Primary genome scan to identify putative quantitative trait loci for feedlot growth rate, feed intake, and feed efficiency of beef cattle

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Primary genome scan to identify putative quantitative trait loci for feedlot growth rate, feed intake, and feed efficiency of beef cattle[1]

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ABSTRACT: Feed intake and feed efficiency of beef cattle are economically relevant traits. The study was conducted to identify QTL for feed intake and feed efficiency of beef cattle by using genotype information from 100 microsatellite markers and 355 SNP genotyped across 400 progeny of 20 Angus, Charolais, or Alberta Hybrid bulls. Traits analyzed include feedlot ADG, daily DMI, feed-to-gain ratio [F:G, which is the reciprocal of the efficiency of gain (G:F)], and residual feed intake (RFI). A mixed model with sire as random and QTL effects as fixed was used to generate an *F*-statistic profile across and within families for each trait along each chromosome, followed by empirical permutation tests to determine significance thresholds for QTL detection. Putative QTL for ADG (chromosome-wise $P < 0.05$) were detected across families on chromosomes 5 (130 cM), 6 (42 cM), 7 (84 cM), 11 (20 cM), 14 (74 cM), 16 (22 cM), 17 (9 cM), 18 (46 cM), 19 (53 cM), and 28 (23 cM). For DMI, putative QTL that exceeded the chromosome-wise $P < 0.05$ threshold were detected on chromosomes 1 (93 cM), 3 (123 cM), 15 (31 cM), 17 (81 cM), 18 (49 cM), 20 (56 cM), and 26 (69 cM) in the across-family analyses. Putative across-family QTL influencing F:G that exceeded the chromosome-wise $P < 0.05$ threshold were detected on chromosomes 3 (62 cM), 5 (129 cM), 7 (27 cM), 11 (16 cM), 16 (30 cM), 17 (81 cM), 22 (72 cM), 24 (55 cM), and 28 (24 cM). Putative QTL influencing RFI that exceeded the chromosome-wise $P < 0.05$ threshold were detected on chromosomes 1 (90 cM), 5 (129 cM), 7 (22 cM), 8 (80 cM), 12 (89 cM), 16 (41 cM), 17 (19 cM), and 26 (48 cM) in the across-family analyses. In addition, a total of 4, 6, 1, and 8 chromosomes showed suggestive evidence (chromosome-wise, $P < 0.10$) for putative ADG, DMI, F:G, and RFI QTL, respectively. Most of the QTL detected across families were also detected within families, although the locations across families were not necessarily the locations within families, which is likely because of differences among families in marker informativeness for the different linkage groups. The locations and direction of some of the QTL effects reported in this study suggest potentially favorable pleiotropic effects for the underlying genes. Further studies will be required to confirm these QTL in other populations so that they can be fine-mapped for potential applications in marker-assisted selection and management of beef cattle.

Key words: beef cattle, feed intake, feed efficiency, quantitative trait loci

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INTRODUCTION

Feed intake and efficiency are economically relevant traits that influence production cost and the environmental sustainability of the beef industry. Improvements in the efficiency of feed use by beef cattle would lead to better economic returns in the whole production system (Archer et al., 1999). Johnson et al. (2003) listed the reasons for the lack of change in beef cattle efficiency, despite several years of intensive production, as including concentration on output traits, inconsistent selection goals, loose and inconsistent definitions of efficiency, and emphasis on population similarities rather than individual variation.

Traditionally, feed efficiency has been measured as feed-to-gain ratio [**F:G**, which is the reciprocal of the

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efficiency of gain (G:F)]. It is correlated with growth and thus has the potential to increase growth rate in young animals (Archer et al., 1999). However, it could also result in substantial increases in the feed consumption of animals in the breeding herd, which may become heavy and expensive to maintain (Archer et al., 1999). Residual feed intake (**RFI**), an alternative measure of feed efficiency, is the difference between an animal's actual intake and its expected intake based on its BW and growth rate over a time period (Koch et al., 1963; Archer et al., 1999). With a moderate heritability, RFI has been shown to have great potential as an index of efficiency for beef cattle (Archer et al., 1999; Herd et al., 2003; Crews, 2005).

Genomic information can be used to increase the rate of genetic progress toward improvement in beef cattle feed efficiency. However, despite its importance, very few attempts at identifying QTL for beef cattle feed intake and feed efficiency have been made in the past (Nkrumah et al., 2005; Moore et al., 2006). Recently, Barendse et al. (2007) reported a whole-genome association study for feed efficiency traits in beef cattle. In this study, we report an autosomal genome scan for QTL affecting growth, feed intake, and feed efficiency in feedlot cattle.

MATERIALS AND METHODS

Animals and Phenotypic Data

The animals used in the study were cared for according to the guidelines of the Canadian Council on Animal Care (1993).

Growth and feed intake data collected over 3 yr (between 2002 and 2005) on beef steers were used in this study. The steers were sired by Angus, Charolais, or Alberta Hybrid bulls. The dams used in the study were produced from crosses among 3 composite cattle lines, namely, Beef Synthetic 1, Beef Synthetic 2, and Dairy × Beef Synthetic (Goonewardene et al., 2003). Cows and heifers for the study were bred in multiple-sire breeding groups on pasture, and the sire of each calf was later determined in a parentage test by using a panel of bovine microsatellite markers.

Details of the procedures for the feedlot tests were given by Nkrumah et al. (2007). Briefly, the steers were managed and tested for feed efficiency under feedlot conditions by using the GrowSafe automated feeding system (GrowSafe Systems Ltd., Airdrie, Alberta, Canada) at the University of Alberta's Kinsella Research Station. The BW of the steers was 353 (SD = 61) kg, and they were 252 (SD = 42) d of age at the beginning of testing. Two tests using approximately 80 steers per test were conducted each year. The test diet in yr 1 was composed of 80.0% dry-rolled corn, 13.5% alfalfa hay pellets, 5% feedlot supplement (32% CP beef mineral supplement containing 440 mg/kg of monensin, trace minerals, and vitamins) and 1.5% canola oil, supplying approximately 2.90 Mcal/kg of DM of ME and 12.5% CP (as-fed). In yr 2 and 3, the same test procedures were used, but the test diet contained 64.5% barley grain, 20% oat grain, 9.0% alfalfa hay pellets, 5.0% beef feedlot supplement, and 1.5% canola oil, supplying 14.0% CP and 2.91 Mcal/kg of DM of ME. Corn was used in yr 1 instead of barley and oats because of a feed barley shortage that particular year.

The traits considered in this study included feedlot ADG, daily DMI, F:G, and RFI. Procedures for obtaining the measures of feedlot F:G have been described previously (Nkrumah et al., 2007). Linear regression with PROC REG (SAS Inst. Inc., Cary, NC) weekly or 2-wk BW measurements against time (d) was used to derive ADG and midtest metabolic BW (**MWT**, $BW^{0.75}$) for each animal. The total feed intake of each animal over a 70-d test period (Arthur et al., 2001a,b) was used to compute the daily DMI. Feed-to-gain ratio was computed as the ratio of DMI to ADG on test. Residual feed intake was calculated from linear phenotypic regression (**RFIp**, Arthur et al., 2001a,b) or genetic regression (**RFIg**, Kennedy et al., 1993; Crews, 2005) of DMI on ADG and MWT. The calculation of RFIp takes into account the phenotypic co(variances) between ADG and MWT, whereas the calculation of RFIg takes into account the appropriate genetic co(variances).

DNA Isolation and Genotyping

A 10-mL blood sample was collected by jugular venipuncture from each steer during the F:G tests, from which genomic DNA was extracted by using a standard saturated salt, phenol-chloroform procedure (Miller et al., 1988). A whole-genome scan covering all 29 BTA was performed with 455 genetic markers. The marker panel used to genotype the steers and their sires consisted of 100 microsatellites and 355 SNP. The approximate locations of the microsatellite markers and SNP, respectively, were based on the observations of Snelling et al. (2005) and McKay et al. (2007). The markers were chosen to be approximately evenly distributed across all 29 autosomes. In addition to chromosomal position, criteria for selection of microsatellite markers were polymorphism information content, sire heterozygosity, number of alleles, and ease of scoring. The criteria for choosing the SNP markers were location and sire heterozygosity. The 455 markers spanned approximately 2,814 cM of the bovine autosomal genome, with an average marker spacing of 6.18 cM and a range of 3.13 cM for BTA 28 to 9.68 cM for BTA 1. The number of markers per chromosome averaged 16 and ranged from 7 on BTA 26 to 33 on BTA 5.

Microsatellite marker genotypes were determined by automated fragment analysis by using the ABI Prism 377 and ABI 3730 DNA sequencers (Applied Biosystems, Foster City, CA). A second examiner evaluated all microsatellite marker genotypes before data analyses. Genotyping of all SNP used in the study was carried out by using the Illumina GoldenGate assay on the BeadStation 500G Genotyping System (Illumina Inc.,

San Diego, CA). The version of the GoldenGate assay used in this study utilizes an allele-specific extension reaction and universal PCR technology to multiplex and genotype simultaneously up to 1,536 SNP loci from approximately 250 ng of genomic DNA (Oliphant et al., 2002; Shen et al., 2005). All marker data were checked for typing errors through an examination of Mendelian segregation.

Statistical Analyses

Phenotypic variances for the traits were obtained with PROC MEANS (SAS Inst. Inc.). Genetic variances were obtained with the statistical software ASREML (Gilmour et al. 2000) by using information that included 813 animals, of which 464 had complete phenotypic records for all traits. A total of 400 progeny belonging to 20 sire families with approximately 20 progeny per sire (range 10 to 56) had genotype and phenotype information available for use in the QTL analyses. Systematic effects considered in the analyses included sire breed, test group (6 levels), and age of steer on test. The study used the multiple-marker, interval mapping approach for half-sib families, as described by Knott et al. (1996). Examples of applications of this methodology in cattle include the work of Spelman et al. (1996), De Koning et al. (1999), and Schnabel et al. (2005).

In this procedure, the QTL allele of interest is arbitrarily assigned to one of the phases of each linkage group within each family. The conditional probability, Q, that a calf inherited the first allele of a putative QTL from its sire is then calculated, based on information from the closest informative flanking markers at 1-cM intervals. The probabilities were obtained by using the Web-based software QTL Express (Seaton et al., 2002). Similar to the approach used in QTL Express (Seaton et al., 2002), an across-family analysis was first carried out by using information from all progeny of all sires. This analysis tests for evidence of the segregation of QTL in the overall experimental population, and the test statistic gives an indication of whether a QTL was segregating in the population for the tested linkage group. No assumptions were made regarding the phase of sires for QTL alleles in the across-family analysis because the QTL effects are nested within sire family and could differ for each family (Ashwell et al., 2004). Subsequent to the across-family analysis, within-family analyses were carried out by using information from the progeny of each sire at a time to determine which sires are potentially segregating for putative QTL for each linkage group.

In both the across-family and within-family analyses, a mixed model fitting a fixed 1-QTL effect with sire effects as random was applied to the data (Nagamine and Haley, 2001; Van Eenennaam et al., 2007). The basic mixed model used was:

$$\mathbf{Y} = \mathbf{X}\beta + \mathbf{Gs} + \mathbf{Q}\alpha + \mathbf{e},$$

where $\mathbf{Y}$ is a vector of observations on the progeny of each sire; $\mathbf{X}$ is a known incidence matrix relating observations to their fixed effect levels; β is a vector of fixed effects, $\mathbf{G}$ relates observations to sires; $\mathbf{s}$ is a vector of random additive polygenic effects of sires; $\mathbf{Q}$ is a vector of the conditional probabilities, at each interval, that a calf inherited the first allele of a putative QTL from the sire based on the flanking marker information; α is the regression coefficient corresponding to the fixed allele substitution effect for a putative QTL (Falconer and Mackay, 1996); and $\mathbf{e}$ is a vector of random residuals. Sires were considered to be unrelated, and thus the present data were not useful for a detailed evaluation of polygenic effects. The random effect of sire was included only in the across-family analyses to account for the expected covariances among paternal half-sibs (Van Eenennaam et al., 2007). The chosen mixed model allowed the data analyses to be carried out by using a macro in SAS, which then permitted the numerous repeated analyses required in the subsequent permutation tests to generate empirical threshold values.

In each analysis, an *F*-statistic profile for a putative QTL was generated at 1-cM intervals along each chromosome (Spelman et al., 1996). The location along the chromosome with the largest *F*-statistic was considered to be the most likely location of a putative QTL. Significance thresholds from an empirical distribution of the *F*-statistic under the null hypothesis of no QTL associated with the chromosome under study were determined by permutations by using a modified version of the method described by Churchill and Doerge (1994). In the permutation tests, the phenotypic records of the 400 progeny were randomly shuffled, while keeping the QTL probability values unchanged, by invoking the CALL RANUNI routine of SAS. The shuffled phenotypic records were then assigned back to the steers and the data were reanalyzed. This process was repeated a number of times to determine critical values for QTL detection. In the current study, 2,000 permutation tests were studied to determine the chromosome-wise 10, 5, and 1% significance thresholds.

Because the genome-wide association study reported by Barendse et al. (2007) was based on single-marker associations based on potential linkage disequilibrium (**LD**) between the markers and possible underlying QTL, the current study is essentially the first report of the results of a genome scan utilizing genetic linkage information to identify QTL for feed intake and F:G. As a result, and based on Lander and Kruglyak (1995), all the detected QTL effects were considered putative. However, to report information that may be useful to other researchers in future studies, an *F*-statistic for a putative QTL that exceeded the chromosome-wise empirical *F*-critical threshold at $P < 0.10$ was reported as weak evidence for a QTL (suggestive QTL), $P < 0.05$ was considered moderate evidence for a putative QTL (significant QTL), and $P < 0.01$ was considered strong evidence for a putative QTL (highly significant QTL). In the within-family analyses, a nominal threshold of

Table 1. Descriptive statistics for the traits considered in the study (n = 464)

Trait	Abbreviation	Mean	SD	Heritability
ADG, kg/d	—	1.46	0.27	0.59 ± 0.17
DMI, kg/d	—	10.45	1.61	0.54 ± 0.15
Feed-to-gain ratio, kg of DM/kg of gain	F:G	7.29	1.26	0.41 ± 0.15
Phenotypic residual feed intake, kg of DM/d	RFIp	0.00	0.88	0.21 ± 0.12
Genetic residual feed intake, kg of DM/d	RFIg	–0.14	1.01	0.42 ± 0.15

$P \leq 0.01$ was used as the criterion to determine whether a sire family was potentially segregating for a QTL for the particular linkage group.

RESULTS AND DISCUSSION

Considerable genetic variation was observed among the steers in the traits analyzed, which made it ideal for QTL identification (Table 1). The genetic and phenotypic relationships among the traits considered in this study are reported in Nkrumah et al. (2007). Strong genetic and phenotypic correlations observed between RFIp and RFIg (r > 0.90) indicated that the 2 indices were very similar (Hoque et al., 2005). Both indices of RFI were also favorably correlated with DMI and F:G. Identification of QTL for the different traits was carried out by using genotype information from 455 markers on 400 steers across and within 20 half-sib families.

Several QTL were detected across families that exceeded the chromosome-wise critical values corresponding to $P < 0.10$, $P < 0.05$, and $P < 0.01$. Chromosomes 4, 13, 25, and 27 did not demonstrate any evidence of QTL influencing the traits considered in the current study. The across-family QTL locations reported in Tables 2, 3, and 4 are based on several sire families, some of which may not be segregating for the QTL. A putative across-family QTL could result from a few sires with highly significant QTL effects or many sires with weak to moderate QTL effects. In the across-family analyses, there were 8 QTL on 5 chromosomes, 33 QTL on 19 chromosomes, and 27 QTL on 18 chromosomes that exceeded the chromosome-wise probability thresholds of $P < 0.01$, $P < 0.05$, and $P < 0.10$, respectively.

Results from the within-family analyses are presented in Tables 5, 6, and 7. The within-family analyses corresponded to contrasts between the 2 putative QTL alleles within a family, and give an indication of the sires that are potentially heterozygous for the QTL. The reported within-family effects may represent a biased list from the study and are by no means the complete list of possibly true within-family QTL effects. The reported effects were selected for reporting based solely on the criterion previously defined (nominal $P \leq 0.01$), and several unreported within-family QTL effects with $P < 0.05$ may indeed be true QTL. In addition, because of the relatively small number of progeny per half-sib family (average n = 20), the reported QTL effects may be generally overestimated, as evidenced by the relatively large SE estimates.

The linkage phase between a marker and a QTL can differ among families (Ashwell et al., 2004), and because the QTL allele of interest was arbitrarily assigned to one of the phases of each linkage group during the calculation of the conditional probabilities, it is not possible to determine unequivocally the phase of each sire. It must also be noted that, in a half-sib model, the most likely position of a putative QTL across families is not necessarily the most likely position of the QTL within families because of differences among families in marker informativeness (De Koning et al., 1999).

Evidence for QTL affecting ADG was detected on 14 different chromosomes. Two, 8, and 4 QTL affecting ADG exceeded the chromosome-wise thresholds of $P < 0.01$, $P < 0.05$, and $P < 0.10$, respectively, in the across-family analyses. The most significant ADG QTL were detected on BTA 16 and 11. Of the ADG QTL detected, those on BTA 6 and 19 were within the same chromo-

Table 2. Across-family QTL locations and test statistics on BTA 1, 2, 3, 5, 6, 7, 8, 10, and 11

BTA	Trait[1]	Location, cM	*F*-statistic[2]
1	DMI	93	4.18*
	RFIg	90	3.34†
	RFIp	90	5.15*
2	RFIg	87	2.79†
	RFIp	92	2.88†
3	DMI	123	4.15*
	F:G	62	4.72*
5	ADG	130	4.11*
	DMI	48	3.08†
	F:G	129	13.42***
	RFIg	129	9.04**
	RFIp	129	6.98**
6	ADG	42	4.81*
7	ADG	84	3.75*
	F:G	27	5.75*
	RFIg	22	4.20*
	RFIp	22	3.82*
8	DMI	78	2.62†
	RFIg	80	5.57*
	RFIp	80	5.81*
10	F:G	31	3.79†
11	ADG	20	9.11**
	DMI	20	2.99†
	F:G	16	4.31*

[1]RFIg = genetic residual feed intake; RFIp = phenotypic residual feed intake; F:G = feed-to-gain ratio. Units are presented in Table 1.

[2]Chromosome-wise significance thresholds, †$P < 0.10$, *$P < 0.05$, **$P < 0.01$, and ***$P < 0.001$, were obtained from 2,000 permutation tests.

Table 3. Across-family QTL locations and test statistics on BTA 12, 14, 15, 16, 17, 18, and 19

Chromosome	Trait[1]	Location, cM	*F*-statistic[2]
12	DMI	93	3.64†
	RFIg	89	4.21*
	RFIp	89	4.75*
14	ADG	74	4.13*
	DMI	88	3.68†
	RFIg	107	3.52†
	RFIp	107	3.76†
15	DMI	31	3.84*
16	ADG	22	7.79**
	F:G	30	4.56*
	RFIg	41	5.36*
	RFIp	42	4.68*
17	ADG	9	4.26*
	DMI	81	5.69*
	F:G	25	5.05*
	RFIg	19	7.73**
	RFIp	18	7.07*
18	ADG	46	6.30*
	DMI	49	6.30**
	RFIg	64	3.39†
	RFIp	64	3.54†
19	ADG	53	4.29*
	RFIg	104	3.09†
	RFIp	100	3.50†

[1]RFIg = genetic residual feed intake; RFIp = phenotypic residual feed intake; F:G = feed-to-gain ratio. Units are presented in Table 1.

[2]Chromosome-wise significance thresholds, †$P < 0.10$, *$P < 0.05$, **$P < 0.01$, were obtained from 2,000 permutation tests.

Table 4. Across-family QTL locations and test statistics on BTA 20, 21, 22, 23, 24, 26, 28, and 29

Chromosome	Trait[1]	Location, cM	*F*-statistic[2]
20	ADG	64	3.64†
	DMI	56	6.47*
	RFIp	55	3.81†
21	DMI	2	3.67†
	RFIp	2	3.75†
22	ADG	67	3.98†
	F:G	72	4.59*
23	ADG	2	3.79†
24	F:G	55	4.95*
	RFIg	6	3.13†
	RFIp	6	2.96†
26	ADG	69	3.35†
	DMI	69	5.33*
	RFIg	48	4.42*
	RFIp	52	5.70*
28	ADG	23	4.85*
	F:G	24	9.47**
	RFIg	24	3.44†
29	RFIg	50	3.40†
	RFIp	50	3.37†

[1]RFIg = genetic residual feed intake; RFIp = phenotypic residual feed intake; F:G = feed-to-gain ratio. Units are presented in Table 1.

[2]Chromosome-wise significance thresholds, †$P < 0.10$, *$P < 0.05$, **$P < 0.01$, were obtained from 2,000 permutation tests.

somal regions previously reported to harbor growth QTL by Kneeland et al. (2004). Eight chromosomes demonstrated evidence of ADG QTL from the within-family analyses that met the criterion of nominal $P \leq 0.01$. The most significant within-family ADG QTL was detected on chromosome 5 in the same region reported by Li et al. (2002). In addition, Kneeland et al. (2004) reported QTL for growth traits on chromosomes 6, 14, 19, and 23, some of which are in the same regions as those reported from the within-family analyses in the current study. Other reports of QTL for growth-related traits include those by Casas et al. (2001, 2003) and Kim et al. (2003).

The study demonstrated evidence of across-family QTL affecting DMI on 14 different chromosomes. Of these, 1, 6, and 6 QTL exceeded the chromosome-wise $P < 0.01$, $P < 0.05$, and $P < 0.10$ significance thresholds, respectively. The most significant QTL for DMI were detected on BTA 1, 18, 20, and 26, with the largest significance effect being the BTA 18 QTL. The within-family analyses provided evidence of QTL influencing DMI on 9 different chromosomes that met the criterion of nominal $P \leq 0.01$, with the most significant effect being the chromosome 14 QTL at 101 cM.

A total of 10 across-family QTL were detected for F:G in the study. The most significant F:G QTL was detected on BTA 5, and it exceeded the chromosome-wise $P < 0.001$ threshold. Of the remaining F:G QTL, 1 QTL on BTA 28 exceeded the chromosome-wise $P < 0.01$ threshold, 7 QTL effects were detected that exceeded the chromosome-wise $P < 0.05$ threshold, and 1 QTL effect exceeded the chromosome-wise $P < 0.05$. A total of 8 chromosomes demonstrated evidence of within-family QTL for F:G ratio based on the stated nominal $P \leq 0.01$ criterion, with the most significant within-family QTL detected on chromosome 7.

Two measures of residual feed intake, RFIg and RFIp (Kennedy et al., 1993), were evaluated for QTL effects, and there were very high levels of concordance between the 2 traits in terms of QTL locations and significance. This is highly reflective of the reported strong genetic correlation between the 2 traits (Hoque et al., 2005; Nkrumah et al., 2007). With the exception of 1 suggestive QTL on BTA 28 and 2 suggestive QTL on BTA 20 and 21, all the QTL detected for RFIp in the across-family analyses were also significant, or at least suggestive, for RFIg, albeit with slight variations in QTL locations. Indeed, the 2 suggestive QTL for RFIp were very close to also being suggestive for RFIg, and the suggestive QTL for RFIg was nearly suggestive for RFIp ($P < 0.20$; data not shown).

Considering the remaining QTL that showed concordance between RFIg and RFIp, the across-family analysis revealed 1 QTL on BTA 5 that was significant for both RFIg and RFIp at the chromosome-wise $P < 0.01$ threshold. An additional QTL on BTA 17 was highly significant for RFIg ($P < 0.01$) and was moderately significant for RFIp ($P < 0.05$). An additional 5 QTL were concordantly significant for both RFIg and RFIp at the chromosome-wise $P < 0.05$ threshold. Additionally, 6

Table 5. Within-family QTL locations and effects on BTA 2, 3, 5, 6, 7, 8, 10, and 11

Chromosome	Trait[1]	Location, cM	Family	Estimate	SE	*P*-value[2]
2	RFIG	7	15	–0.936	0.353	0.01295
3	DMI	11	5	1.854	0.548	0.00214
	F:G	90	3	0.961	0.286	0.00731
5	ADG	67	8	–0.470	0.092	0.00017
	DMI	80	10	2.316	0.774	0.01124
	F:G	47	4	–0.960	0.327	0.01017
	F:G	46	3	0.871	0.288	0.01277
	F:G	75	8	2.748	0.684	0.00127
	RFIP	35	7	1.125	0.330	0.00519
6	ADG	28	7	–0.634	0.190	0.00594
	ADG	69	5	0.017	0.136	0.00418
7	F:G	83	6	1.247	0.403	0.00459
	F:G	19	15	1.250	0.419	0.00585
	F:G	9	11	1.447	0.244	0.00058
	RFIG	35	13	1.316	0.468	0.0132
	RFIP	119	5	–0.756	0.289	0.01419
	RFIP	74	6	1.054	0.324	0.00308
	RFIP	38	13	1.124	0.393	0.01184
8	DMI	61	10	–2.104	0.567	0.00297
	DMI	120	14	1.411	0.440	0.00241
	DMI	17	3	2.534	0.853	0.01404
	RFIP	31	7	–1.245	0.348	0.00382
	RFIP	13	3	1.290	0.402	0.00935
10	F:G	65	13	–2.800	0.857	0.0052
	F:G	77	11	1.833	0.474	0.00614
11	F:G	23	4	1.434	0.509	0.01301

[1]RFIg = genetic residual feed intake; RFIp = phenotypic residual feed intake; F:G = feed-to-gain ratio. Units are presented in Table 1.
[2]Only within-family QTL effects with nominal $P \leq 0.01$ are reported.

Table 6. Within-family QTL locations and effects on BTA 12, 14, 15, 17, 18, and 19

Chromosome	Trait[1]	Location, cM	Family	Estimate	SE	*P*-value[2]
12	RFIG	93	7	–0.956	0.293	0.00684
	RFIP	91	7	–0.912	0.273	0.00596
14	ADG	53	6	0.398	0.100	0.00048
	DMI	101	6	1.422	0.335	0.00023
	RFIG	41	15	2.461	0.597	0.0003
	RFIP	41	15	2.353	0.594	0.00046
15	DMI	36	7	–1.514	0.340	0.00079
	DMI	110	6	1.058	0.318	0.00254
	DMI	107	3	1.985	0.507	0.00287
17	ADG	44	13	0.696	0.251	0.01425
	DMI	85	7	–1.181	0.333	0.00406
	DMI	86	5	–0.892	0.301	0.00522
	F:G	40	13	–3.580	1.147	0.00702
	F:G	83	9	–2.701	0.801	0.01187
	RFIG	4	6	–0.902	0.283	0.00365
	RFIP	71	9	–2.746	0.655	0.00407
	RFIP	5	6	–0.959	0.249	0.00067
18	DMI	63	14	–0.857	0.302	0.00661
	RFIG	5	5	–0.762	0.250	0.00427
	RFIP	5	5	–0.758	0.246	0.00384
19	ADG	25	4	0.303	0.109	0.01378
	RFIG	68	13	–1.753	0.450	0.00144
	RFIG	36	14	0.723	0.217	0.00165
	RFIP	67	13	–1.666	0.376	0.00049
	RFIP	38	14	0.720	0.209	0.00119

[1]RFIg = genetic residual feed intake; RFIp = phenotypic residual feed intake; F:G = feed-to-gain ratio. Units are presented in Table 1.
[2]Only within-family QTL effects with nominal $P \leq 0.01$ are reported.

Table 7. Within-family QTL locations and effects on BTA 20, 21, 22, 23, 24, 26, and 29

Chromosome	Trait[1]	Location, cM	Family	Estimate	SE	*P*-value[2]
20	DMI	24	4	2.320	0.519	0.00045
	RFIP	74	11	–2.002	0.602	0.01265
	RFIP	60	5	1.012	0.369	0.00935
	RFIP	24	4	1.462	0.395	0.00213
21	DMI	54	6	–1.253	0.363	0.00184
	RFIP	74	9	–2.557	0.651	0.00568
	RFIP	39	3	1.269	0.308	0.00208
22	F:G	96	15	–1.174	0.385	0.00501
23	ADG	67	6	–0.393	0.122	0.00334
	ADG	3	5	0.187	0.072	0.01326
	ADG	0	13	0.368	0.129	0.012
24	RFIG	64	14	–0.663	0.200	0.00176
	RFIG	57	7	1.476	0.362	0.00155
	RFIG	6	9	2.035	0.597	0.01132
	RFIP	64	14	–0.624	0.197	0.0027
	RFIP	57	7	1.330	0.329	0.00162
	RFIP	8	9	2.165	0.649	0.01249
26	ADG	61	11	–0.304	0.085	0.00907
	ADG	24	5	0.273	0.075	0.00103
	F:G	52	9	–2.417	0.548	0.00312
	F:G	16	5	–1.137	0.348	0.00287
	RFIG	32	3	–1.144	0.335	0.00654
29	RFIG	45	11	2.081	0.440	0.00214
	RFIP	44	11	2.275	0.501	0.00266

[1]RFIg = genetic residual feed intake; RFIp = phenotypic residual feed intake; F:G = feed-to-gain ratio. Units are presented in Table 1.
[2]Only within-family QTL effects with nominal $P \leq 0.01$ are reported.

suggestive (chromosome-wise $P < 0.10$) QTL were detected for both RFI traits in the across-family analyses. Combining those QTL that are not common to the 2 RFI indices, a total of 17 chromosomes demonstrated evidence of QTL for RFI, with the most significant QTL for both measures occurring on BTA 5. Similar to the across-family analyses, 8 chromosomes provided evidence of within-family QTL effects that were concordant for both RFIp and RFIg based on the criterion of nominal $P \leq 0.01$. In addition, evidence of within-family QTL for RFIp alone was detected on chromosomes 5, 20, and 21, whereas QTL for RFIg alone were detected on chromosomes 2 and 28.

According to Heyen et al. (1999), if the heterozygosity of the QTL in the population is low because of selection, many QTL will be detected in certain linkage groups in the within-family, but not in the across-family, analysis, and this was the case in this study. Typically, not all families are informative for a QTL in an outbred population. There are some instances in which QTL detected in the across-family analyses are not reported among the within-family results, mainly because none of the individual within-family effects for the particular linkage group met the criterion of nominal $P \leq 0.01$. The estimates of QTL effects and test statistics in the across-family analysis are derived from a combined analysis of families that are heterozygous or homozygous for the QTL (Hiendleder et al., 2003). Because the number of heterozygous sires segregating for each particular QTL detected was generally small in relation to the total number of families tested, the combined estimate for the test statistic in the across-family analysis is expected to be considerably lower than in the within-family analysis. In addition, a significant across-family QTL effect could be as a result of either a few sires with highly significant QTL effects or several sires with merely suggestive to moderate QTL effects.

Comparison of the feed intake and feed efficiency QTL results of the current study with other published work, especially in beef cattle, is difficult because there have been very limited attempts at identifying QTL for feed intake and feed efficiency in cattle in general. We have previously reported QTL mapping studies for feed intake and feed efficiency by using data on a subset of the steers presented in the current study (Nkrumah et al., 2005; Moore et al., 2006). Although Pitchford et al. (2002) reported the identification of QTL for feed intake and associated traits, the detailed results from that study are yet to be made public. Recently, Barendse et al. (2007) reported a whole-genome association for feed conversion efficiency traits, but this analysis was based mainly on LD single-marker associations instead of genetic linkage analyses. To avoid inappropriate speculation, we have made no attempts to determine the potential relationships of the LD SNP reported by Barendse et al. (2007) and QTL effects detected in the current study, although it is entirely possible that some of the LD SNP may in fact underlie QTL reported in the current study. In other species, Van Kaam et al. (1999) reported significant QTL for feed intake on chicken chromosomes 1 (234 cM), 2 (41 cM), and 4 (147 cM). Recently, Minvielle et al. (2005), working with Japa-

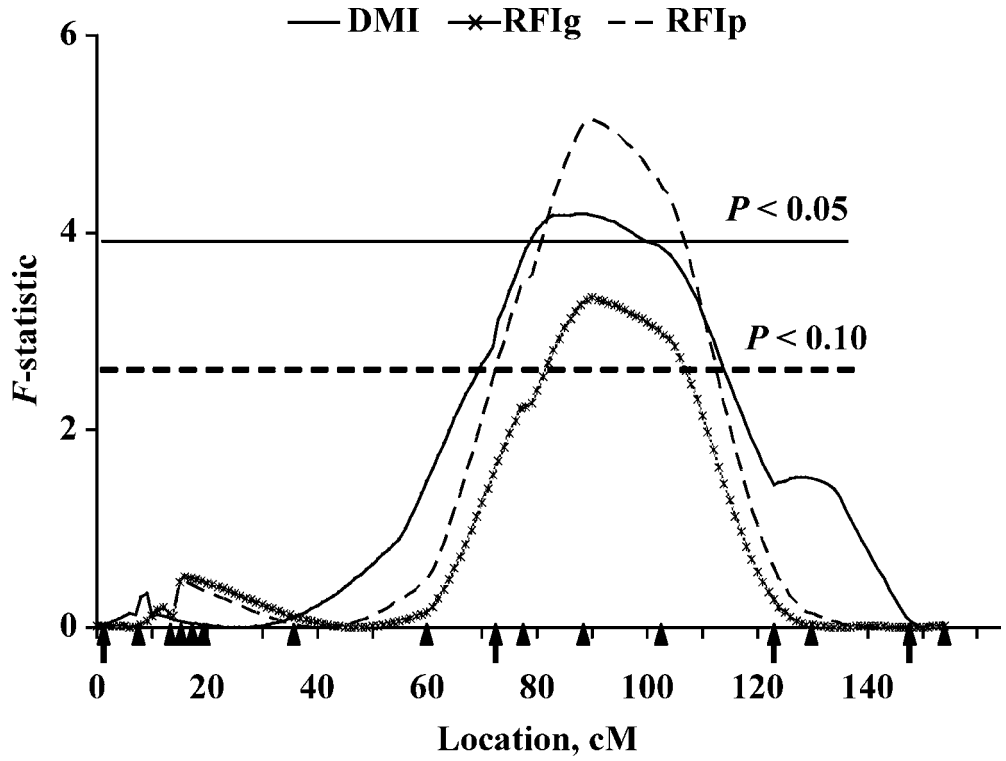

Figure 1. Across-family *F*-statistic profile from across-family analyses for DMI and residual feed intake on chromosome 1. Horizontal lines represent the chromosome-wise threshold values from 2,000 permutations. Relative marker locations are indicated by triangles (SNP) or arrows (microsatellite) on the horizontal axis. RFIg = genetic residual feed intake; RFIp = phenotypic residual feed intake.

nese quail, reported significant QTL for feed intake on chromosome 1 and for feed intake and RFI on chromosome 20. Other reports of QTL detection for feed intake and associated traits include De Koning et al. (2003) and Hansen et al. (2005) in chickens, Houston et al. (2005) in pigs, and Pomp et al. (2006) in mice.

Because of the generally strong genetic correlations among some of the traits considered in the study, as well as those reported in other studies (Arthur et al., 2001a; Herd et al., 2003; Crews, 2005), it is logical for one to expect that some of the QTL detected in the current study will influence several of the traits at the same time. Genetic correlations among traits may result from pleiotropy or may be due to the effects of multiple linked genes that affect the different traits. It may be relevant to determine whether the QTL allele that shows an increasing effect for one trait also shows favorably correlated effects on the correlated traits. That is, a pleiotropic QTL for multiple traits should show significant associations with the relevant traits in the progeny of sires segregating for the QTL in such a way that the direction of the QTL effects will be favorably consistent in all those traits. It is possible to make deductions on potential pleiotropy based on knowledge of the genetic relationships among the traits and the direction and location of the observed QTL effects. Indeed, several QTL in the across-family analyses showed weak to strong significant effects for 2 or more traits in the current study, and in most instances, the potential pleiotropic effects were favorably consistent, albeit with slight differences in the locations along the chromosome where the largest *F*-statistic was detected for specific traits.

Figures 1 to 5 are presented to give a perspective on some graphical summaries of potentially pleiotropic QTL effects observed in this study. For instance, ADG and F:G are negatively genetically correlated (Herd et al., 2003; Crews, 2005; Nkrumah et al., 2007), and considering QTL locations that are either the same or very close, enough for the effects on the 2 traits to be considered as being potentially pleiotropic, there were potentially favorable pleiotropic effects for ADG and F:G on chromosomes 5, 11, 16, 17, 22, and 28. This implies that the putative QTL allele that increases ADG also favorably decreases F:G, because higher F:G values indicate lower efficiencies for growth. Similarly, DMI and RFI are positively genetically correlated (Herd et al., 2003; Crews, 2005; Nkrumah et al., 2007), and there were potentially favorable pleiotropic QTL effects for DMI and RFI on chromosomes 1, 8, 12, 20, 21, and 26. Similarly, the QTL effects for F:G and RFI detected on chromosomes 5, 7, 16, 17, and 28 were potentially reflective of the positive genetic correlation between the 2 traits (Herd et al., 2003; Crews, 2005; Nkrumah et al., 2007). With respect to the relationship between ADG and RFI, it appears from the results of this study

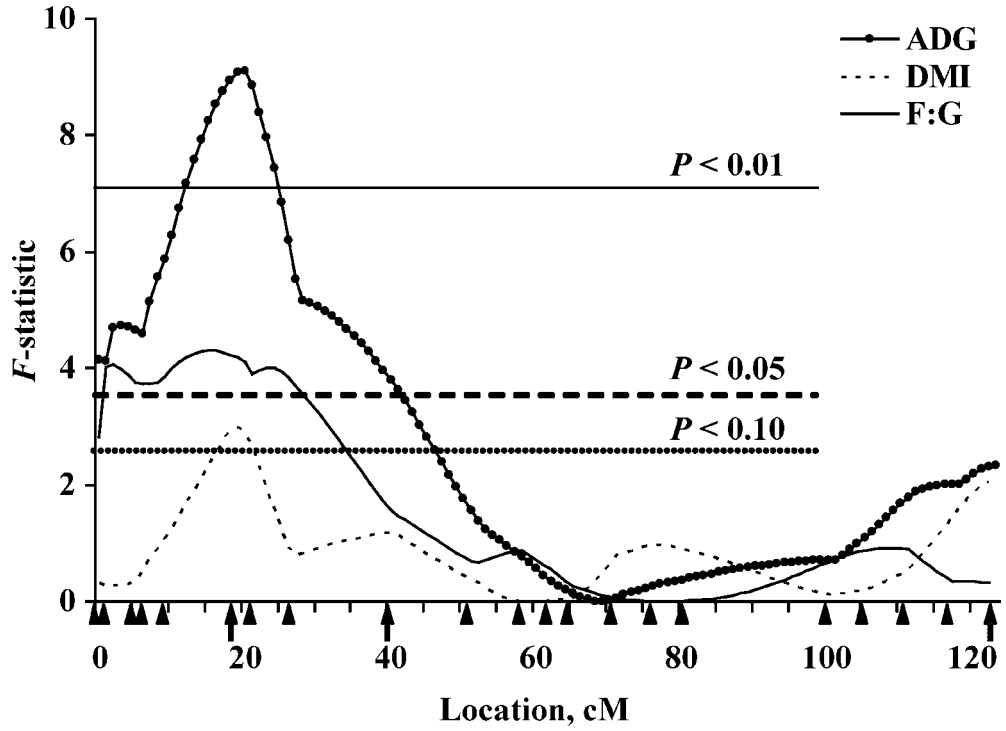

Figure 2. Across-family *F*-statistic profile for ADG, DMI, and feed-to-gain ratio (F:G) on chromosome 11. Horizontal lines represent the chromosome-wise threshold values from 2,000 permutations. Relative marker locations are indicated by triangles (SNP) or arrows (microsatellite) on the horizontal axis.

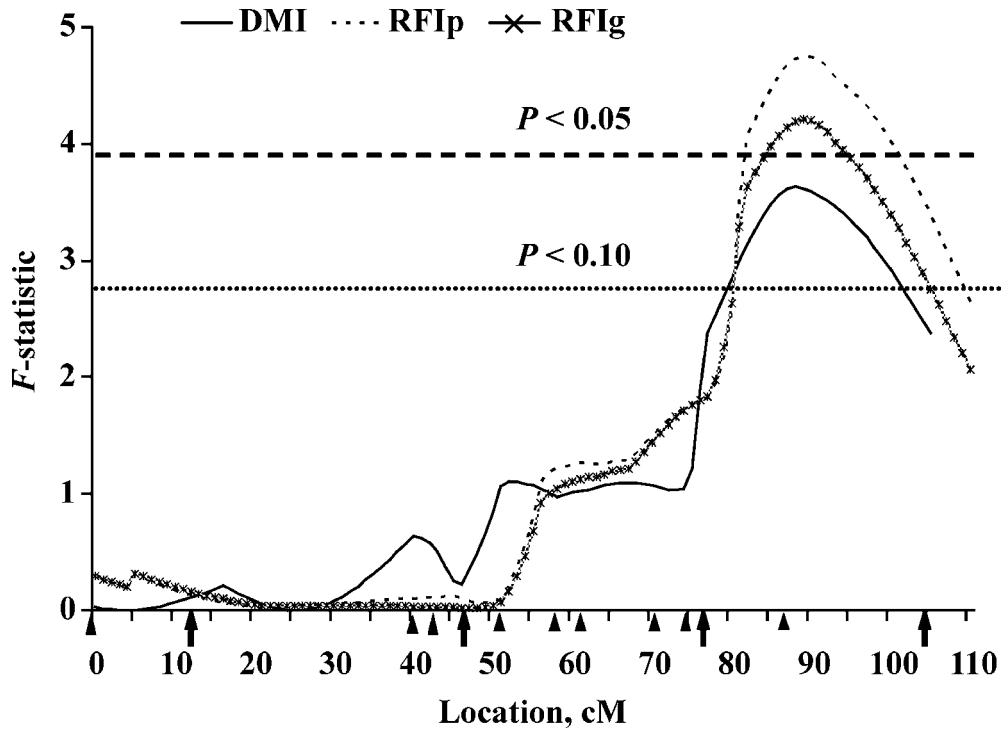

Figure 3. Across-family *F*-statistic profile for DMI and residual feed intake on chromosome 12. Horizontal lines represent the chromosome-wise threshold values from 2,000 permutations. Relative marker locations are indicated by triangles (SNP) or arrows (microsatellite) on the horizontal axis. RFIg = genetic residual feed intake; RFIp = phenotypic residual feed intake.

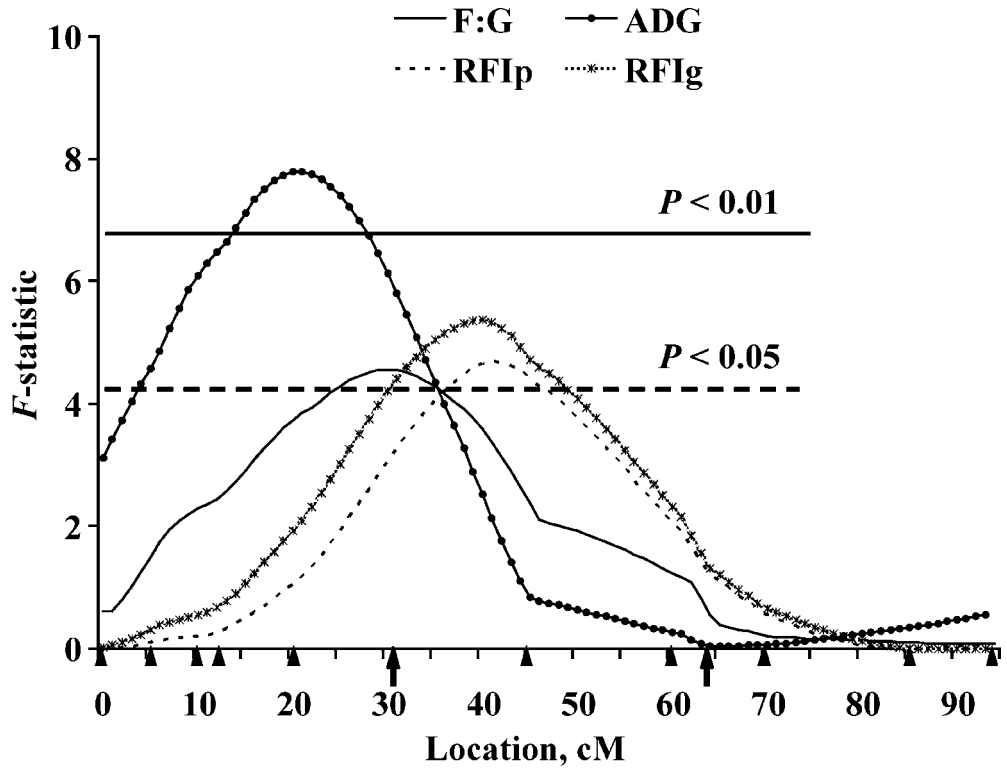

Figure 4. *F*-statistic profile for ADG, feed-to-gain ratio (F:G), and residual feed intake on chromosome 16. Horizontal lines represent the chromosome-wise threshold values from 2,000 permutations. Relative marker locations are indicated by triangles (SNP) or arrows (microsatellite) on the horizontal axis. RFIg = genetic residual feed intake; RFIp = phenotypic residual feed intake.

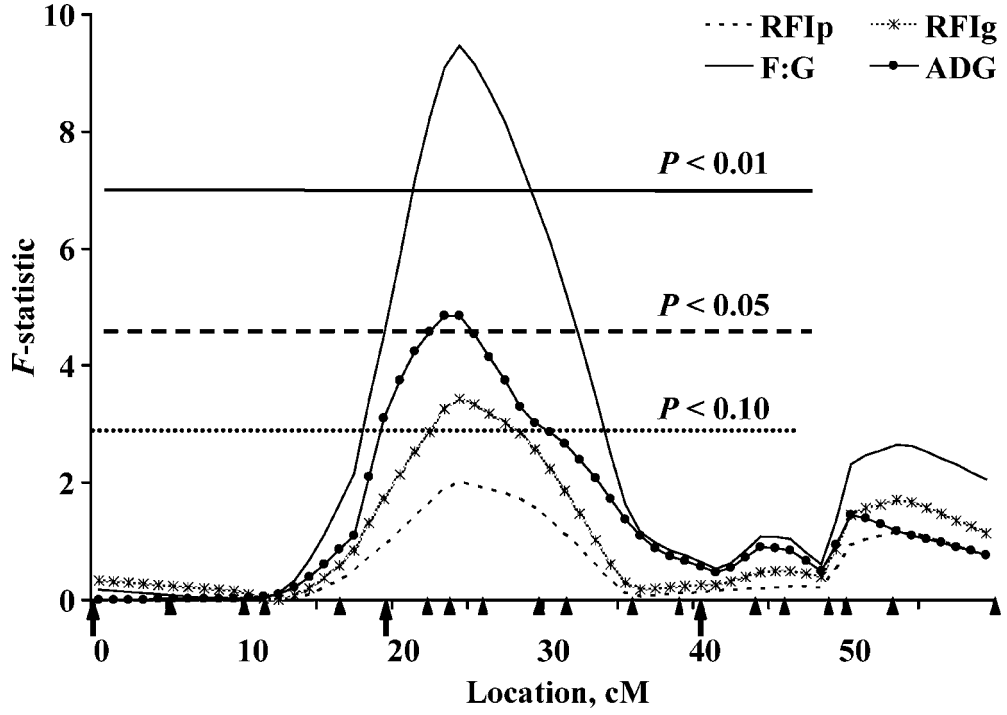

Figure 5. Across-family *F*-statistic profile for ADG, feed-to-gain ratio (F:G), and residual feed intake on chromosome 28. Horizontal lines represent the chromosome-wise threshold values from 2,000 permutations. Relative marker locations are indicated by triangles (SNP) or arrows (microsatellite) on the horizontal axis. RFIg = genetic residual feed intake; RFIp = phenotypic residual feed intake.

that a number of genes have potential pleiotropic effects on the 2 traits, even though the overall levels of phenotypic variation between the traits in a given population could be forced to be statistically independent (Kennedy et al., 1993; Crews, 2005).

In summary, we have reported an autosomal genome scan for QTL influencing feedlot growth rate, feed intake, and feed efficiency in the current study. Several putative QTL were detected for feed intake and feed efficiency across and within half-sib families. It is likely that the different QTL detected for the different traits may be the result of the pleiotropic effects of a fewer number of causal genes, obviously because of strong genetic relationships among some of the traits. Results of this study, in terms of the number of QTL and effects sizes, indicate considerable potential for further fine-mapping and application in marker-assisted selection and management.

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