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The effect of lean growth rate on puberty attainment in gilts[1,2]

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ABSTRACT: Two hundred sixteen prepubertal Genex Manor hybrid F_1 gilts were used to determine the impact of lean growth rate on sexual development of gilts. This study was composed of two experiments (Exp. 1 and Exp. 2). In Exp. 1, at approximately 96 d of age and 54 kg weight, gilts were allocated with respect to growth rate and litter origin to one of two dietary treatments: 1) a diet formulated to maximize lean growth potential (LP; n = 84) or 2) a diet formulated to produce a lower lean growth rate (LL; n = 84). In Exp. 2, at approximately 88 d of age and 50 kg weight, gilts were allocated with respect to growth rate and litter origin to one of two dietary treatments: 1) a diet formulated to maximize lean growth potential (LP; n = 24) or 2) a diet formulated to restrict lean growth further than was achieved in LL in Exp. 1 (RL; n = 24). All gilts were fed treatment diets for ad libitum consumption and housed in groups of six. Weight, backfat depth and loin depth, and feed intake were measured weekly. Starting at 135 d of age, gilts received 20 min of direct daily exposure to a boar as a pen group for pubertal stimulation. Puberty attainment was determined as the day gilts first exhibited the standing reflex in response to contact with a boar. At pubertal estrus, body weight, backfat depth, and loin depths were recorded. Diet affected ($P \leq 0.05$) estimated fat-free lean gain (LP, 424 vs LL, 347 g/d, Exp. 1; LP, 397 vs RL, 376 g/d, Exp. 2) during the growth period (start to stimulation). However, age at puberty was not affected by diet (LP, 157.3 vs LL, 157.6, Exp. 1; LP, 166.7 vs RL, 167.3, Exp. 2) or overall lean growth at stimulation ($P \geq 0.05$ in both experiments), confirming that innate variability in sexual development of commercial genotypes, rather than growth performance, determines onset of sexual maturity. A negative correlation between age at puberty and growth rate from 50 kg until puberty ($P \leq 0.05$) (LP, r = –0.40, LL, r = –0.36, Exp. 1; LP, r = –0.64, RL, r = –0.48, Exp. 2) was a consequence of reduced lean tissue growth during the stimulation period in later-maturing gilts.

Key Words: Gilts, Lean, Leptin, Puberty

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Introduction

Relationships between age at sexual maturation and growth have been reported (Kirkwood and Aherne, 1985; Young et al., 1990; Rozeboom et al., 1995). Beltranena et al. (1993) suggested that with acceptable commercial growth rates, onset of puberty is determined by inherent differences in the rate of sexual maturation rather than growth performance. However, growth restriction generally delays puberty attainment in gilts (Le Cozler et al., 1999). Moreover, the reduction in protein mass or an impairment of the metabolic status of the animal (Cia et al., 1998) may result in the failure to reach the optimal protein retention necessary to maintain physiological activity (Gaughan et al., 1997), which may have detrimental effects on subsequent reproductive performance.

However, data on the impact of high lean growth rate on sexual development of gilts are extremely limited. Rydhmer et al. (1992) and Beltranena et al. (1993) suggested that high lean growth rates may delay the onset of puberty. In contrast, Kerr and Cameron (1998) and Cameron et al. (1999) suggested that selection for lean growth rate had no adverse affects on sexual development.

Changes in metabolic status mediate short-term effects on reproductive function in gilts (Booth et al.,

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Table 1. Ingredient and nutrient composition of diets as formulated (form) and as fed (fed). Diets were formulated to maximize lean potential (LP), to produce lower lean growth (LL), or to further reduce lean growth (RL)[a]

	LP		LL		RL	
Treatment	Form	Fed	Form	Fed	Form	Fed
Ingredient composition						
Wheat, %	44.5	—	19.2	—	—	—
Barley, %	4	—	43.8	—	13.8	—
Oats, %	—	—	—	—	45.0	—
Corn, %	8.5	—	—	—	—	—
Clean field peas, %	11.2	—	—	—	5.4	—
Canola meal, %	15	—	15.0	—	—	—
Soy meal, %	7.2	—	—	—	—	—
Wheat millrun (10% CF), %	—	—	17.5	—	29.0	—
Nutrient composition						
ME, Mcal/kg	3.282	—	2.869	—	2.550	—
Crude protein, %	18.98	18.7	16.10	15.2	13.0	13.4
Crude fat, %	7.39	—	3.43	—	4.10	—
Crude fibre, %	4.29	—	7.79	—	9.82	—
Lysine, %	1.11	1.18	0.82	0.84	0.647	0.64

[a]All diets were commercially formulated by Vic Pouteaux, Landmark Feeds, Strathmore, Alberta, Canada.

1994), modified through changes in circulating hormones such as leptin, insulin, and IGF-I (Booth et al., 1994; Foster and Nagatani, 1999). Therefore, measurement of blood leptin, insulin, and IGF-I concentrations at critical time points may give an indication of the metabolic status of gilts receiving diets that would influence the rate of lean and fat deposition.

The objective of this study was to use dietary manipulation to determine the effect of high lean growth rate on puberty attainment in gilts and to characterize those growth components that appear to influence age of onset of puberty.

Materials and Methods

Animals

Gilts were fed a standard grower diet until they reached approximately 50 kg, after which they were assigned to treatment and fed one of two treatment diets until they reached puberty. The NRC (1998) computer model program for predicting nutrient requirements was used to estimate the nutrient requirements for growing gilts. All diets were commercially formulated and manufactured in collaboration with Vic Pouteaux, Landmark Feeds, Strathmore, Alberta, Canada. Each experiment was divided into two phases: growth (50 kg to 135 d) and stimulation (135 d to puberty).

Experiment 1. One hundred sixty-eight prepubertal Genex Manor hybrid F_1 gilts (Genex Swine Group, Regina, Saskatchewan), born to primiparous sows, were assigned to either 1) a diet formulated to maximize lean growth potential (**LP**; n = 84) or 2) a diet formulated to produce lower lean growth, but similar fat growth to LP (**LL**; n = 84) (Table 1). Gilts were housed in either barn 1 (n = 72) or barn 2 (n = 96) in groups of six. Space allowance was calculated as recommended for growing pigs on fully slatted (barn 1) and partially slatted (barn 2) floors (Agriculture and Agri-Food Canada, 1993), with each gilt permitted at least 1.2 m^2 space allowance throughout the entire trial. In barn 1, gilts were housed on fully slatted floors, with natural daylight from overhead skylights. Gilts were stratified by weight, age, and litter origin to treatment. Littermate gilts were only selected from litters that had at least eight pigs born alive, of which five were gilts. Each treatment had two littermates, housed in separate pens. In barn 2, gilts were housed in pens with partially slatted floors, with artificial light from 0700 until 1700. Gilts were stratified by weight, age, and litter. Littermates were allocated across treatment as far as possible. In both barns, all gilts were allowed ad libitum access to water and to treatment diets from two-holed feeders.

Experiment 2. Forty-eight prepubertal Genex Manor hybrid F_1 gilts, born to second-parity sows, were used in this trial. At approximately 50 kg, gilts were stratified by weight, age, and litter origin and assigned to either 1) the same diet formulated to maximize lean growth potential in Exp. 1 (LP; n = 24); or 2) a diet formulated to restrict lean growth potential further than was achieved in LL in Exp. 1 (**RL**; n = 24) (Table 1). Littermates were allocated across treatment as far as possible. Gilts were housed in a sub-section of barn 2 in groups of six, on partially slatted floors with at least 1.2 m^2 of pen space per gilt, with artificial light from 0700 until 1700. Gilts were fed treatment diets for ad libitum consumption from an electronic feeder (Feed Intake Recording Equipment, FIRE, Hunday Electronics, Newcastle, U.K.), which allowed access by only one pig at a time. One week prior to beginning the trial, all gilts were fitted with an ear tag transponder

and became accustomed to the tag and feeder. When gilts visited the feeder, they were identified from an ear tag transponder. Entry and exit times and total amount of feed consumed (kg) were transmitted to a dedicated on-line computer and all data were stored for later analysis.

Stimulation Period

Starting at approximately 135 d of age, pens of gilts were taken to a boar stimulation pen and received 20 min of direct exposure to a boar daily as a pen group for pubertal stimulation. In barn 1, Exp. 1, four mature, vasectomized boars were used in rotation. In barn 2, Exp. 1, 2 mature, vasectomized boars and four intact boars of the same age as the gilts were used in rotation. In Exp. 2, four mature, vasectomized boars were used on a rotational basis. Physical signs of pending estrus, such as redness, degree of swelling, and mucosal discharge from the vulva, were recorded daily during the stimulation period. Puberty attainment was determined as the day gilts first exhibited the standing reflex in response to contact with a boar. Boars were not permitted to breed the gilts. At pubertal estrus, body weight, backfat depth, and loin depths were recorded. A cutoff date of 200 d was established because it is not likely that a producer would keep an unproductive gilt longer than 65 d.

Ultrasound and Weight Measurements

At weekly intervals, commencing at 50 kg body weight until the pubertal estrus, gilts were weighed and backfat depth and loin depth were determined ultrasonically (Ultra Scan 900, Alliance Medical, St-Laurent, Quebec). A single longitudinal scan using a 3.5-MHz linear probe was obtained by placing the transducer probe parallel to the midline of the gilt approximately 5 cm from the midline. The scan was measured from the last rib until approximately the third or fourth last rib. A mark was then placed on the gilt's back with a permanent marker and was still visible the following week, allowing successive scans to be taken from the same location. At the time of ultrasonography, the scan was visually appraised and loin and backfat depth measured. Scans were completed by one of three experienced technicians.

Blood and Feces Collection

To determine metabolic hormone status at the beginning of the stimulation period (135 d), a 5-mL jugular blood sample was taken between 0900 and 1400 from all available gilts during a brief period of nose-snare restraint. Overnight access to feed was not prevented at this critical stage of the study, but feeding patterns dictated that the sample would be obtained in the period after initial food intake that day. All samples were centrifuged at 1,500 $\times$ *g* for 15 min and plasma was decanted and stored at –20°C until analysis for leptin, IGF-I, and insulin concentration.

Based on previous experience in our research group on the effective estimation of metabolic hormone status, a more precisely timed postprandial blood sample was obtained, when possible, to determine metabolic hormone status in the immediate postpubertal period. Gilts in Exp. 1, barn 1, were relocated to a stall on the 1st d of pubertal estrus (d 1). Gilts from Exp. 2 continued to be housed in their original group pen until d 6 after estrus. These gilts were given ad libitum access to feed until d 6, when access to feed was denied from 1600. At 0745 on d 7, each gilt was individually given access to an amount of feed equaling 2 $\times$ maintenance requirements. After 75 min remaining feed was removed and weighed to determine feed consumed. At 0915, a 5-mL jugular blood sample was taken from all gilts during a brief period of nose-snare restraint and processed as described previously. Due to limitations in barn space and layout, this precisely timed postpubertal sample could not be obtained from the gilts in Exp. 1, barn 2.

To ensure that the first observed and recorded estrus was truly the pubertal estrus, fecal samples were taken as an effective, noninvasive technique to monitor corpus luteum function. Every 10 d from the start of puberty stimulation until the gilts achieved their pubertal estrus, a fecal sample was taken from each gilt using the technique of Sanders et al. (1994) on the basis that this would allow for detection of an existing luteal phase in cyclic, but anestrous, gilts. All samples were stored at –30°C until subsequent analysis for progestagen concentration to determine reproductive activity in the gilts. Gilts were considered not to have been prepubertal prior to the first detected estrus if fecal progesterone concentrations were high, and data from these gilts were removed from the analysis.

Radioimmunoassay

For all radioimmunoassays (RIA), all samples were analyzed in duplicate. The sensitivity of the assay was calculated using the following equation: average of the zero binding tube [(Bmax) – 2SD(Bmax)/average (Bmax)] $\times$ 100. Plasma IGF-I concentrations were determined using the homologous double-antibody RIA described by Cosgrove et al. (1992), with modifications relating to the antiserum as described by Novak et al. (2000). One hundred microliters of plasma was initially extracted with 3 mL of acid ethanol. Radio-inert recovery efficiency was 109%. The intraassay CV was 9.7%, and sensitivity of the single assay run was 14.9 ng/mL. Plasma leptin concentrations were determined using the multispecies double-antibody kit assay previously validated by Mao et al. (1999) for use in our laboratory. For the two assays run, the intraassay CV was 9.1%, the interassay CV was 10.6%, and the sensitivity of the assay was 1.6 ng/mL. Plasma insulin concentrations were determined using the homologous double-anti-

Table 2. Number of gilts removed from Exp. 1 and Exp. 2 for various reasons

	Exp. 1		Exp. 2	
Reason	LP	LL	LP	LL
Not pubertal by 200 d	2	3	2	1
Lame	4[a]	1	1	0
Hermaphrodite	1	0	0	0
High fecal progesterone	3	1	0	0
Mistakenly removed from trial	1	0	0	0
Poor health	0	0	0	1

[a]Two gilts were considered lame due to injuries not related to treatment (injury during transportation and stimulation).

body RIA described by Cosgrove et al. (1992), modified with the use of an anti-porcine insulin first antibody purchased from ICN Biomedicals (Aurora, OH) and used at a final assay dilution of 1/350,000. The intraassay CV was 9.2% and the interassay CV was 2.2%. One hundred microliters of plasma was initially assayed. If necessary, high-potency samples giving less than 26% binding were diluted and re-assayed. Sensitivity of the assays was 0.051 ng/mL.

Franz Schwarzenberger (Institut für Biochemie and Ludwig Boltzmann für Veterinärmedizinische Endokrinologie, Vienna, Austria, personal communication) provided the procedure for fecal progesterone analysis. Fecal samples were thawed and thoroughly mixed by hand and 0.5 g of each sample, 0.5 mL water, and 4.0 mL of 88% methanol were added to a screw-topped extraction vial. Tubes were vortexed for 30 min. After centrifugation (2,000 × *g* for 30 min), 0.06 mL of the methanol extract was diluted 1:5 with assay buffer and 0.05 mL of the diluted sample extract was taken to assay. Samples were assayed using an RIA kit (Coat-a-Count Progesterone, Diagnostic Products Corp., Los Angeles, CA). The intra- and interassay CV for the three assays run were 13.4 and 7.0%, respectively. Assay sensitivity was 9.5 pg/tube. Percentage hot recovery was 58.2 ± 5% and data were not corrected for hot recovery. No difference was found between the slope of the standard curve and a control curve established with 0.050, 0.025, and 0.0125 mL of diluted extract per tube.

Statistical Analysis

Live weight, ultrasonic 10th rib backfat depth, and 10th rib loin depth were used as inputs into equations (Allan Schinckel, personal communication) used to predict loin eye area (cm^2), total carcass fat-free lean mass (kg), and total carcass fat tissue mass (kg). Specific equations were used to predict live body composition at weight ranges of 20 to 32, 32 to 40, 40 to 55, 55 to 80, 80 to 100, and 100 to 140 kg (Allan Schinckel, personal communication; Thompson et al., 1996; Wagner et al., 1999).

Data were analyzed in relation to the two experimental phases: growth and stimulation. The growth phase covered the period from the start of the experiment until initiation of boar exposure (~ 50 kg to 135 d). The stimulation phase was from the initiation of boar exposure until detection of pubertal estrus (135 d to puberty). Differences between treatments for weight, backfat depth, and loin depth at the start of each phase and at puberty, and also age at puberty and days to puberty from initial boar exposure, were analyzed using the SAS GLM procedure (SAS Inst. Inc., Cary, NC). In Exp. 1, treatment, barn, pen, and litter were used as class variables, and pen(barn × trt) was used as the error term. In Exp. 2, treatment and litter were used as class variables (Steel et al., 1997). For the analysis of weekly growth characteristics and feed intake over the growth and stimulation periods in Exp. 1, treatment, barn, pen, time, and litter were used as class variables in the analysis; pen(barn × trt) was used as the error term (Steel et al., 1997). For Exp. 2, treatment, time, and litter were used as class variables in the analysis (Steel et al., 1997). If treatment differences were detected, multiple comparisons were performed between least squares means, adjusted by Tukey-Kramer test (SAS, 1990). Relationships between measured characteristics were analyzed using SAS REG (SAS Inst. Inc.). Finally, to detect differences in the number of gilts pubertal and prepubertal at each week after boar stimulation, a chi-square analysis was performed (SAS Inst. Inc.).

Results

General Results

Of the 168 gilts that were allocated to treatment in Exp. 1, a total of 16 animals were removed from the analysis (Table 2). Of the 48 gilts that were allocated to treatment in Exp. 2, a total of 5 animals were removed from the analysis (Table 2). Removals were unrelated to treatments.

Feed Intake and Growth Characteristics

Differences in average daily feed, lysine, and metabolizable energy intake are shown in Tables 3 and 4. In Exp. 1, there was no difference in feed intake ($P > 0.05$) during the growth period; however, LP consumed more lysine and energy than RL ($P < 0.05$). In Exp. 2, even

Table 3. Summary statistics for performance measurements in Exp. 1, for diets formulated to maximize lean potential (LP) and to produce lower lean growth (LL)

				P-value				
Item[a]	LP	LL	Pooled SEM	Trt	Barn	Time	Trt × time	Litter
Feed intake, kg/d								
Growth	2.5	2.6	0.02	—	—	***	—	
Stimulation	2.7	3.1	0.03	**	—	**	—	
Overall	2.6	2.9	0.02	**	—	***	*	
Lysine intake, g/d								
Growth	28.0	21.5	0.20	***	—	***	—	
Stimulation	30.7	25.4	0.33	***	—	**	—	
Overall	29.4	23.6	0.20	***	—	***	—	
Metabolizable energy intake, Mcal/d								
Growth	8.1	7.5	0.06	*	—	***	—	
Stimulation	8.9	8.9	0.10	—	—	—	**	—
Overall	8.5	8.3	0.06	*	—	***	—	
Growth rate, g/d								
Growth	946	863	4.6	**	**	***	—	***
Stimulation	906	859	2.9	*	—	—	***	***
Overall	914	869	2.7	†	*	***	*	***
Fat-free lean gain, g/d								
Growth	424	367	3.9	**	*	***	—	***
Stimulation	347	328	5.7	—	—	***	—	***
Overall	369	339	1.8	†	—	***	*	***
Total fat gain, g/d								
Growth	188	157	2.9	*	—	***	—	***
Stimulation	332	298	3.2	—	—	***	—	***
Overall	280	244	2.2	†	—	***	—	***
Gain:feed								
Growth	0.36	0.33	0.007	†	—	***	—	
Stimulation	0.33	0.30	0.056	*	—	**	*	
Overall	0.35	0.31	0.001	**	—	***	*	

[a]Growth = period from start of trial until 135 d of age; Stimulation = period from 135 d of age until puberty; Overall = period from start of trial until puberty.
†$P < 0.10$.
*$P < 0.05$.
**$P < 0.01$.
***$P < 0.001$.

though RL consumed more feed than LP ($P > 0.05$) during the growth period, LP consumed more lysine and energy than RL ($P < 0.05$).

In Exp. 1, for the 152 gilts that completed the trial, there were no differences ($P > 0.05$) between LP and LL gilts in weight (54.2 ± 0.3, and 53.8 ± 0.3 kg), age (96.1 ± 0.3, and 96.6 ± 0.3 d), backfat depth (11.7 ± 0.3, and 12.1 ± 0.3 mm), and loin depth (34.7 ± 0.7 and 34.2 ± 0.6 mm), respectively, at the start of treatment. Although differences were detected ($P < 0.05$) at the start of puberty stimulation between LP and LL gilts in weight (88.3 ± 0.8 and 85.5 ± 0.8 kg), backfat depth (13.4 ± 0.3 and 12.4 ± 0.3 mm), and loin depth (48.5 ± 0.4 and 45.8 ± 0.4 mm), treatment did not affect ($P > 0.05$) weight (109.3 ± 1.6 and 106 ± 1.6 kg), age (157.3 ± 1.7 and 157.6 ± 1.7 d), backfat depth (16.9 ± 0.5 and 14.3 ± 0.5 mm), or loin depth (51.2 ± 0.6 and 50.6 ± 0.6 mm) at the onset of puberty.

In Exp. 2, for the remaining 43 gilts that completed the trial, there were no differences ($P > 0.05$) between LP and RL gilts in weight (50.0 ± 0.7 and 51.2 ± 0.8 kg), age (88.0 ± 0.8 and 87.3 ± 0.8 d), backfat depth (9.7 ± 0.3 and 10.0 ± 0.3 mm), and loin depth (35.7 ± 0.8 and 35.5 ± 0.9 mm), respectively, at the start of treatment, nor in weight (97.8 ± 1.3 and 98.5 ± 1.4 kg), backfat depth (17.3 ± 0.6 and 15.9 ± 0.6 mm), and loin depth (49.3 ± 0.6 and 50.9 ± 0.6 mm) ($P < 0.05$) at the start of puberty stimulation. Similarly, treatment did not affect ($P > 0.05$) weight (124.8 ± 3.0 and 127.0 ± 3.3 kg), age (166.7 ± 3.4 and 167.3 ± 3.7 d), backfat depth (19.8 ± 0.9 and 19.9 ± 1.0 mm), and loin depth (56.9 ± 1.07 and 57.9 ± 1.2 mm) at puberty.

Over the growth period, dietary treatment induced differences ($P < 0.05$) in whole-body growth rate and fat-free lean gain in Exp. 1 (Table 3) and in whole-body gain, total fat-free lean, and total fat gain in Exp. 2. The effect of litter origin was highly significant ($P = 0.0001$) for all the parameters measured in both experiments.

Table 4. Summary statistics for performance measurements in Exp. 2, for diets formulated to maximize lean potential (LP) and to reduce lean growth (RL)

				P-value				
Item	LP	RL	Pooled SEM	Trt	Barn	Time	Trt × time	Litter
Feed intake, kg/d								
Growth	2.1	2.5	0.02	***		***	**	***
Stimulation	2.5	3.0	0.03	***		***	**	***
Overall	2.3	2.8	0.02	***		***	***	***
Lysine intake, g/d								
Growth	24.0	16.6	0.17	***		***	—	***
Stimulation	28.0	19.8	0.30	***		***	*	***
Overall	26.3	18.5	0.17	***		***	**	***
Metabolizable energy intake, Mcal/d								
Growth	7.0	6.5	0.05	***		***	—	***
Stimulation	8.1	7.7	0.10	†		***	**	***
Overall	7.6	7.2	0.05	**		***	**	***
Growth rate, g/d								
Growth	1,038	969	10.6	***		—	—	***
Stimulation	969	968	7.9	—		**	—	***
Overall	997	963	8.6	**		**	**	***
Fat-free lean gain, g/d								
Growth	397	376	4.6	*		—	—	***
Stimulation	350	346	2.5	—		***	—	***
Overall	368	360	2.2	—		***	—	***
Total fat gain, g/d								
Growth	257	226	3.8	**		***	—	***
Stimulation	368	363	5.0	—		**	—	***
Overall	323	312	3.3	—		***	—	***
Gain:feed								
Growth	0.49	0.39	0.006	***		***	—	**
Stimulation	0.45	0.30	0.030	*		*	—	—
Overall	0.47	0.34	0.0001	*		***	—	—

[a]Growth = period from start of trial until 135 d of age; Stimulation = period from 135 d of age until puberty; Overall = period from start of trial until puberty.
†$P < 0.10$.
*$P < 0.05$.
**$P < 0.01$.
***$P < 0.001$.

Pubertal Data

Although no treatment-related differences in age at puberty were detected in either Exp. 1 or 2 ($P > 0.05$), in Exp. 1 more LP gilts reached puberty within 7 d of initial boar contact than did LL gilts ($P = 0.046$), with no difference in subsequent weeks. In Exp. 2, there was no difference ($P > 0.05$) in the weekly proportion of gilts reaching puberty.

Estimated average lean growth rate from the start of the growth phase until the start of the stimulation phase is plotted against age at puberty in Figure 1. No relationship between age at puberty and estimated lean growth rate during the growth period was detected using either a linear or second-degree polynomial model ($P > 0.05$). Average lean growth rate at puberty was negatively correlated to age at puberty ($P < 0.05$) in both experiments (Figure 2). At puberty, body composition of gilts varied considerably and the plot of fat-free lean mass at puberty represented a normal distribution (Figure 3).

Leptin, IGF-I, and Insulin Concentrations

Total feed intake, total energy intake, IGF-I, leptin, and insulin concentrations on d 7 following the onset of standing estrus are shown in Table 5. In Exp. 1, total feed consumed and insulin concentration differed ($P < 0.05$). In Exp. 2, total energy consumed differed between treatments ($P < 0.05$); no other significant relationships were detected. No significant relationships were detected between plasma concentrations of leptin, IGF-I, and insulin at the time of initiation of boar exposure and age at the onset of puberty ($P > 0.05$) (Figure 4).

Discussion

Puberty has been associated with a critical growth rate (Beltranena, 1992; Rydhmer et al., 1994), fatness (Frisch, 1980; Gaughan et al., 1997), and age (Hughes and Varley, 1980); however, the interactions among these factors are unclear. Although the current trend in pig production places emphasis on selection for lean

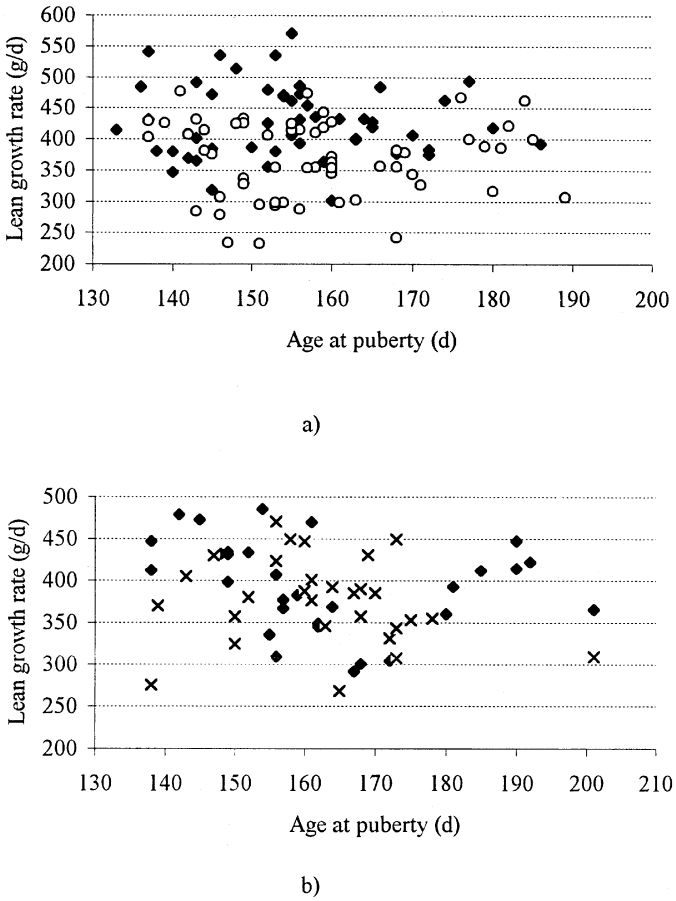

Figure 1. Relationship between average fat free lean gain during the growth period (~ 50 kg to 135 d) and age at puberty. a) Exp. 1, closed diamonds represent LP, open circles represent LL; b) Exp. 2, closed diamonds represent LP, and crosses represent RL.

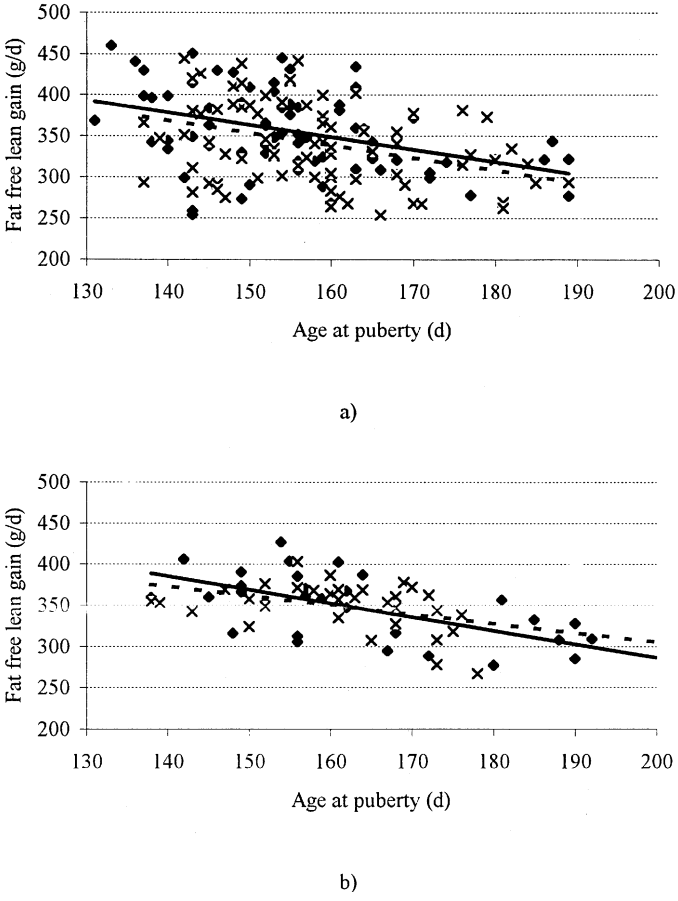

Figure 2. Relationship between fat-free lean gain from start (~ 50 kg) until age at puberty. a) Exp. 1: closed diamonds and solid line represent LP, [Age at puberty = 192.4 – 0.10 (fat free lean gain), P = 0.0005, r = –0.40]; crosses and broken line represent LL, [Age at puberty = 186.9 – 0.86 (fat free lean gain), P = 0.001, r = –0.36]. b) Exp. 2: closed diamonds and solid line represent LP, [Age at puberty = 250.5 – 0.25 (fat free lean gain), P = 0.0001, r = –0.64]; crosses and broken line represent RL, [Age at puberty = 231.7 – 0.20 (fat free lean gain), P = 0.005, r = –0.48].

tissue growth, few data are available concerning the effect of high lean growth rates on sexual development in commercial dam-line gilts. Therefore, to examine the effects of high lean growth rate on puberty attainment, we used dietary manipulation to produce differences in body composition in commercial dam-line gilts in our research herd.

It is well established that method of puberty stimulation (Zimmerman et al., 1998; Patterson et al., 2002), frequency and duration of stimulation (Paterson et al., 1989; Hughes and Thorogood, 1999), and age and sexual behavior of the boar (Levis, 1997; Zimmerman, 1997) are important factors regulating the effectiveness of the boar in puberty onset. For this reason, all gilts were taken to the boar pen for stimulation for a period of 20 min once a day. Pubertal stimulation began at 135 d in this trial, which was earlier than in some other studies (Gaughan et al., 1997; Cia et al., 1998; le Cozler et al., 1999). However, when puberty stimulation was initiated at similar younger ages (140 d by Young et al., 1990; 120 to 130 d by Rozeboom et al., 1995; 130 d by Zimmerman et al., 2000) age at puberty (167.2, 172.5, and 168.0 d, respectively) was similar to the results of the present experiments.

Efficient production closely matches the rate of nutrient intake to the pig's capacity for lean tissue growth (Castell et al. 1994) and daily intake of sufficient protein to meet requirements will depend on the pig's capacity for growth and the concentration of nutrients (Castell et al., 1994). Consequently, maximal lean growth occurs when nutrients and energy are provided in the appropriate ratio (Lawrence et al., 1994). The diets utilized in this study were formulated and manufactured in collaboration with a commercial feed mill (Vic Pouteaux, Landmark Feeds, Strathmore, Alberta, Canada). In contrast, the diets formulated to reduce lean growth contained ingredients that "bulked up" the diet utilizing various commercially available ingredients. Because energy intake and gut-fill capacity generally limit feed

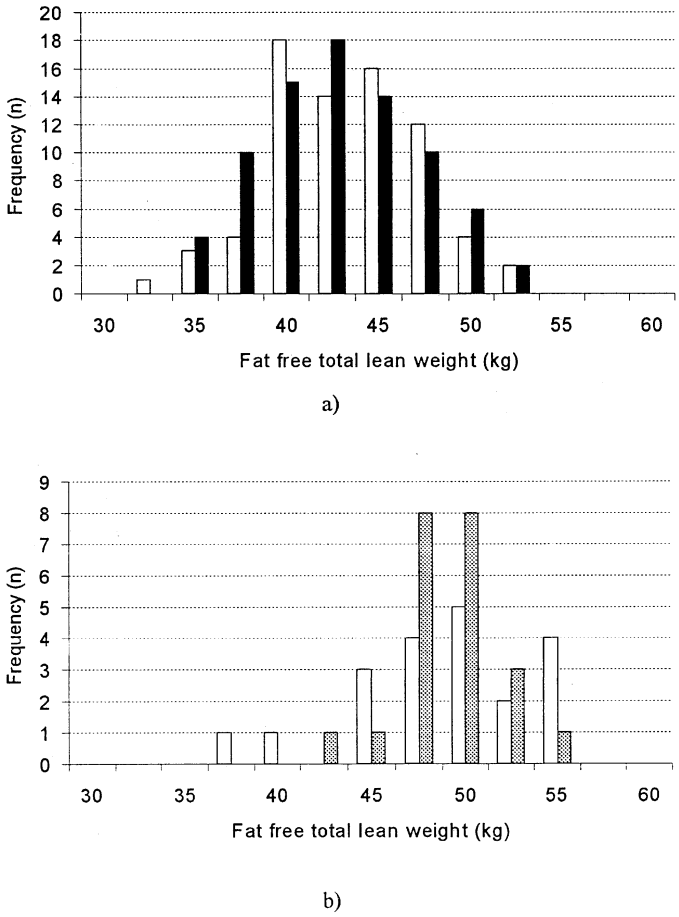

Figure 3. Distribution of fat-free lean mass at puberty. a) Exp. 1; white bars LP, black bars LL; b) Exp. 2, white bars LP, shaded bars RL.

intake in growing pigs (Whittemore, 1998), we hypothesized that maximum gut capacity would limit total feed intake, and energy and lysine consumption would therefore differ between treatments, and differences in lean growth would be observed. Although differences in feed intake were not detected, in Exp. 1, during the growth period, the differences in lysine and energy content of the diets still resulted in differences in total intake of lysine and energy. In Exp. 2, total feed consumed differed between treatments, and, as expected, energy and lysine intake both differed between dietary treatments.

Differences in total feed intake between the common treatment within the two experiments may be related to the method of measuring feed intake. In Exp. 1, average daily feed intake was measured on a group pen basis, whereas in Exp. 2 the FIRE system was used to electronically measure individual feed intake. Also, feed wastage in Exp. 1 may have been higher at the single two-holed feeder placed in the pen as a result of competition to eat. In comparison, use of the FIRE system in Exp. 2 most likely minimized feed wastage, because only one pig had access to the feeder at a time.

Total body, lean, and fat growth rates achieved in this trial were similar to growth rates achieved in other trials. Utilizing finishing crossbred gilts (~ 60 to 100 kg), Grandhi and Cliplef (1997) reported growth rates ranging from 912 to 937 g/d. Similarly, the lean growth rates achieved in this experiment were within the range previously published (491 to 520 g/d, Cameron and Curran, 1995; 429 g/d, Rozeboom et al., 1995; 300 to 350 g/d, NRC, 1998). Mean fat-free lean gain can be used to estimate the daily protein accretion rate curve (Schinckel and de Lange, 1996). Typically, whole-body protein accretion increases from birth to approximately 45 to 65 kg, whereas after 65 kg the percentage of body protein decreases as the percentage of lipid increases (Schinckel and de Lange, 1996). Therefore, the period over which lean growth rate is measured will influence the lean growth rate measured. In this study, the growth period corresponded with the period in which the highest lean growth rates have been reported. Various factors such as stress, environmental conditions, and space allowance, if not ideal, have been shown to have detrimental consequences on the lean tissue growth rates achieved (Schinckel and de Lange, 1996). Hence, in this experiment, care was taken to ensure that ample space was provided for the gilts and that the environment was such that there would be no adverse effects on total body and lean tissue growth rate.

Despite our expectations, based on the diets formulated, differences in estimated lean gain between LP and LR in Exp. 2 were not as large as those between LP and LL in Exp. 1. Also, even though the LP diet was common to the two experiments, LP gilts in Exp. 2 achieved higher overall growth rates than in Exp. 1. This appears to be attributed to overall total fat gain, because overall estimated fat free lean gain was similar. Several factors could have contributed to these differences, including differences in birth weight (1.35 vs 1.41 kg, respectively) and weaning weight (5.65 vs 7.30 kg, respectively) in Exp. 1 and Exp. 2. Differences in piglet birth weight and subsequent differences in growth are most likely due to the difference in parity of the dams. The gilts in Exp. 1 were born to primiparous sows and comparative undernutrition *in utero* may well have contributed to low birth weight, a decrease in muscle fiber number, and a reduction in postnatal growth rate as seen in situations of restricted feeding during gestation (Dwyer et al., 1994). Average daily gain from birth to 25 kg has been correlated with birth weight but not with total muscle fiber number, whereas the converse was true for average daily gain from 25 kg to slaughter (Dwyer et al., 1993). Thus, we suggest that the upper limit to protein deposition may have been higher for gilts from Exp. 2 born to parity-2 sows. The effects of the nursery environment may also limit the expression of the pig's genetic potential for lean growth from 22 to 50 kg (Schinckel and de Lange, 1996). However, given the similar housing and management during the nursery phase of the gilts used in these experiments, the detected differences in growth are un-

Table 5. Total feed offered, total feed and estimated energy consumed, and IGF-I, leptin, and insulin concentration on d 6 after puberty onset, for diets formulated to maximize lean potential (LP), to produce lower lean growth (LL), and to further reduce lean growth (RL)[a]

Item	LP (n = 33)	LL (n = 30)	Pool SEM	*P*-value
		Exp. 1		
Total feed offered, kg	2.2	2.7	0.04	0.0001
Total feed consumed, kg	1.4	1.7	0.06	0.0039
Total energy consumed, Mcal	4.5	4.6	0.2	—
Leptin, ng/mL	2.1	1.9	0.08	—
IGF-I, ng/mL	178.1	179.7	3.6	—
Insulin, ng/mL	2.5	4.8	0.2	0.0001
		Exp. 2		
	LP (n = 20)	RL (n = 21)		
Total feed offered, kg	2.5	3.3	0.04	0.0001
Total feed consumed, kg	1.2	1.1	0.07	—
Total energy consumed, Mcal	3.8	2.9	0.2	0.0368
Leptin, ng/mL	2.1	1.8	0.13	—
IGF-I, ng/mL	193.4	174.8	5.4	—
Insulin, ng/mL	1.2	1.7	0.23	—

[a]75 min after total feed was offered, the remaining feed was removed and weighed to determine feed consumed and a single blood sample was taken to determine leptin, IGF-I and insulin concentration.

likely to be due to the pre-experiment environment the gilts were exposed to. Finally, season may also have been a factor; Exp. 1 was completed March to August and Exp. 2 from November to March.

As in our study, Rozeboom et al. (1995) reported large ranges in weight, backfat depth, growth rate, and lean and fat growth rate at the onset of puberty. In contrast to evidence that selection for lean growth delayed puberty onset in gilts (Rydhmer et al., 1992), our data support the results of Cameron et al. (1999) and suggest that lean growth rate from approximately 50 kg until the initiation of puberty stimulation has no effect on age at puberty. It has been suggested that protein mass may be a regulator of reproductive performance (Cia et al., 1998). In this context Young et al. (1990) speculated that if the attainment of puberty were dependent on achieving a certain threshold of a particular body component (e.g., fat), variation of this component around the time of puberty would not be normally distributed. Using this approach (Rozeboom et al., 1995) determined that achieving a critical threshold for empty protein weight was not a determining factor in the attainment of puberty in gilts. Similarly, we determined that fat-free lean weight at puberty was normally distributed and, therefore, not a critical factor in pubertal onset. All these data are consistent with the suggestions of Foxcroft et al. (1996) that the age at which gilts are first genetically capable of reaching puberty would be achieved well after they have reached the permissive growth rate, body weight, or other component of tissue growth needed to reach sexual maturity.

Age at puberty was negatively correlated to lean growth rate from the start of the trial until puberty, apparently supporting the suggestion that lean growth rate may limit age at puberty (Rozeboom et al., 1995). However, as reviewed by Schinckel and de Lange (1996), protein accretion rates are greatest between 35 to 65 kg body weight, after which protein accretion rates begin to decline. Therefore, protein accretion rates at older ages and heavier weights will be lower than protein accretion rates achieved at younger ages and lighter weights. It is then not surprising that gilts that reach puberty at older ages, due to inherent differences in sexual maturation (Beltranena et al. 1993), will have a relatively lower lean growth rate than gilts that reach puberty at a younger age, if the estimate of lean growth rate includes the variable period from stimulation to first estrus. When inherent age at sexual maturation is removed as a confounding factor, and the relationship between estimated lean tissue growth rate and age at puberty is assessed, as in Figure 1, there is no apparent effect of the rate of lean growth on age at puberty.

Litter origin was highly related to growth characteristics in both experiments and related to age at puberty in Exp. 1. Deligeorgis et al. (1985) suggested that gilts originating from larger litters, litters with higher male:female ratios, and litters with smaller variation in birth weight were more sexually mature at 160 d of age. Litter origin of gilts also affected number of corpora lutea, follicles greater than 5 mm, ovarian and uterine weight, and LH and FSH responses to a GnRH challenge at 55 d of age in the study of Deligeorgis et al. (1985). Recently, Almeida et al. (2001) provided further evidence that litter had significant effects on growth characteristics such as body weight, growth rate, backfat depth, and on reproductive characteristics such as

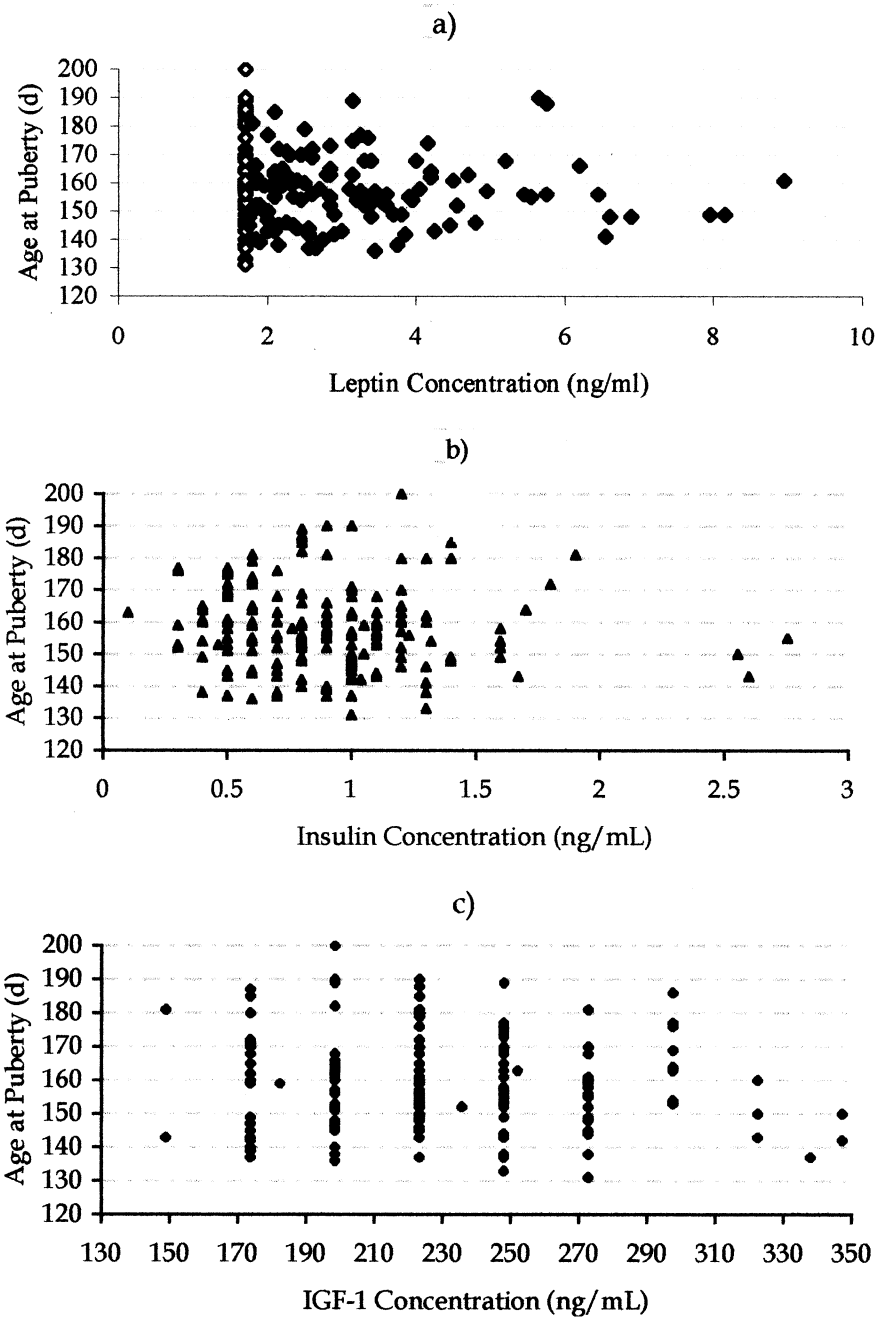

Figure 4. Lack of a relationship between age at puberty and a) leptin concentration at d 135 (age at puberty = 160.2 – 0.6 (leptin concentration d 135), $R^2 = 0$, $P = 0.63$); b) insulin concentration at d 135 (age at puberty = 161.5 – 3.2 (insulin concentration d 135), $R^2 = 0.01$, $P = 0.22$); and c) IGF-I concentration at d 135 (age at puberty = 169.8 – 0.05 (IGF-I concentration d 135), $R^2 = 0.02$, $P = 0.07$). Open symbols in 4 a) indicate leptin concentrations below the sensitivity of the assay.

estrous cycle length, estrus duration, and various endocrine parameters. Similarly, Le Cozler et al. (1999) found that birth litter had a significant effect on several factors including age, weight, and backfat depth at first physiological estrus and at first breeding. Although Foster et al. (1994) suggested that age at pubertal onset may be regulated by a genetic clock that is invariant in its time course of development, the genetic and environmental components of litter effects on age at puberty still need to be clearly defined. As suggested earlier, nutrition of the dam may affect the intrauterine environment and hence fetal and postnatal development, and would represent an environmental, rather than a genetic, effect on age at puberty.

No relationship between leptin concentration and backfat depth was detected in this trial. In contrast to previous reports that leptin concentration at 160 d of age was positively correlated to backfat depth (Pat-

terson et al., 2002), that the amount of leptin secreted from adipocytes is significantly correlated to body mass index and percentage of body fat in humans (Hamann and Matthaei, 1996), and that leptin expression could be associated with subcutaneous fat accumulation in pigs (Robert et al., 1998), no relationships were detected between age at puberty and insulin, IGF-I, or leptin concentrations at 135 d of age. As with phenotypic traits such as weight and growth rate, we interpret these data as indicating that with the lean growth rates achieved in the gilts used in the present experiment, the metabolic determinants of sexual maturation were above the minimum threshold required for effective induction of puberty in response to the pheromonal stimulation from mature boars.

Implications

Our results demonstrate that over the considerable range of lean growth rates achieved during the growth period, there were no adverse effects on age at boar-induced puberty in gilts. Although the consequences of high lean growth rates in replacement gilts on their subsequent performance in the breeding herd still need to be clarified, gilt age or body composition at first breeding are reported not to effect sow productivity or longevity. Therefore, the possible adverse consequences of maximizing lean growth rates in replacement gilts, such as increased mature body weight, increased maintenance costs, housing limitations, and welfare concerns, need careful consideration.

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