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EVALUATION OF A CANDIDATE LOCUS FOR SATIETY IN A FAMILY OF PIGS ORIGINATING FROM LINES DIVERGENTLY SELECTED FOR AVERAGE DAILY GAIN

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Story in Brief

A previous study of lines of pigs divergently selected for postweaning average daily gain (ADG) had revealed that pigs selected for fast ADG (F) expressed a lower concentration of the putative satiety hormone cholecystokinin-8 (CCK-8) per unit of feed consumed than did pigs selected for slow ADG (S). A resource family had been produced by crossing each of two F1 (F x S) boars to 15 unrelated females; 124 (family A) and 115 (family B) offspring were produced in the two half-sib families. A genetic marker for the CCK gene was studied in these families to determine if relationships existed between segregation of the gene and ADG or backfat thickness. Both sires were informative (heterozygous) for the marker, but genotypes were only obtainable for 60 and 36 of the offspring in families A and B, respectively. There was an association of the CCK marker with ADG in family A, but not in family B. Segregation of the CCK marker tended to be associated with backfat thickness in family A, but there was no relationship in family B. These preliminary results indicate that the CCK locus, or linked loci on chromosome 13, may be affecting growth rate. Additional genotypes in family A for CCK and other chromosome 13 markers are needed to verify this result.

Key Words: Pigs, Genome, Growth, Selection, Cholecystokinin

Introduction

Feed intake represents the largest single component of cost in a pork production system. An adequate amount of feed is necessary for lean tissue growth and animal reproduction, but excess feed consumption results in wasteful deposition of fat. A moderate amount of genetic variation exists for feed intake, making it a viable target for optimization through selection, but individual feed intake is expensive to measure. Identification of some of the many genes responsible for genetic variation in feed intake and related tissue growth may lead to molecular information that can be used to assist in traditional selection methods.

Ten generations of selection in pigs were conducted at Oklahoma State University for either fast (Line F) or slow (Line S) postweaning average daily gain (ADG). The resulting lines differed significantly in ADG (F > S), average daily feed intake (F > S) and average backfat thickness at market weight (F > S). Because a primary difference between the lines was feed intake, the protein hormone cholecystokinin-8 (CCK-8), a putative satiety

hormone, was previously studied in the lines (Clutter et al., 1998). Concentration of CCK-8 per unit of feed consumed was less in F than in S. Our laboratory has also developed a genetic marker for the CCK gene (Clutter et al., 1996). The objective of the present study was to determine if an association exists between the CCK locus and genetic variation for ADG and backfat thickness in this population of pigs.

Materials and Methods

Animals and Phenotypes. Lines of pigs were established at Oklahoma State University in 1981 to evaluate responses to divergent selection for postweaning ADG. Ten generations of single-trait divergent selection were completed for either fast (Line F) or slow (Line S) ADG. After generation 10, pigs from F consumed ~36% more feed per day, grew ~47% faster and had ~13% more backfat than pigs from S (Clutter et al., 1998).

The F and S lines were crossed to produce a resource family in which gene searches could be carried out. Two F1 (F x S) sires were mated to a total of 29 unrelated dams to produce two half-sib families (124 and 115 offspring from sires 1 and 2, respectively) in which alleles that differ between the F and S lines are expected to be segregating. All progeny were weighed at weaning (28 d) and just before slaughter (approximately 105 kg) to determine postweaning ADG. Backfat at the 10^{th} rib (BF) was measured on cold carcasses 24 hr after slaughter, as an indicator of body composition.

Genotyping. A restriction fragment length polymorphism (RFLP) generated using polymerase chain reaction (PCR) had been previously developed in our laboratory as a marker of the CCK gene (Clutter et al., 1996). The F1 sires, their mates and offspring in families A and B were genotyped for this marker.

Statistical Analysis. One sire allele of the CCK marker was arbitrarily designated as "1" and the alternative allele as "2". Paternal allele inheritance was coded as the probability of receiving allele "1" from the sire. For each phenotypic trait (ADG and BF), the following model was used to detect effects associated with alternative marker alleles by regressing phenotype on the probability of receiving the designated allele:

$$Y_{ij} = \mu + L_i + S_j + \beta_{wwt} X_{wwt} + \beta_{marker} X_{marker} + e_{ijk}$$

where Y_{ij} = j^{th} ADG or BF observation from the I^{th} litter, μ = the population mean, L_i = effect of the I^{th} litter, S_j = effect of the j^{th} sex, β_{wwt} = regression coefficient of ADG on weaning weight, X_{wwt} = weaning weight used as a covariate, β_{marker} = regression on the probability of inheriting marker allele 1 from the sire, X_{marker} = probability of inheriting marker allele 1 from the sire, and e_{ijk} = random error.

Results and Discussion

Both sires were informative (heterozygous) for the CCK marker, but genotypes were only obtained for 60 and 36 of the offspring from families A and B, respectively. The relatively large region of the CCK gene amplified for this marker (approximately 3570 base pairs) may explain the difficulty experienced in obtaining product from many of these samples.

Results from the regression analyses are presented in Table 1. Because of the limited number of genotypes obtained, results must be considered preliminary. There was an association of the CCK marker with ADG in family A ($P<.05$), but not in family B ($P>.60$). Segregation of the CCK marker tended ($P<.10$) to be associated with backfat thickness in family A, but there was no relationship in family B ($P>.80$).

The CCK gene resides on chromosome 13 in the pig (Clutter et al., 1996). A previous scan of the entire genome in these same half-sib families for loci affecting ADG (Casas et al., 1997) did not detect significant affects associated with chromosome 13. However, it is possible that the location and informativeness of the markers used in that scan prevented the detection of effects in the region of CCK. These preliminary results indicate that the CCK locus, or linked loci on chromosome 13, may be affecting growth rate. Additional genotypes in family A for CCK and other chromosome 13 markers are needed to verify this result.

Literature Cited

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Table 1. Relationships of a marker for the CCK gene with postweaning average daily gain (ADG) and 10th rib backfat thickness (BF).

Phenotype	Probability of a Type I error[a]	b-value[b]	Standard error
Family A:			
ADG	.02	.032 kg/d	.014
BF	.10	.52 cm	.31
Family B:			
ADG	.65	.014 kg/d	.022
BF	.85	.13 cm	.51

[a]Probability of rejecting the hypothesis that the regression is equal to zero, when in fact it is

true.

[b]Regression of phenotype on probability of receiving designated sire allele.

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