.2	–361.0	–305.7
Slope*	24.5	22.9	22.2
R^2	.73	.83	.93

[a]Values were obtained by regressing the slaughter weight of eight pigs per genotype on carcass composition for estimation of initial carcass composition for the test group of pigs.
[b]D × Wc = Duroc × White composite crossbred; M = Meishan; M × Wc = Meishan × White composite crossbred.
*$P < .05$ that value ≠ 0.

Table 5. Carcass components and characteristics of the initial slaughter group[a]

Item	Genotype[b] D × Wc	M	M × Wc	SD	Significance[c]
Age, d	133	136	141	4	G23
BW, kg	53.6	45.7	59.3	8.4	G2
Hot carcass, kg	33.4	23.3	36.3	5.6	G12
Dressing percentage	62.2	50.9	61.0	2.0	G12
Head, %	7.60	8.37	7.36	.68	G12
Feet, %	1.80	2.04	1.85	.18	G12
Skin, %	8.38	11.47	8.94	1.19	G12
Leaf fat, %	.99	1.77	1.31	.29	G123
Longissimus muscle (10th rib)[d]					
Area, cm^2	20.71	12.86	21.06	2.03	G12
Area, cm^2 [e]	20.57	14.23	19.83	2.03	G12
Temperature, °C[f]	5.51	3.31	6.43	1.80	G2
pH[f]	5.94	6.13	5.78	.21	G23
Semitendinosus[d]					
Weight, g	201.0	90.6	189.6	28.5	G12
Weight, %	.38	.20	.32	.03	G123
pH[f]	6.04	6.01	5.88	.26	—
Organs, %					
Heart	.35	.37	.35	.02	—
Lung	.76	.70	.63	.13	—
Liver	1.88	2.28	1.89	.31	G12
Kidney	.34	.40	.33	.04	G12
PVDO[g]	4.77	6.52	5.12	.63	G12
Spleen	.17	.21	.15	.05	G2
Pancreas	.16	.21	.18	.02	G12
Stomach	.69	.87	.76	.08	G12
Small intestine	1.98	2.44	1.87	.30	G12
Cecum	.16	.23	.19	.04	G12
Large intestine	1.62	2.55	1.96	.30	G123

[a]Values are means of eight pigs each and are expressed as the percentage of slaughter weight where designated.
[b]D × Wc = Duroc × White composite crossbred; M = Meishan; M × Wc = Meishan × White composite crossbred.
[c]Comparisons for genotype: (G [1 = D × Wc vs M; 2 = M vs M × Wc; 3 = D × Wc vs M × Wc]; $P < .05$).
[d]Values were obtained at approximately 6 h postmortem unless otherwise noted.
[e]Slaughter weight was included as a covariate.
[f]Time from slaughter to measurement was included as a covariate.
[g]Portal vein-drained organs including spleen, pancreas, stomach, small intestine, cecum, and large intestine.

chill at 2°C for 6 h, at which time longissimus muscle area (**LMA**) of the left side between the 10th and 11th ribs was traced onto acetate paper for measurement. Internal temperature of the longissimus taken from between the 7th and 10th ribs was recorded, and a 2.5-g sample was removed from the interior of the muscle for pH determination (Bendall, 1973). The semitendinosus muscle was removed in its entirety from the left side at 6 h postmortem and weighed, and a 2.5-g interior sample was removed for pH determination.

The right side of each carcass was frozen at –20°C and then ground (model 1109, Weiler and Co., Whitewater, WI) successively three times through a face plate with 6-mm holes. Duplicate samples (100 g) were analyzed for moisture by freeze-drying (Labconco Freeze Dry-12 With Tray Dryer, model 75011, Labconco Co., Kansas City, MO) and for lipid, ash, and Kjeldahl N by AOAC (1984) procedures. Accretion rates for each individual component and total carcass were calculated from estimated initial composition and analyzed final composition. Initial composition estimates were obtained by regressing the composition (Table 3) of the initial slaughter group on slaughter weight by genotype (Table 4), and rates of tissue deposition were computed using the actual number of days for each pig until slaughter. For comparison, those slaughter and carcass criteria measured for the test group are also presented for the initial slaughter group (Table 5).

Statistical Analyses. Data were analyzed as a completely randomized design using the GLM procedure of SAS (1988). Analysis of covariance was conducted on select muscle criteria. Individual pig was considered the experimental unit, and the variance was partitioned using orthogonal contrasts appropriate for a 2 × 2 × 3 factorial treatment arrangement including all main effects and two-way and three-way

interactions. A protected LSD was used to separate genotype and interaction means when the *F*-test was significant for these effects. Because two pigs were excluded from the data set, values presented are least squares means.

Results

Initial Slaughter Group. Because the pigs used in this research were born over a 5-wk period, the average age was slightly younger for D × Wc pigs than for M or M × Wc pigs. However, M pigs had lower slaughter BW and hot carcass weights than D × Wc or M × Wc pigs (Table 5). Head, feet, and skin weights represented a greater proportion of the live BW in M pigs than in D × Wc and M × Wc pigs ($P < .05$). The LMA and semitendinosus weight (total grams) were less for M pigs than for the crossbred pigs ($P < .001$). However, when semitendinosus weight was expressed relative to BW, pigs ranked in descending order by increasing percentage of M in the genotype ($P < .001$). Longissimus muscle temperature and pH seemed to be inversely related and highly dependent upon slaughter weight, because as pigs having a heavier slaughter or hot carcass weight had higher 6-h temperatures and lower pH. This relationship was most evident when comparing M × Wc and M pigs ($P < .01$), which were heaviest and lightest, respectively.

With the exception of heart, lung, and spleen, visceral organ weights relative to slaughter weight were greatest in M pigs ($P < .05$) and seemed to increase as the percentage of M in the genotype increased (i.e., numerically higher for M × Wc than D × Wc pigs). However, statistical differences were detected only between M × Wc and D × Wc pigs for large intestine weight. Also, spleen weight was not different between M and D × Wc pigs, but M × Wc pigs had heavier spleens than did M pigs.

Carcass lipid (Table 3) tended to be higher and moisture lower for M pigs than for D × Wc pigs ($P = .14$ and $P = .12$, respectively). However, no differences were observed among genotypes for carcass protein or ash ($P > .31$). Regression equations for the initial slaughter group (Table 4) demonstrated different intercept and slope coefficients for the three genotypes, but these were not statistically compared. Correlation coefficients for regression equations were high and suggest that predictability was accurate for estimation of initial composition of the test group based on initial weight.

Growth Performance and Calorimetry. Results for growth performance and calorimetry criteria are presented in Table 6. An interaction ($P < .03$) was observed between genotype and salbutamol for ADG resulting from M × Wc pigs gaining 146 g/d faster with salbutamol, whereas M pigs were unaffected and D × Wc pigs had a numerical increase (+80 g/d, $P = .14$) in

M × Wc	pST		SD	Significance[b]
alb	Basal	Salb		
8	7	8		
2	142	142	3	G13
3	67	61	6.5	G12
9	90	87	8.7	G12
5	832	913	148	G × S, P
5	388	418	47	P, S
4	3	4		
7.5	90.6	88.7	8.7	G12
.98	1.12	1.15	.08	G × P
.87	.92	.95	.08	G × S, P
4.83	5.45	5.59	.39	G × P
.89	.82	.82	.04	G × S, P

otropin; pST = 4 mg/d somatotropin; Basal = 0 ppm

3 = D × Wc vs M × Wc]). See Results section for a

ADG. Independent of salbutamol treatment, M pigs had the lowest ADG, M × Wc pigs were intermediate, and D × Wc pigs ranked highest among genotypes ($P < .01$); however, ADG of M × Wc pigs receiving salbutamol was similar to that of D × Wc pigs with or without salbutamol treatment. Daily gain was improved by individual treatment with pST (+70 g/d, $P < .03$), but combined treatment with pST and salbutamol tended not to yield an additive improvement (pST × salbutamol; $P = .09$). Daily feed intake was lower ($P < .001$) for M pigs than D × Wc or M × Wc pigs and was reduced independently of genotype or salbutamol when pigs were injected with pST ($P < .001$) and was unaffected by salbutamol treatment. Efficiency of feed utilization was not affected by genotype, whereas pST and salbutamol additively increased G/F ($P < .01$), but to differing degrees (126 vs 29 g/kg, respectively).

Somatotropin differentially increased O_2 consumption and heat production among genotypes ($P < .04$), whereas salbutamol had no effect. Both criteria were similar across genotypes with no pST and were increased by pST administration ($P < .001$). The interaction was due to M pigs having a greater increase in O_2 consumption and heat production (+34 and 31%, respectively) than the other two genotypes (18 and 17%, respectively). Somatotropin injection expectedly increased the production of CO_2 (+18%, $P < .01$) as a result of increased O_2 consumption, but the degree of increase was similar among genotypes. Salbutamol reduced CO_2 production in D × Wc pigs (−10%, $P < .05$) but not in M or M × Wc pigs. Untreated D × Wc pigs produced more CO_2 than did untreated M × Wc pigs (−9%, $P = .05$). A tendency for a genotype × pST × salbutamol interaction ($P = .07$) was observed for CO_2 production, which apparently resulted from M × Wc pigs having increased CO_2 production rates in the absence of pST and numerical decreases in the presence of pST. The ratio of O_2 consumed to CO_2 produced (respiratory quotient, **RQ**) was decreased by pST (−5%, $P < .001$) across genotypes, whereas salbutamol tended to reduce RQ only in M pigs (−5%, $P = .08$). Furthermore, in the absence of salbutamol, M pigs had a higher RQ (+6%, $P = .05$) than did M × Wc pigs, and D × Wc pigs were intermediate.

Slaughter and Carcass Criteria. Slaughter and carcass measurements for the test group are presented in Table 7. Pigs were of similar age, but M pigs had the lowest slaughter and hot carcass weights ($P < .001$). Dressing percentage decreased with increasing M in the genotype ($P < .001$), and pST decreased it compared with controls (58.4 vs 60.2%, respectively). Salbutamol caused dressing percentage to be increased across genotypes (58.6 vs 60.0%). Head and feet weights were greater ($P < .001$) for M pigs than for M × Wc or D × Wc, which were not different, and both were increased by pST administration ($P < .02$) but unaffected by salbutamol. Even though a genotype × pST interaction was observed for skin weight, M and M × Wc pigs had greater weights than did D × Wc pigs ($P < .05$), and the degree of change was related to the amount of M represented in the genotype. Interactions were accounted for by increased skin weight in M pigs with pST (13.37 vs 14.50%; $P < .01$), whereas no difference was observed between pST treatments in the crossbred pigs. Leaf fat weight among genotypes was also interactively influenced by pST, resulting from M pigs having greater reductions (−52%, $P < .001$) in leaf fat than either D × Wc or M × Wc pigs (−33 and 28%, respectively). Meishan pigs receiving no pST had the greatest amount of leaf fat, and only numerical differences were observed between the crossbred pigs.

The semitendinosus weight and LMA were greatest for D × Wc, intermediate for M × Wc, and lowest for M pigs ($P < .02$). In general, LMA was increased by 13% with pST or salbutamol ($P < .001$), but semitendinosus weight was increased more by pST than by salbutamol (41 vs 25 g, respectively). Longissimus temperature at 6 h postmortem was interactively influenced by pST and salbutamol ($P < .02$) independent of genotype, although M pigs had lower temperatures ($P < .001$) as a result of reduced carcass mass. The pST × salbutamol interaction resulted from an increase in temperature caused by salbutamol in the absence of pST, whereas temperature was reduced by salbutamol in the presence of pST. However, pST treatment alone elicited no effect. Longissimus pH was unaffected by pST or salbutamol at 6 h postmortem, although pH was highest for M pigs ($P < .001$), and seemed to be related to muscle temperature. Semitendinosus pH was higher than that of longissimus and followed a similar pattern across genotypes, but salbutamol caused an increase only in semitendinosus pH ($P < .001$).

Heart weight was increased more ($P < .001$) by pST in M pigs than in either D × Wc or M × Wc pigs. Weight of lungs seemed ($P = .12$) to be reduced by salbutamol when pigs received pST, although pST independently increased weight. However, lung weight was increased by salbutamol in D × Wc pigs and decreased in M × Wc pigs ($P < .03$), but no effect was observed in M pigs. Liver weight seemed ($P = .15$) to be increased more by pST in M pigs than either D × Wc or M × Wc pigs, but differences among genotypes were more evident when evaluated independently of pST treatment. Regardless of genotype, pST increased liver weight ($P < .001$). Salbutamol decreased liver weight in D × Wc ($P < .01$) and M ($P = .07$) pigs but not in M × Wc pigs. Kidneys of M pigs were larger than those of the crossbred pigs ($P < .02$), and pST increased weight similarly across genotypes ($P < .001$).

The combined PVDO represented a greater proportion of BW as the amount of M in the genotype

Table 7. Slaughter and carcass characteristics of pigs receiving pST and salbutamol[a]

	D × Wc				M				M × Wc					
	Buffer		pST		Buffer		pST		Buffer		pST			
Item	Basal	Salb	Basal	Salb	Basal	Salb	Basal	Salb	Basal	Salb	Basal	Salb	SD	Significance[b]
Age, d	168	165	166	165	177	178	177	177	176	176	177	176	3	G13
BW, kg	86.6	89.9	90.9	91.4	63.8	62.9	68.1	61.4	85.1	87.8	91.1	87.3	8.3	G12
Hot carcass, kg	56.4	58.9	56.7	59.4	32.7	33.3	34.1	31.0	53.4	55.7	54.5	54.6	5.4	G123
Dressing percentage	65.1	65.4	62.2	65.0	51.3	52.9	50.3	50.5	62.8	63.4	59.9	62.6	1.9	G123, P, S
Head, %	6.80	6.61	7.13	7.15	9.12	9.08	9.43	9.85	7.07	6.58	7.66	7.38	.69	G12, P
Feet, %	1.63	1.60	1.73	1.66	2.08	2.00	2.13	2.27	1.52	1.55	1.74	1.64	.25	G12, P
Skin, %	8.98	8.82	9.02	8.37	14.03	12.71	14.53	14.47	9.90	9.40	10.16	9.44	1.07	G × P, S
Leaf fat, %	1.56	1.56	1.15	.94	2.14	2.11	1.16	.85	1.90	1.73	1.47	1.15	.41	G × P, S
Longissimus muscle (10th rib)[c]														
Area, cm^2	29.39	34.36	33.69	36.90	14.11	15.53	17.60	18.58	25.98	31.70	31.24	33.84	4.29	G123, P, S
Area, cm^2 [d]	28.34	32.75	31.90	35.04	16.89	18.45	19.64	21.74	25.17	30.45	29.43	32.66	4.09	G123, P, S
Temperature, °C[e]	7.10	7.94	6.12	5.99	2.71	3.31	2.02	.51	6.31	8.18	5.83	5.10	1.84	G12, P × S
pH[e]	5.61	5.71	5.59	5.63	5.85	5.83	5.69	5.88	5.65	5.65	5.66	5.62	.13	G12
Semitendinosus[c]														
Weight, g	326	369	384	423	119	127	148	146	247	282	295	317	36	G123, P, S
Weight, %	.38	.41	.42	.47	.19	.20	.22	.24	.29	.32	.32	.36	.04	G123, P, S
pH[e]	5.83	6.04	5.78	5.93	5.95	6.15	5.91	5.99	5.82	5.96	5.83	6.00	.19	G12, S
Organs, %														
Heart	.32	.30	.35	.35	.35	.35	.43	.43	.30	.31	.35	.36	.03	G × P
Lung	.59	.50	.75	.60	.50	.57	.77	.70	.49	.65	.70	.76	.14	G × S, P
Liver	1.57	1.45	2.09	1.72	1.65	1.50	2.21	2.07	1.50	1.54	1.89	1.88	.23	G12, P, S
Kidney	.34	.31	.41	.39	.35	.35	.51	.51	.30	.33	.40	.37	.11	G12, P
PVDO[f]	3.69	3.44	4.28	3.86	5.22	5.06	5.62	5.22	3.92	3.80	4.56	4.13	.34	G123, P, S
Spleen	.22	.17	.28	.25	.18	.17	.30	.28	.21	.18	.26	.24	.07	P, S
Pancreas	.13	.13	.15	.14	.17	.16	.18	.17	.13	.13	.15	.14	.02	G12, P
Stomach	.53	.52	.61	.59	.76	.75	.81	.77	.56	.56	.67	.61	.07	G123, P
Small intestine	1.44	1.28	1.67	1.46	1.89	1.82	1.99	1.88	1.41	1.37	1.66	1.57	.18	G12, P, S,
Cecum	.14	.14	.16	.13	.20	.20	.21	.19	.16	.14	.17	.15	.03	G123, S
Large intestine	1.24	1.21	1.42	1.29	2.03	1.96	2.12	1.93	1.45	1.42	1.64	1.41	.17	G123, P × S

[a]D × Wc = Duroc × White composite crossbred; M = Meishan; M × Wc = Meishan × White composite crossbred; Buffer = 0 mg/d somatotropin; pST = 4 mg/d somatotropin; Basal = 0 ppm salbutamol; Salb = 2.75 ppm salbutamol. See Table 6 for the number of pigs representing each mean.

[b]Significant ($P < .05$) main or interactive effects of pST (P), salbutamol (S), and genotype (G [1 = D × Wc vs M; 2 = M vs M × Wc; 3 = D × Wc vs M × Wc]). See Results section for a detailed description of interactions.

[c]Values were obtained at approximately 6 h postmortem unless otherwise noted.

[d]Slaughter weight was included as a covariate.

[e]Time from slaughter to measurement was included as a covariate.

[f]Portal vein-drained organs including spleen, pancreas, stomach, small intestine, cecum, and large intestine.

increased ($P < .01$). Salbutamol decreased ($P < .001$) and pST increased ($P < .001$) PVDO, although salbutamol tended ($P = .09$) to cause a greater reduction in the presence of pST. Individually, proportions of pancreas and small intestine were greatest in M pigs (M vs others, $P < .001$), whereas proportions of stomach, cecum, and large intestine increased as the representation of M increased in the genotype ($P < .03$). Somatotropin increased ($P < .001$) the weights of spleen, pancreas, stomach, and small intestine across genotypes, whereas salbutamol tended to decrease the weights of these organs ($P < .05$, $P = .11$, $P = .13$, and $P < .01$, respectively). Large intestine weight was also interrelated to pST and salbutamol treatment; pigs fed salbutamol and injected with pST had less large intestine mass ($P < .05$) than pigs receiving buffer. Similar findings were observed for cecum weight (pST × salbutamol, $P = .16$), but pST had no effect ($P > .20$) and salbutamol consistently reduced cecum weight ($P < .01$).

Carcass Composition and Accretion Rates. Carcass compositions of moisture, protein, and lipid (Table 8) were not different among genotypes ($P > .17$), whereas ash tended to be higher for M pigs ($P = .09$). Proportions of moisture and protein were increased in carcasses ($P < .001$) by either pST or salbutamol treatment, although salbutamol had comparatively less effect (increases of 5 and 7%, respectively) than did pST (increases of 16 and 18%, respectively). Similarly, carcass lipid content was reduced ($P < .001$) more by pST (−33%) than by salbutamol (−12%) across genotype. Percentage of ash was increased by pST (+14%, $P < .001$) and decreased by salbutamol (−3%, $P < .05$).

Salbutamol elicited differential effects among genotypes for total carcass and protein accretion rates ($P < .03$). The interaction resulted from M pigs not being affected by salbutamol, whereas M × Wc pigs had a greater increase in total carcass accretion compared with D × Wc pigs (+117 vs 71 g/d, respectively). Also, protein accretion was not changed by salbutamol in M pigs, whereas it was significantly improved in M × Wc and D × Wc pigs (+33 and 24 g/d, respectively, $P < .001$). Furthermore, salbutamol increased moisture accretion across genotypes (+60 g/d, $P < .001$), although there was a slight tendency ($P = .16$) for a response similar to those of total carcass and protein accretion. Among genotypes, accretion rates of moisture and protein generally had an inverse relationship and accretion rate of lipid a positive relationship to the amount of M present in the genotype. Somatotropin increased accretion rates of carcass moisture, protein, and ash (115, 35, and 6 g/d, respectively) and decreased that of lipid (146 g/d) independently of salbutamol or genotype ($P < .001$). However, because of the dramatic reduction in lipid accretion, total carcass gain was not significantly affected by pST.

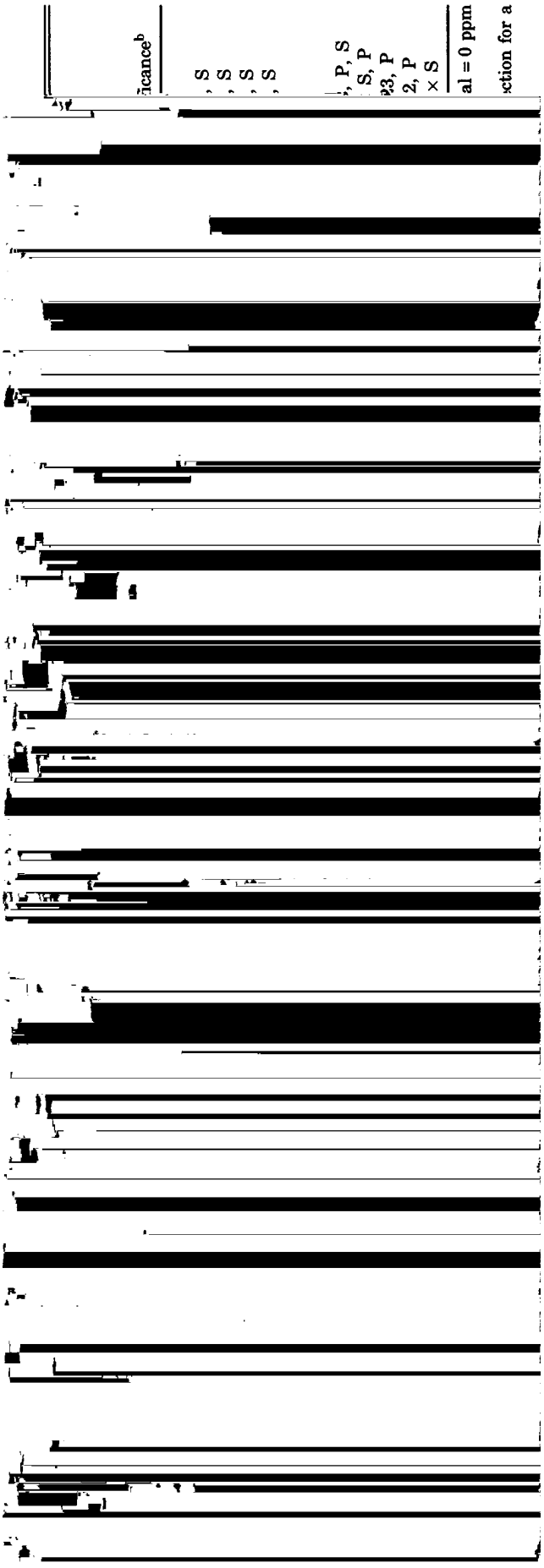
Discussion

We injected a constant amount of pST (4 mg/d), and the calculated delivery based on BW (60 μg/kg) was within an effective dose range (Etherton et al., 1987; Evock et al., 1988; Krick et al., 1992). Also, because growth and tissue deposition can be limited by the amount of nutrients provided by the diet (Campbell et al., 1990, 1991; Caperna et al., 1990, 1991; Goodband et al., 1990), we chose the diet for the present experiment based on a preliminary investigation (Hansen et al., 1994) with a lysine content of 1.2%, which is consistent with previous research (Goodband et al., 1990) for pST-treated pigs. The dosage of salbutamol (2.75 ppm) was based on the findings of Warriss et al. (1990a,b).

The effects of pST on BW change do not provide an adequate estimate of its effectiveness or overall value, nor do they explain what changes are occurring in an individual tissue. This is evident when comparing the change in growth rate (+70 g/d) to the changes in accretion of carcass protein, moisture, lipid, and ash (+35, +115, −146, and +6 g/d, respectively) with pST administration in the present experiment. The net increase in growth rate attributable to the carcass components was only 10 g/d, and the difference of 60 g/d was due to increased organ weight. Caperna et al. (1991), Bidanel et al. (1991), van der Steen et al. (1991), and Krick et al. (1992) found carcass accretion rates that were altered in a similar manner by pST administration in pigs of similar size and type as those in the present experiment. The genotypes used in this experiment demonstrated performance consistent with results of previous research with animals of similar genotype and age (Bidanel et al., 1991; Yen et al., 1991).

Because pST depresses daily feed consumption of animals allowed to eat ad libitum (Knight et al., 1991), the expression of nutrient needs on a grams per day basis is warranted. Somatotropin consistently increased the efficiency of nutrient utilization for protein deposition even when pigs consumed nutrients similar to those consumed by untreated controls (Campbell et al., 1988; Caperna et al. 1991). Also, because an increase in G/F is consistently observed with pST administration, the relative change in efficiency of nutrient utilization for lean tissue deposition is far greater than that for total BW gain.

Evaluation of the interactions observed between genotype and pST indicates that pST elicits its effects predominantly by causing proportional changes in tissue growth. For example, skin weight was increased by pST, but the increase was greater in M pigs, which generally had greater quantities of skin. Although we expressed skin relative to slaughter weight, the relatively high biological activity of skin would cause the tissue to increase proportionally to its weight (i.e., tissues that turn over quickly are greatly influenced



by pST). Likewise, Bidanel et al. (1991) observed that visceral organ weights were consistently increased to a greater degree by pST in M pigs. Our findings support their research, although we found significant interactions only for heart weight. Therefore, the interactions of pST seems to be directly related to the relative size of the tissues it affects in each genotype. Furthermore, alterations may occur in the metabolic end products available at other tissue locations because of shifts in organ output potential, thus providing peripheral tissues with greater quantities of nutrients or biologically active peptides (as suggested by Harris et al., 1990). Many reports have demonstrated that pST administration increases systemic pST and IGF-I (Evock et al., 1988; Buomono et al., 1990; Klindt et al., 1992; Spicer et al., 1992) and IGF-II (Buomono et al., 1990; Klindt et al., 1992; Spicer et al., 1992), which are believed to mediate the effects of pST. Visceral organs represent a greater proportion of the mass of M pigs, and injection of pST has been shown to increase their weight (Bidanel et al, 1991; van der Steen et al., 1991; Klindt et al., 1992); therefore, it is reasonable that a greater amount of endogenous metabolites or hormones may be produced. The differential effects observed for increased O_2 consumption and consequently increased heat production caused by pST are not surprising. The greater increase of visceral organ mass and skin weight in M pigs suggests that basal metabolic rate would follow proportional shifts, as noted by Noblet et al (1992a). Yen et al. (1989) observed that portal vein-drained viscera can account for up to 25% of the energy requirement in feed-deprived swine. Thus, increasing the size of these organs with pST should increase heat production during feed deprivation.

Salbutamol increased live BW, carcass protein, and total carcass weight gain in the crossbred pigs but not in the M pigs. The differential effects on live BW and total carcass gain could have been due to slight alterations in accretion of lipid, moisture, and ash, but most likely they were more closely related to changes in feed consumption. Other researchers have found reduced feed consumption with cimaterol (Jones et al., 1985; Prince et al., 1985; Moser et al., 1986), though Yen et al. (1991) observed improved carcass muscling and ADG without a change in feed intake when feeding ractopamine to pigs of similar genetic makeup. One of the possible reasons why M pigs had a general reduction in feed consumption and depressed growth when fed salbutamol relates to animal handling. Because we obtained a blood sample from the pigs at each weigh date, we observed that M pigs responded negatively to restraint and would go off-feed for several days after bleeding. Also, because LMA, semitendinosus weight, and carcass protein were increased and total carcass lipid content was reduced across genotypes with salbutamol, the change in rate of weight gain most likely was related to feed consumption.

Yen et al. (1991) found that M and crossbred pigs had similar improvements in ADG when fed ractopamine, but LMA was unaffected, whereas we demonstrated an improvement in the latter. Also, Yen et al. (1990a) observed that lean and obese pigs of predominantly U.S. ancestry fed ractopamine had similar improvements in carcass leanness, as in the present experiment, but showed no differences in ADG. This may be a result of different porportions of muscle fiber types among genotypes, or possibly M pigs have a different reactivity to β-adrenergic agonists compared with Western breeds. Genotypes of pigs vary with respect to the density of β-receptors in different tissues (Böcklen et al., 1986), and the response could be related to the density of these receptors in organs that influence rate of growth. This seems most likely based on the interactions between salbutamol and genotype that caused greater improvements in performance or carcass traits of M × Wc pigs than of D × Wc pigs, whereas salbutamol treatment did not affect M pigs. Hence, a possible synergistic effect could be occurring with respect to receptor type and density for this genotype because of the divergent genetic bases of M and Western breeds. Obviously, characterization of specific responses at the cellular level would allow a more complete understanding of the mechanisms by which salbutamol elicits its differential effects.

The combination of pST and salbutamol resulted in additive increases in carcass protein and water. Furthermore, it brought about additive improvements in LMA and semitendinosus weight. Though these responses were not entirely expected, it is reasonable to believe that the mechanisms by which each elicits its effects are different. Whereas pST increases organ mass, salbutamol tends to reduce it, suggesting that salbutamol increases protein mass locally at the tissue level. Alternatively, receptors for pST are found on skeletal muscle but pST effects seem to be more indirect through organs or endocrine means (Louveau and Etherton, 1992). β-Adrenergic agonists are known to stimulate the release of growth hormone (Perkins et al., 1983, 1985; Beermann et al., 1987), which further suggests that more interactions should have been observed in the present study. However, systemic changes in endocrine profiles seem to be of less importance than the direct receptor-mediated effects in the skeletal muscle or adipose tissue (see review by Pell and Bates, 1990).

Adipose tissue seemed to be affected to a lesser degree than muscle tissue with salbutamol administration. Although several reports (Fiems, 1987; Rule et al., 1987; Mersmann and McNeil, 1989) have implicated β-adrenergic agonists in altering adipocyte metabolism, chronic exposure may cause profound metabolic alterations. Webster et al. (1986) suggested that insulin binding in skeletal muscle can be enhanced via a β-adrenergic receptor, which would partially explain the less pronounced effects of β-adrenergic agonists on lipid compared with protein (Jones et al., 1985; Walker et al., 1989; Yen et al., 1990b, 1991). The interaction between pST and salbutamol for large intestine weight is unclear and cannot be explained at present. However, the interaction for longissimus temperature is believed to be directly related to degree of lipid covering the longissimus of pigs. Possibly, different lipid deposits in pig carcasses are more responsive to salbutamol. Yen et al. (1991) did not find a response to ractopamine for fat depth in genotypes similar to those used in this study. Thus, characterization of the receptors and the effects of chronic exposure to β-adrenergic agonists on them should help our understanding of the variation that occurs.

In summary, individual and combined use of pST and salbutamol increased the relative distribution of muscle in the carcass of finishing pigs at the expense of lipid. Although rates of tissue accretion were inconsistent across genotypes, the growth modifiers did not interact in any way that depressed growth. Also, interactions were observed between genotype and pST for several visceral organs, but these effects seem to be more related to the relative proportion that the organs represent of the live BW. Our data support the combined use of these compounds to improve carcass lean composition; however, growth rate and some carcass traits may vary among genotypes with salbutamol and pST treatment.

Implications

The concomitant use of porcine somatotropin and salbutamol resulted in additive increases in carcass protein and water accretion in crossbred genotypes, but not in purebred Meishan pigs. Although porcine somatotropin caused greater reductions in carcass lipid than did salbutamol, the combined effects of these two growth modifiers were additive and consistent with regard to genetic background. The combined use of these compounds should increase lean tissue deposition at the expense of lipid in a wide variety of swine. However, salbutamol may act differentially in various genotypes of swine.

Literature Cited

AOAC. 1984. Official Methods of Analysis (14th Ed.). Association of Official Analytical Chemists, Washington, DC.

Bates, P. C., and J. M. Pell. 1991. Action and interaction of growth hormone and the β-agonist clenbuterol, on growth, body composition and protein turnover in dwarf mice. Br. J. Nutr. 65:115.

Beermann, D. H., W. R. Butler, D. E. Hogue, V. K. Fishell, R. H. Dalrymple, C. A. Ricks, and C. G. Scanes. 1987. Cimaterol-induced muscle hypertrophy and altered endocrine status in lambs. J. Anim. Sci. 65:1514.

Bendall, J. R. 1973. Postmortem changes in muscle. In: G. H. Bourne (Ed.) Structure and Function of Muscle. p 243. Academic Press, New York.

Bidanel, J.-P., M. Bonneau, A. Pointillart, J. Gruand, J. Mourot, and I. Demade. 1991. Effects of exogenous porcine somatotropin (pST) administration on growth performance, carcass traits, and pork meat quality of Meishan, Pietrain and crossbred gilts. J. Anim. Sci. 69:3511.

Böcklen, E., S. Flad, E. Müller, and H. von Faber. 1986. Comparative determination of beta-adrenergic receptors in muscle, heart and backfat of Piétrain and large white pigs. Anim. Prod. 43:335.

Boyd, R. D., and D. E. Bauman. 1989. Mechanisms of action for somatotropin in growth. In: D. R. Campion, G. J. Hausman, and R. J. Martin (Ed.) Animal Growth Regulation. p 257. Plenum Press, New York.

Brier, B. H., and P. D. Gluckman. 1991. The regulation of postnatal growth: Nutritional influences on endocrine pathways and function of the somatotrophic axis. Livest. Prod. Sci. 27:77.

Buonomo, F. C., and J. Klindt, J. T. Yen, and W. G. Pond. 1990. Effects of porcine somatotropin (PST) administration on endocrine parameters in genetically lean and obese swine. J. Anim. Sci. 68(Suppl. 1):292 (Abstr.).

Campbell, R. G., R. J. Johnson, R. H. King, M. R. Taverner, and D. J. Meisinger. 1990. Interaction of dietary protein content and exogenous porcine growth hormone administration on protein and lipid accretion rates in growing pigs. J. Anim. Sci. 68:3217.

Campbell, R. G., R. J. Johnson, M. R. Taverner, and R. H. King. 1991. Interrelationships between exogenous porcine somatotropin (PST) administration and dietary protein and energy intake on protein deposition capacity and energy metabolism of pigs. J. Anim. Sci. 69:1522.

Campbell, R. G., N. C. Steele, T. J. Caperna, J. P. McMurtry, M. B. Solomon, and A. D. Mitchell. 1988. Interrelationships between energy intake and endogenous porcine growth hormone administration on the performance, body composition and protein and energy metabolism of growing pigs weighing 25 to 55 kilograms live weight. J. Anim. Sci. 66:1643.

Caperna, T. J., D. R. Komarek, D. Gavelek, and N. C. Steele. 1991. Influence of dietary protein and recombinant porcine somatotropin administration in young pigs: II. Accretion rates of protein, collagen and fat. J. Anim. Sci. 69:4019.

Caperna, T. J., N. C. Steele, D. R. Komarek, J. P. McMurtry, R. W. Rosebrough, M. B. Solomon, and A. D. Mitchell. 1990. Influence of dietary protein and recombinant porcine somatotropin administration in young pigs: Growth, body composition and hormone status. J. Anim. Sci. 68:4243.

Etherton, T. D., J. P. Wiggins, C. M. Evock, C. S. Chung, J. F. Rebhun, P. E. Walton, and N. C. Steele. 1987. Stimulation of pig growth performance by porcine growth hormone: Determination of the dose-response relationship. J. Anim. Sci. 64:433.

Evock, C. M., T. D. Etherton, C. S. Chung, and R. E. Ivy. 1988. Pituitary porcine growth hormone (pGH) and a recombinant pGH analog stimulate pig growth performance in a similar manner. J. Anim. Sci. 66:1928.

Fiems, L. O. 1987. Review: Effect of beta-adrenergic agonists in animal production and their mode of action. Ann. Zootech. 36: 271.

Goodband, R. D., J. L. Nelssen, R. H. Hines, D. H. Kropf, R. C. Thaler, B. R. Schricker, G. E. Fitzner, and A. J. Lewis. 1990. The effects of porcine somatotropin and dietary lysine on growth performance and carcass characteristics of finishing swine. J. Anim. Sci. 68:3261.

Hansen, J. A., J. L. Nelssen, R. D. Goodband, and J. L. Laurin. 1994. Interactive effects among porcine somatotropin, the beta-adrenergic agonist salbutamol, and dietary lysine on growth performance and nitrogen balance of finishing swine. J. Anim. Sci. 72:1540.

Harris, P. M., G. C. Waghorn, and J. Lee. 1990. Nutritional partitioning of growth for productive gain. Proc. N.Z. Soc. Anim. Prod. 50:81.

Jones, D. J., D. B. Anderson, W. P. Waitt, J. F. Wagner, and D. H. Mowrey. 1989. Effect of ractopamine hydrochloride (RAC) and pituitary derived porcine somatotropin (pPST) alone and in combination on swine growth and carcass parameters. J. Anim. Sci. 67(Suppl. 1):221 (Abstr.).

Jones, R. W., R. A. Easter, F. K. McKeith, R. H. Dalrymple, H. M. Maddock, and P. J. Bechtel. 1985. Effect of the β-adrenergic agonist cimaterol (CL 263,780) on the growth and carcass characteristics of finishing swine. J. Anim. Sci. 61:905.

Klindt, J., F. C. Buonomo, and J. T. Yen. 1992. Administration of porcine somatotropin by sustained-release implant: Growth and endocrine responses in genetically lean and obese barrows and gilts. J. Anim. Sci. 70:3721.

Knight, C. D., T. R. Kasser, G. H. Swenson, R. L. Hintz, M. J. Azain, R. O. Bates, T. R. Cline, J. D. Crenshaw, G. L. Cromwell, H. B. Hedrick, S. J. Jones, D. H. Kropf, A. J. Lewis, D. C. Mahan, F. M. McKeith, C. L. McLaughlin, J. L. Nelssen, J. E. Novakofski, M. W. Orcutt, and N. A. Parrett. 1991. The performance and carcass composition responses of finishing swine to a range of porcine somatotropin doses in a 1-week delivery system. J. Anim. Sci. 69:4678.

Krick, B. J., K. R. Roneker, R. D. Boyd, D. H. Beermann, P. J. David, and D. J. Meisinger. 1992. Influence of genotype and sex on the response of growing pigs to recombinant porcine somatotropin. J. Anim. Sci. 70:3024.

Louveau, I., and T. D. Etherton. 1992. Characterization of somatotropin binding sites in pig skeletal muscle. J. Anim. Sci. 70:1801.

Maltin, C. A., M. I. Delday, S. M. Hay, G. M. Innes, and P.E.V. Williams. 1990. Effects of bovine pituitary growth hormone alone or in combination with the β-agonist clenbuterol on muscle growth and composition in veal calves. Br. J. Nutr. 63:535.

McLaren, D. G., P. J. Bechtel, G. L. Grebner, J. Novakofski, F. K. McKeith, R. W. Jones, R. H. Dalrymple, and R. A. Easter. 1990. Dose response in growth of pigs injected daily with porcine somatotropin from 57 to 103 kilograms. J. Anim. Sci. 68:640.

Mersmann, H. J., and M. D. McNeil. 1989. Lipid mobilization by obese and lean pigs infused with the β-adrenergic agonist isoproterenol. J. Anim. Sci. 67:1992.

Moore, S. 1963. On the determination of cystine as a cysteic acid. J. Biol. Chem. 238 1:235.

Moser, R. L., R. H. Dalrymple, S. G. Cornelius, J. E. Pettigrew, and C. E. Allen. 1986. Effect of cimaterol (CL 263,780) as a repartitioning agent in the diet for finishing pigs. J. Anim. Sci. 62:21.

Nienaber, J. A., and A. L. Maddy. 1985. Temperature controlled multiple chamber indirect calorimeter-design and operation. Trans. Am. Soc. Agric. Eng. 28:555.

Noblet, J., S. Dubois, P. Herpin, and S. Seve. 1992a. Effects of porcine somatotropin on utilization of energy and protein in pigs. Consequences on nutritional requirements. J. Rech. Porcine Fr. 24:237.

Noblet, J., P. Herpin, and S. Dubois. 1992b. Effect of recombinant porcine somatotropin on energy and protein utilization by growing pigs: Interaction with capacity for lean tissue growth. J. Anim. Sci. 70:2471.

Nossaman, D. A., A. P. Schinckel, L. F. Miller, and S. E. Mills. 1991. Interaction of somatotropin and genotype on the requirement for energy in two lines of finishing pigs. J. Nutr. 121:223.

NRC. 1988. Nutrient Requirements of Swine (9th Ed.). National Academy Press, Washington, DC.

Oksbjerg, N, A. Blackshaw, P. Henckel, J. A. Fernández, and N. Agergaard. 1990. Alterations in protein accretion and histochemical characteristics of the M. longissimus dorsi in pigs caused by salbutamol (a β-adrenergic agonist). Acta Agric. Scand. 40:397.

Pell, J. M., and P. C. Bates. 1990. The nutritional regulation of growth hormone action. Nutr. Res. Rev. 3:163.

Perkins, S. N., W. S. Evans, M. O. Thorner, and M. J. Cronin. 1983. Beta-adrenergic stimulation of growth hormone release from perifused rat anterior pituitary cells. Neuroendocrinology 37: 473.

Perkins, S. N., W. S. Evans, M. O. Thorner, D. M. Gibbs, and M. J. Cronin. 1985. β-Adrenergic binding and secretory responses of the anterior pituitary. Endocrinology 117:1818.

Prince, T. J., R. H. Dalrymple, and D. N. Marple. 1985. Effects of feeding cimaterol (CL 263,780) on performance and carcass characteristics of finishing pigs. J. Anim. Sci. 61(Suppl. 1):301 (Abstr.).

Rule, D. C., S. B. Smith, and H. J. Mersmann. 1987. Effects of adrenergic agonists and insulin on porcine adipose tissue lipid metabolism in vitro. J. Anim. Sci. 65:136.

SAS. 1988. SAS/STAT® User's Guide (Release 6.03). SAS Inst. Inc., Cary, NC.

Spicer, L. J., J. Klindt, F. C. Buonomo, R. Maurer, J. T. Yen, and S. E. Echternkamp. 1992. Effect of porcine somatotropin on number of granulosa cell luteinizing hormone/human chorionic gonadotropin receptors, oocyte viability, and concentrations of steroids and insulin-like growth factors I and II in follicular fluid of lean and obese gilts. J. Anim. Sci. 70:3149.

van der Steen, H.A.M., J. van Dijk, P. N. de Groot, and E. Kanis. 1991. Effects of recombinant porcine somatotropin on growth and carcass traits in Meishan pigs. Livest. Prod. Sci. 27:211.

Walker, W. R., D. D. Johnson, J. H. Brendemuhl, R. H. Dalrymple, and G. E. Combs. 1989. Evaluation of cimaterol for finishing swine including a drug withdrawal period. J. Anim. Sci. 67:168.

Warriss, P. D., S. N. Brown, T. P. Rolph, and S. C. Kestin. 1990a. Interactions between the beta-adrenergic agonist salbutamol and genotype on meat quality in pigs. J. Anim. Sci. 68:3669.

Warriss, P. D., S. C. Kestin, T. P. Rolph, and S. N. Brown. 1990b. The effects of the beta-adrenergic agonist salbutamol on meat quality in pigs. J. Anim. Sci. 68:128.

Webster, B., S. R. Vigna, T. Paquette, and D. J. Koerker. 1986. β-Adrenergic modulation of insulin binding in skeletal muscle. Am. J. Physiol. 250:E198.

Yen, J. T., H. J. Mersmann, D. A. Hill, and W. G. Pond. 1990a. Effects of ractopamine on genetically obese and lean pigs. J. Anim. Sci. 68:3705.

Yen, J. T., H. J. Mersmann, J. A. Nienaber, D. A. Hill, and W. G. Pond. 1990b. Responses to cimaterol in genetically obese and lean pigs. J. Anim. Sci. 68:2698.

Yen, J. T., J. A. Nienaber, D. A. Hill, and W. G. Pond. 1989. Oxygen consumption by portal vein-drained organs and by whole animal in conscious swine. Proc. Soc. Exp. Biol. Med. 190:393.

Yen, J. T., J. A. Nienaber, J. Klindt, and J. D. Crouse. 1991. Effect of ractopamine on growth, carcass traits, and fasting heat production of U.S. contemporary crossbred and Chinese Meishan pure- and crossbred pigs. J. Anim. Sci. 69:4810.