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QTL MAPPING FOR FEED CONVERSION EFFICIENCY ON PORCINE CHROMOSOME 10 IN AN AUSTRALIAN COMMERCIAL POPULATION

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SUMMARY

A genetic linkage map of 23 markers on porcine chromosome 10 was constructed with a resource pedigree based on an Australian commercial pig population. Six new markers (UMNP885, UMNP1049, UMNP875, UMNP925, UMNP876 and UMNP519) were linkage mapped to porcine chromosome 10 for the first time. Phenotypes were available for juvenile IGF-I along with feed intake, average daily gain and feed conversion ratio (FCR) recorded over a 6 week performance test period; associations between QTL and residuals for these traits were investigated. A significant QTL for FCR was found between marker UMNP875 and UMNP925 by single family and across family analysis with maximum likelihood using composite interval mapping, confirmed by linkage disequilibrum and linkage analysis (LDLA). In single family analyses, a significant QTL for average daily gain was found in sire family 896TS and an IGF-I QTL was found in sire family 52103. The study provided strong justification for further fine mapping and positional cloning of causative genes for FCR on chromosome 10 for marker assisted selection in pig breeding.

INTRODUCTION

In the Australian pig industry, feed costs account for ~60% of the costs of production (Henman 2003). All other things being constant, the less feed that it takes to bring an animal to market weight, the more profitable the enterprise will be. Many pig breeders would like to include feed efficiency in their breeding programmes. However, feed efficiency measurement is difficult and expensive, since individual feed intake must be recorded. Gene markers for feed efficiency would be very useful as alternative methods for improving feed efficiency without the need to measure feed intake individually. Initially QTL (quantitative trait locus) mapping in pigs used crosses between divergent breeds to find chromosome regions that affect particular traits. Since the first publication of a QTL detected with a cross of European Wild Boar and Large White (Andersson *et al*. 1994), 1831 QTLs have been reported in the PigQTL database from 113 publications representing 317 different pig traits (http://www.animalgenome.org/cgi-bin/QTLdb/SS/summary) on 28 March 2009. Eight QTLs for feed conversion ratio have been reported on chromosomes 3, 4, 5, 6, 8, 13 18 X (Geldermann *et al*. 2003; Lee *et al*. 2003; Stratil *et al*. 2006). This paper reports the first QTL for feed conversion ratio mapped in an Australian commercial pig population from an extensive QTL mapping project funded by Australian Pig Research and Corporation (unpublished).

* AGBU is a joint venture of the NSW Department of Primary Industries and the University of New England

MATERIALS AND METHODS

Data collection. An Australian resource population consisting of 430 progeny of eight sires was bred for QTL mapping at QAF Meat Industries, Corowa, NSW, Australia, between 1999 and 2001. The animals were from two closed lines of Large White and Landrace origin. Daily feed intake was recorded during the 6 week performance test period (from 18-24 weeks). Animals were single penned and fed *ad-libitum*. Weight of the animal was recorded at the beginning of the testing period and shortly before slaughter at 24 weeks. Average daily gain (ADG2), average daily feed intake (ADFI) and feed conversion ratio (FCR) were calculated from the performance test data. Blood was collected 3-5 days after weaning and juvenile pigs for concentrations of insulin-like growth factor-I (IGF-I) were carried out by Primegro (see Bunter *et al.* 2005). Residuals, or phenotypes corrected for non-genetic systematic effects, were used for the association study.

Markers and map construction. A total of 26 markers were genotyped on pig chromosome 10 for the resource pedigree (SW830, SWR136, SW249, SW767, SW1894, SW2491, UMNP885, UMNP1049, UMNP875, UMNP925, SW2195, SWC19, SW173, KS115, S0070, UMNP876, ACO1, SWR1849, UMNP519, SW1041, SW1405, SW1991, SW1626, UMNP104, UMNP599 and UMNP238)**.** The linkage map was constructed using CRIMAP (Green *et al.* 1990) with options FIXED, FLIP and CHROMPIC.

QTL analysis with maximum likelihood using composite interval mapping. A segregating QTL in a sire family causes a phenotype contrast between progeny inheriting alternative QTL alleles Q and q when the sire is heterozygous for the QTL (Qq). At a given map position, genotypes of two flanking markers were used to calculate prior probabilities for progeny having inherited the Q or q allele. As the linkage phases between markers and QTL cannot be considered consistent across families, QTL analyses were performed separately for each sire family. Maximum likelihood estimates were obtained by using the expectation/conditional maximization (ECM) (Zeng 1994). The QTL was tested at every 2 cM along the chromosome with chromosome-wide critical value obtained by the empirical threshold determined by 1000 permutations at each point.

QTL analysis with linkage disequilibrum and linkage analysis (LDLA). Marker haplotypes of sires and dams were reconstructed from progeny genotypes. For the midpoint of each marker interval (putative QTL locations), a matrix of the probability of identity by descent (IBD matrix) was constructed among the base haplotypes using the LD method of Meuwissen and Goddard (2001). This method requires an assumption to be made about the effective size of the population The average value of the effective population size was calculated from chromosomal segment homozygosity (CSH) which is defined as the probability that two gametes drawn at random from the population carry homologous chromosome segments descended from the same common ancestor. The IBD probability was then used in a covariance structure were you fitted random QTL effects.

RESULTS AND DISCUSSION

Genetic Linkage map. Most markers showed significant linkages to other markers by pair-wise linkage analysis. Markers UMNP104, UMNP599 and UMNP238 were excluded from the final genetic linkage maps because there were insufficient informative genotypes to determine their map positions (Table 1). The genetic linkage map of chromosome 10 derived from this data is

significantly bigger than the USDA-MARC map (v2). However, the order of the markers in our study was consistent with the map of USDA-MARC map (v2). Six new markers (UMNP885, UMNP1049, UMNP875, UMNP925, UMNP876 and UMNP519) were linkage mapped for the first time and were consistent with their positions in the physical map of chromosome 10.

Table 1. Linkage map of genotyped markers on porcine chromosome 10

Marker	Position	USDA(2.0) §	Marker	Position	USDA(2.0) §
SW830	0	0.0	SW173	147.7	56.1
SWR136	27.2	7.6	KS115	154.1	58.4
SW249	29.3	17.3	S0070	165.2	62.3
SW767	48.5	20.4	UMNP876*	170.5	63.3
SW1894	48.6	23.2	AC01	175.8	64.3
SW2491	66.4	43.0	SWR1849	179.9	65.1
UMNP885	80.1	43.3*	UMNP519*	189.9	66.5
UMNP1049	97.9	43.6*	SW1041	196.1	67.5
UMNP875	109.1	43.8*	SW1405	196.2	67.5
UMNP925	121.4	44.0*	SW1991	223.5	79.4
SW2195	121.5	44.0	SW1626	269.3	108.0
SWC19	135.3	50.5			

§ map position on MARC-Map; *no linkage map position is available in the MARC-Map; the indicative position was derived from the high resolution IMpRH physical map.

Single family and across family analyses with maximum likelihood using composite interval mapping. The maximum likelihood analysis revealed chromosome-wide significance ($p<0.01$) QTL for FCR at position 112 cM in family 80496 (Table 2). A point-wise significant QTL ($p<0.01$) for FCR was also found in sire family 896TS at position 136. A significant QTL for ADG2 was found at 110 cM in sire family 896TS. Evidence from ADFI, FCR and ADG suggested that a QTL at 110 cM was segregating in this sire family. A QTL for IGF-I levels was found in sire family 52103 at position 122 with chromosome-wide significance. Multi-family analysis revealed QTL for FCR at chromosome-wide significance ($p<0.01$) at position 118 cM (between SW2195 and SWC19). An IGF-I QTL was found at this position with point-wise significance ($p<0.01$).

QTL analysis with linkage disequilibrum and linkage analysis (LDLA). A very significant FCR QTL was found at position 115 ($p<0.01$) between markers UMNP875 and UMNP925, corresponding to USDA-MARC map position 43.9 cM. It was consistent with the finding of single and across family analyses with the maximum likelihood interval mapping. There was a suggestive QTL ($p<0.05$) for ADG2 at position 73.5 between markers SW2491and UMNP875. No significant QTL was found for ADFI. This is the first reported QTL for FCR found in a commercial pig population and no FCR-QTL have previously been reported on porcine chromosome 10. The position and effect were consistent by single family and across family analysis with maximum likelihood using composite interval mapping linkage disequilibrum and linkage analysis (LDLA). It provides strong justification for further fine mapping and positional cloning of causative genes for marker assisted selection in pig breeding.

Table 2. QTL for FCR, ADFI, ADG2 and IGF-I from single trait, single family Maximum Likelihood analyses of chromosome 10

Trait	Family	n	cM	Effect	LRT[1]
ADFI (kg/day)	896TS	45	34	0.25	4.97*
	896TS	45	110	0.21	4.48*
	80475	33	98	0.54	8.59**
	R7292	56	224	0.20	4.92*
ADG2 (g/day)	80393	52	0	88.9	6.63*
	80496	61	66	113	5.48*
	896TS	45	110	127	8.1***
FCR (kg/kg)	80393	51	18	0.17	4.64*
	52103	56	66	0.15	5.15*
	896TS	43	136	0.18	7.42**
	80496	59	112	0.28	21.4****
IGF-I (ng/ml)	80475	33	264	1.61	4.48*
	52103	63	122	2.15	8.46***

n number of progeny in the family;[1] * 5% point-wise significant; ** 1% point-wise significant; *** 5% chromosome-wide significant; **** 1% chromosome wide significant; see text for trait abbreviations

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REFERENCE

Andersson, L., C. S. Haley, H., Ellegren, S. A., Knott, M., Johansson, K., Andersson, L.. Anderssoneklund, I., Edforslilja, M., Fredholm, I., Hansson, J., Hakansson and Lundstrom, K. (1994). *Science* **263**:1771.

Bunter, K. L., S. Hermesch, B. G., Luxford, H. U., Graser H. and Crump, R.E. (2005). *Aust.J.Exp.Agri*.**45**:783.

Geldermann, H., E. Muller, G., Moser, G., Reiner, H., Bartenschlager, S., Cepica, A., Stratil, J., Kuryl, C., Moran, R., Davoli and Brunsch, C. (2003). *J.Anim.Breed.Genet.***120**:363.

Henman, D. (2003) In "Perspectives in pig science" 11-132. editors J. Wiseman, M. A. Varley and B. Kemp

Lee, S. S., Chen, Y., Moran, C., Stratil, A., Reiner, G., Bartenschlager, H., Moser G. and Geldermann, H. (2003). *J..Anim.Breed. Genet.***120**:38.

Stratil, A., Van Poucke, M., Bartenschlager, H., Knoll, A., Yerle, M., Peelman,L.J., M. Kopecny M. and Geldermann, H. (2006). *Anim. Genet.* **37**: 415.

Zeng, Z. B. (1994). *Genetics* **136**: 1457.