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Quantitative trait loci associated with body weight and abdominal fat traits on chicken chromosomes 3, 5 and 7

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ABSTRACT. Body weight and abdominal fat traits in meat-type chickens are complex and economically important factors. Our objective was to identify quantitative trait loci (QTL) responsible for body weight and abdominal fat traits in broiler chickens. The Northeast Agricultural University Resource Population (NEAURP) is a cross between broiler sires and Baier layer dams. We measured body weight and abdominal fat traits in the F_2 population. A total of 362 F_2 individuals derived from four F_1 families and their parents and F_0 birds were genotyped using 29 fluorescent microsatellite markers located on chromosomes 3, 5 and 7. Linkage maps for the three chromosomes were constructed and interval mapping was performed to identify putative QTLs. Nine QTL for body weight were identified at the 5% genome-wide level, while 15 QTL were identified at the 5% chromosome-wide level. Phenotypic variance explained by these QTL varied from 2.95 to 6.03%. In particular, a QTL region spanning 31 cM, associated with body weight at 1 to 12 weeks of age and carcass weight at 12 weeks of age, was first identified on

chromosome 5. Three QTLs for the abdominal fat traits were identified at the 5% chromosome-wide level. These QTLs explained 3.42 to 3.59% of the phenotypic variance. This information will help direct prospective fine mapping studies and can facilitate the identification of underlying genes and causal mutations for body weight and abdominal fat traits.

Key words: Chicken; Body weight; Abdominal fat traits; NEAURP; Quantitative trait loci; Microsatellite marker

INTRODUCTION

The chicken is not only a widely raised farm animal but also an excellent model organism, and studies on chicken genome are of great value to agriculture and medicine. Significant advances on growth rate, in meat-type chickens, have been achieved for more than half a century, and it will continue to be one of the most important economic traits in broiler breeding programs. Progress in rapid growth has been accompanied by an increase in fat deposition in the broiler. Excessive fat deposition is economically and biologically unfavorable in broiler production. Modern broiler breeds contain 150-200 g fat per kg body weight, and 85% of this fat is not physiologically essential (Choct et al., 2000). It is well known that fat deposition has negative influences on feed efficiency and carcass yield and can bring about difficulty in meat processing and rejection by customers (Abasht et al., 2006; Zhou et al., 2006b; Campos et al., 2009). Fat deposition has highly heritability and exhibits positive genetic and phenotypic correlations with body weight. Therefore, it is a problem for broiler genetic improvement, as selection for high growth rate also gives rise to an increased fat deposition (Le Bihan-Duval et al., 1999; Campos et al., 2009).

It is difficult and costly to reduce fat deposition using selection strategies based merely on phenotype (Ikeobi et al., 2002; Lagarrigue et al., 2006). The identification of quantitative trait loci (QTL) for fat deposition could be used in marker assisted selection (MAS) to reduce body fat, without affecting body weight (BW), and generating more rapid genetic improvement (Campos et al., 2009).

Identification of markers and genes that underlie phenotypic variation in quantitative traits remains a major challenge. QTL mapping is a method that has been used successfully to examine genetic contributions to some quantitative traits by correlating allelic variation in polymorphic genetic markers with trait variability (Andersson and Georges, 2004; Tercic et al., 2009). Many studies have successfully detected numerous QTL for economically important traits such as growth and body composition in chickens by using crossbred experimental populations (Abasht et al., 2006). To date, the Chicken QTLdb (http://www.animalgenome.org) contains 2451 QTLs involving 248 different traits from 125 publications. Numerous QTL affecting growth and fat traits were identified on chicken chromosomes 3, 5 and 7. These studies made it convenient to further delve into the potential genes underlying the QTL. However, before attempting to identify potential genes and exploiting them in animal breeding programs by MAS, confirmation is necessary to verify the existence of QTL observed in an initial genome scan, preferably by using independent populations (Spelman and Bovenhuis, 1998; Marklund et al., 1999; Nones et al., 2006). The objective of the present study was to identify

new chromosomal regions affecting BW and abdominal fat traits and also confirm regions already associated with these traits in other chicken populations on chromosomes 3, 5 and 7 using a unique F_2 designed population from a broiler x layer cross.

MATERIAL AND METHODS

Experimental populations

The Northeast Agricultural University Resource Population (NEAURP) was used in the current study. The NEAURP was created by crossing broiler sires, derived from high line at NEAU divergently selected for abdominal fat, with Baier layer dams, a Chinese local breed. The F_1 birds were intercrossed to produce F_2 population. A total of 362 F_2 individuals produced from 4 F_1 families, 22 F_0 individuals and 28 F_1 individuals were used for the study. All F_2 birds had free access to feed and water. Commercial corn-soybean-based diets that met all NRC requirements (National Research Council, 1994) were provided in the study. From hatch to 3 weeks of age, birds received a starter feed (3000 kcal ME/kg and 210 g/kg CP) and from 3 to 12 weeks of age, birds were fed a grower diet (3100 kcal ME/kg and 190 g/kg CP) (Wang et al., 2006).

Phenotyping

The BW was measured at hatch and weekly up to 12 weeks of age. Carcass weight (CW) and abdominal fat weight (AFW) were recorded at 12 weeks of age. The AFW was also expressed as a percentage of BW at 12 weeks of age (AFP).

Genotyping

The 29 fluorescent microsatellite markers on chromosome 3, 5 and 7 were selected from the website (http://www.ncbi.nlm.nih.gov/ and http://www.thearkdb.org/arkdb/) in the current study. They spanned approximately 600 cM, which account for about 16% of whole chicken linkage map (3800 cM). Genomic DNA was isolated from venous blood samples using a phenol-chloroform method (Wang et al., 2006). Polymerase chain reactions (PCRs) for each marker were carried out separately in a reaction volume of 25 μL including 100 ng template DNA, 1X PCR buffer (10 mM Tris-HCl, 50 mM KCl, and 1.5 mM $MgCl_2$, pH 8.3), 0.25 μM of each primer, 200 μM of each deoxynucleotide triphosphate (dNTP), and 1 U Taq polymerase (Takara Biotechnology Co., Ltd., Dalian, China). The PCR products of microsatellite markers were analyzed on an ABI3700 DNA sequencer (Applied Biosystems, Foster City, CA, USA), and genotypes were determined using GeneScan Analysis 3.7 and Genotyper Analysis 3.7 softwares (Applied Biosystems). All F_0 (22), F_1 (28) and F_2 (362) (both males and females) animals were genotyped for all markers.

Statistical analyses

Phenotypic data were analyzed by using the JMP 4.0 software (SAS Institute, 2004). Means, standard deviation (SD), and coefficient of correlations between BW and abdominal fat traits were calculated.

The linkage map was constructed by using CRIMAP (Green et al., 1990). The marker order was explored using the FLIPS command until the marker order that maximized the likelihood was obtained. The Kosambi genetic distances in cM were then estimated using the 'build' option.

The GridQTL express software under an F_2 model at http://www.gridqtl.org.uk/ (Seaton et al., 2006) was utilized for QTL analyses. Data were subjected to a model containing additive and dominant effects of a putative QTL, with sex, hatch, and family as fixed effects in the model. When BW of 1 to 12 weeks of age and CW at 12 weeks of age were analyzed, the BW at hatch (BW0) was used as a covariate trait, and when the AFW was analyzed, the CW at 12 weeks of age was used as a covariate trait. The percentage difference in the residual sums of squares between the full and reduced model was calculated as the phenotypic variance that QTL could explain. Significance thresholds for analyses were calculated using a permutation test (Churchill and Doerge, 1994). A total of 1000 permutations were computed to determine the empirical distribution of the statistical test under the null hypothesis of no QTL associated with the part of the genome under study. Identification of two QTL was declared for a trait when peak F-ratios were ≥40 cM apart. Three significance levels were used: suggestive, 5% chromosome-wide, as well as 5% genome-wide. Suggestive and chromosome-wide significance were directly determined by GridQTL express. The threshold for the 5% genome-wide level was obtained using Bonferoni's correction (Knott et al., 1998), namely, $P_{genome} = \alpha/n$, where $\alpha = 0.05$, n was the total number of tests (15 traits x 3 chromosomes).

RESULTS

Phenotypic data analyses

The means and SD of the traits and the phenotypic correlations between the 16 traits from the F_2 individuals in the QTL analysis were shown in Table 1. BW traits at different weeks of age and CW have positive and significant phenotypic correlations with AFW ($P < 0.05$) and low phenotypic correlation with AFP.

Table 1. Means and standard deviation (SD) of body weight (BW) and abdominal fat traits, and phenotypic correlations between them in the F_2 population (N = 362).

Traits[1]	Mean[2]	SD[2]	BW1	BW2	BW3	BW4	BW5	BW6	BW7	BW8	BW9	BW10	BW11	BW12	CW	AFW	AFP
BW0	38.79	3.67	0.45	0.33	0.25	0.20	0.20	0.16	0.13	0.16	0.13	0.12	0.14	0.14	0.15	0.24	0.15
BW1	73.99	10.09		0.71	0.50	0.41	0.40	0.37	0.28	0.30	0.28	0.29	0.27	0.26	0.27	0.26	0.12
BW2	160.40	22.60			0.82	0.78	0.71	0.65	0.61	0.58	0.57	0.57	0.55	0.53	0.54	0.29	0.01[ns]
BW3	286.53	44.02				0.92	0.81	0.74	0.73	0.68	0.68	0.65	0.63	0.61	0.61	0.24	-0.08[ns]
BW4	446.89	72.09					0.91	0.87	0.87	0.82	0.81	0.78	0.77	0.74	0.75	0.31	-0.06[ns]
BW5	621.66	97.38						0.97	0.93	0.91	0.89	0.88	0.87	0.85	0.86	0.30	-0.12
BW6	819.41	137.09							0.97	0.96	0.94	0.93	0.91	0.89	0.89	0.34	-0.09[ns]
BW7	1,037.31	185.32								0.98	0.97	0.95	0.93	0.91	0.91	0.33	-0.11
BW8	1,250.10	227.73									0.99	0.97	0.96	0.94	0.95	0.32	-0.14
BW9	1,490.69	284.19										0.99	0.98	0.96	0.96	0.32	-0.15
BW10	1,682.60	324.51											0.99	0.98	0.98	0.30	-0.17
BW11	1,887.55	370.31												0.99	0.99	0.30	-0.18
BW12	2,070.75	418.48													0.99	0.28	-0.21
CW	1,832.97	379.15														0.29	-0.20
AFW	77.80	30.72															0.96
AFP	0.038	0.015															

[1]BWn = weight at n weeks of age, g; CW = carcass weight, g; AFW = abdominal fat weight, g; AFP = AFW expressed as a percentage of BW at 12 weeks of age. [2]Data were cited from Liu et al. (2007). [ns]Indicate that coefficients of phenotypic correlation are not significant ($P > 0.05$).

Linkage map construction

In the current study, sex average linkage maps for 3 chromosomes were respectively constructed by multi-locus linkage analysis. The linkage maps in length of chromosomes 3, 5 and 7 were 308.5, 261.0 and 177.8 cM, respectively (Table 2). Locus orders were in general accordance with the consensus linkage map (Schmid et al., 2000). There was one discrepancy that marker ADL0315 and marker MCW0316 was reversed in the chromosome 7 map compared with the consensus linkage map; however, they was in agreement with their physical map.

Table 2. Estimated map positions of microsatellite markers used for analysis.

Microsatellites on chromosome 3	Estimated position (cM)	Microsatellites on chromosome 5	Estimated position (cM)	Microsatellites on chromosome 7	Estimated position (cM)
ADL0177	0.0	LEI0116	0.0	MCW0030	0.0
MCW0222	38.8	MCW0263	45.0	MCW0120	53.8
HUJ0006	48.4	ADL0253	72.6	ADL0107	61.4
LEI0161	101.4	ADL0292	138.9	MCW0183	100.0
ADL0280	150.3	MCW0214	167.3	ADL0180	128.8
MCW0103	171.7	MCW0223	187.6	ADL0109	142.3
GCT0019	177.4	LEI0149	205.4	ADL0315	150.3
MCW0224	190.2	ADL0166	234.8	MCW0316	177.8
MCW0207	213.5	ADL0298	261.0		
ADL0237	243.6				
LEI0166	266.8				
MCW0037	308.5				

QTL analysis for body weight and abdominal fat traits

The QTL with suggestive and significant linkages for each trait, the additive and dominance effects of the QTL, as well as the phenotypic variance explained by the QTL are summarized in Table 3, and details of the markers flanking each QTL, and the estimated location relative to the first marker of 3 linkage maps are shown (Table 3).

For BW, a total of 39 QTL were detected (Table 3). These QTL were distributed over 4 distinct regions on 3 chromosomes, and their effects ranged from 1.94 to 6.03% of the phenotypic variation. On chromosome 3, 3 QTL and 10 QTL were identified at the 5% chromosome wide level and the suggestive level, respectively. The QTL for BW at 2 to 5 weeks and 8 to 10 weeks of age were located in the region of 89 to 104 cM, and the QTL responsible for BW at 6, 7, 10 to 12 weeks of age and CW were mapped in the region of 246 to 248 cM. On chromosome 5, 4 QTL were identified at the 5% genome wide level, 8 QTL at the 5% chromosome wide level, and 1 QTL at the suggestive level. The test statistics for BW of 1 to 12 weeks of age and CW peaked in the region 13 to 44 cM. The genomic region for body weight was firstly reported. On chromosome 7, 5 QTL were identified at the 5% genome wide level, 4 QTL at the 5% chromosome wide level, and 4 QTL at the suggestive level. The QTL affecting BW at 1 to 12 weeks of age and CW were located in the region of 71 to 134 cM.

For abdominal fat traits, three significant and four suggestive QTL were detected on 3 chromosomes, and their effects ranged from 1.77 to 3.59% of the phenotypic variation. Both a significant QTL for AFW at 5% chromosome wide level and a suggestive QTL for AFP were mapped at the same position, 177 cM, and other two QTL affecting AFW and AFP were detected at 88 and 85 cM on chromosome 3, respectively. On chromosome 5, only one

suggestive QTL for AFW was identified at 82 cM. Both a significant QTL for AFW at 5% chromosome wide level and a suggestive QTL for AFP were detected at the same position 129 cM on chromosome 7.

Table 3. QTL locations and effects on body weight and abdominal fat traits.

Position (cM)[1]	Traits	LR	F-ratio	Flanking markers	Additive effect (SE)[2]	Dominant effect (SE)[2]	Phenotypic variance (%)[3]
Chr3							
89	BW2	9.89	5.02†	HUJ0006-LEI0161	6.99 (2.51)	-8.49 (5.47)	3 18
94	BW3	11.38	5.79*	HUJ0006-LEI0161	14.09 (4.37)	-9.64 (8.99)	3.43
89	BW4	12.41	6.33*	HUJ0006-LEI0161	24.39 (7.07)	-13.18 (15.36)	3.71
101	BW5	10.61	5.39*	HUJ0006-LEI0161	25.08 (8.54)	-18.95 (15.43)	3.23
247	BW6	8.72	4.42†	ADL0237-ADL0166	-6.08 (11.71)	52.77 (18.65)	2.63
248	BW7	7.50	3.79†	ADL0237-ADL0166	-1.36 (15.50)	68.08 (25.12)	2.23
104	BW8	6.74	3.41†	LEI0161-ADL0280	50.42 (20.37)	-29.72 (38.31)	2.04
102	BW9	6.51	3.29†	LEI0161-ADL0280	52.60 (23.33)	-48.34 (42.32)	1.94
102	BW10	7.10	3.59†	LEI0161-ADL0280	63.63 (27.09)	-59.31 (48.78)	2.15
246	BW10	11.39	5.12†	ADL0237-ADL0166	-29.96 (24.72)	110.33 (39.75)	3.04
247	BW11	8.57	3.95†	ADL0237-ADL0166	-25.45 (28.24)	114.07 (45.60)	2.31
246	BW12	7.47	3.78†	ADL0237-ADL0166	-24.02 (30.58)	121.37 (48.39)	2.17
248	CW	7.24	3.66†	ADL0237-ADL0166	-22.30 (28.57)	113.15 (46.25)	2.10
177	AFP	8.79	4.45†	MCW0103-GCT0019	1.1E-3 (8.89E-4)	-3.7E-3 (1.42E-3)	2.32
85	AFP	12.83	6.53*	HUJ0006-LEI0161	5.2E-3 (1.49E-3)	3.3E-3 (3.3E-3)	3.54
177	AFW	12.25	6.22*	MCW0103-GCT0019	2.48 (1.79)	-9.01 (2.85)	3.42
88	AFW	9.29	4.71†	HUJ0006-LEI0161	8.89 (2.99)	5.47 (6.44)	2.61
Chr5							
13	BW1	6.30	3.18†	LEI0116-MCW0263	2.75 (1.29)	3.33 (2.32)	1.97
35	BW2	10.84	5.52*	LEI0116-MCW0263	8.70 (2.68)	-1.65 (5.44)	3.48
24	BW3	14.19	7.25**	LEI0116-MCW0263	20.47 (5.52)	-8.30 (11.03)	4.26
29	BW4	11.81	6.01*	LEI0116-MCW0263	28.91 (8.34)	1.18 (16.99)	3.54
34	BW5	13.14	6.71*	LEI0116-MCW0263	38.05 (10.39)	7.89 (20.86)	3.99
35	BW6	16.73	8.58**	LEI0116-MCW0263	60.27 (14.76)	-4.27 (29.18)	4.99
34	BW7	13.69	6.99**	LEI0116-MCW0263	73.82 (19.83)	-0.51 (38.88)	4.03
33	BW8	16.61	8.52**	LEI0116-MCW0263	98.84 (24.38)	-15.81 (48.31)	4.94
34	BW9	12.81	6.53*	LEI0116-MCW0263	102.53 (28.65)	-7.56 (56.65)	3.78
36	BW10	10.80	5.49*	LEI0116-MCW0263	107.89 (32.57)	19.29 (63.01)	3.23
41	BW11	10.66	5.42*	LEI0116-MCW0263	108.82 (33.06)	24.06 (59.91)	3.10
41	BW12	10.21	5.18*	LEI0116-MCW0263	115.73 (36.05)	11.54 (65.49)	2.95
44	CW	10.59	5.38*	LEI0116-MCW0263	101.04 (30.88)	11.05 (53.64)	3.06
82	AFW	6.31	3.18†	ADL0253-ADL0292	6.46 (3.21)	9.47 (6.90)	1.77
Chr7							
111	BW1	9.40	4.77†	MCW0183-ADL0180	2.39 (0.82)	-1.10 (1.39)	2.92
78	BW2	11.13	5.67*	ADL0107-MCW0183	6.07 (2.05)	-6.06 (4.02)	3.57
133	BW3	11.18	5.68*	ADL0180-ADL0109	9.01 (3.08)	8.09 (5.07)	3.37
134	BW4	16.08	8.24**	ADL0180-ADL0109	17.13 (4.77)	15.06 (8.06)	4.78
71	BW5	11.97	6.10*	ADL0107-MCW0183	25.42 (7.38)	-5.48 (13.93)	3.64
124	BW6	20.35	10.5**	MCW0183-ADL0180	41.28 (9.37)	19.54 (15.76)	6.03
121	BW7	15.86	8.12**	MCW0183-ADL0180	50.41 (13.03)	28.04 (22.76)	4.65
120	BW8	14.36	7.34**	MCW0183-ADL0180	60.71 (16.36)	27.60 (28.88)	4.28
119	BW9	15.65	8.01**	MCW0183-ADL0180	77.23 (19.46)	24.93 (34.65)	4.60
117	BW10	9.55	4.85†	MCW0183-ADL0180	70.93 (23.04)	27.49 (41.07)	2.86
80	BW11	10.78	5.48*	ADL0107-MCW0183	91.98 (27.80)	6.80 (52.94)	3.13
116	BW12	9.80	4.97†	MCW0183-ADL0180	85.99 (27.99)	44.15 (49.99)	2.83
116	CW	10.12	5.13†	MCW0183-ADL0180	79.49 (25.40)	39.37 (45.37)	2.92
129	AFW	12.77	6.53*	ADL0180-ADL0109	5.17 (1.98)	-7.21 (2.84)	3.59
129	AFP	10.75	5.46†	ADL0180-ADL0109	2.70E-3 (9.91E-4)	-2.90E-3 (1.4E-3)	2.94

[1]QTL positions relative to the genetic maps in Table 2. [2]Additive and dominance QTL effects correspond to genotype values +a, d, and -a for individuals having inherited two broiler alleles, heterozygotes, and individuals with two layer alleles, respectively. Positive additive effects indicate that broiler alleles increased the trait; negative, that broiler alleles decreased it. Dominance effects are relative to the mean of the two homozygotes. [3]Phenotypic variance = percentage difference in the residual sums of squares between the full and reduced model. †Suggestive linkage; *Chromosome wide significant, $P < 0.05$; **Genome wide significant, $P < 0.05$.

DISCUSSION

QTL analysis for body weight

Body weight is a complex quantitative trait resulting from various developmental processes (Brockmann et al., 1998; Ankra-Badu et al., 2010). Uncovering the molecular mechanism of growth will contribute to more efficient selection for growth in broiler chickens (Deeb and Lamont, 2002).

In the present study, the QTL for BW at 2 to 5 weeks and 8 to 10 weeks of age were identified in the region of 89 to 104 cM on chromosome 3. The flanking markers associated with this region are HUJ0006 and ADL0280. Kerje et al. (2003) reported that when the two estimated QTL positions differed by a recombination distance <30 cM in a chromosome region, a single QTL for the given trait was assumed on that chromosome. At the same time, the phenotypic correlation coefficients between BW at different weeks of age (in particular for adjacent BW) were significant (Table 1) and the QTL positions were close (Table 3). Thus, it was reasonable to assume that the same QTL influenced BW at 2 to 5 weeks and 8 to 10 weeks of age. The additive effects of this QTL were all positive, indicating that the increasing BW allele (higher BW) was derived from the broiler sire. This QTL was consistent with the QTL reported by other studies. Carlborg et al. (2003) identified a QTL for BW at 8 days of age in the region between HUJ0006 and LEI0161. Ambo et al. (2009) identified one QTL flanked by LEI0161 and LEI0029 for BW at 35 and 41 days of age. These two regions all were comprised in the region between HUJ0006 and ADL0280. In the 246 to 248 cM region on chromosome 3, a QTL responsible for BW at 6, 7, 10 to 12 weeks of age and CW was detected in the present study. Zhang et al. (2006) reported that there were significant associations between T123G polymorphism of the APOB gene in the same region and BW at 1 and 3 weeks of age. This QTL with a negative additive effect was a so-called cryptic QTL, which was believed to be caused due to no or limited selection for the trait, drift, and pleiotropic effects of the QTL allele on other traits that are under selection, or close linkage and linkage disequilibrium with QTL that are under selection (Abasht et al., 2006).

The QTL for BW of 1 to 12 weeks of age and CW were detected in the region 13 to 44 cM on chromosome 5 in the current study, which has not been reported in other studies. They all were significant at the 5% chromosome or genome wide level (except BW1), and exhibited positive additive effects, suggesting that the increasing BW allele (higher BW) was derived from the broiler sire. In addition, owing to the fact that their positions were close, it was reasonable that they were considered to have the same QTL affecting BW at 1 to 12 weeks of age and CW. The effects of this QTL ranged from 1.77 to 3.59% of the phenotypic variation.

The QTL affecting BW at 1 to 12 weeks of age and CW were identified in the chromosomal region of 71 to 134 cM on chromosome 7. These QTL all showed positive additive effects, indicating that increasing BW allele was inherited from the broiler line. The percent of phenotypic variance explained by these QTL varied from 2.83 to 6.03%. The microsatellite markers associated with this region are ADL0107, MCW0183, ADL0180, and ADL0109. Previous studies have identified the numerous QTL affecting BW at different weeks or days of age in the above-mentioned region (Sewalem et al., 2002; Kerje et al., 2003; Siwek et al., 2004; Jacobsson et al., 2005; Zhou et al., 2006a; Atzmon et al., 2007, 2008; Wahlberg et al., 2009). These results were in agreement with those reported in this study.

QTL analysis for abdominal fat traits

In the present study, three significant and four suggestive QTL for abdominal fat traits were detected on 3 chromosomes. Two QTL for AFW and AFP mapped at the same position, 177 cM, should be the same one QTL on chromosome 3. This QTL respectively explained 3.42 and 2.32% of the phenotypic variance for AFW and AFP, and had positive and less additive effects than dominance effect, suggesting that the increasing AFW allele was derived from the broiler sire and the QTL mainly acted in an over-dominant fashion. The flanking markers of this QTL were MCW0103 and GCT0019. This region was contained in the QTL region flanked by MCW0277 and MCW0207 reported by McElroy et al. (2006), who identified a QTL affecting AFP that could explain 4.45% of the phenotypic variance in an F_2 cross between 2 commercial broiler lines. Two QTL for AFW and AFP with positive additive effects were identified at 88 and 85 cM on this chromosome. They were in close vicinity to the QTL region (89 to 104 cM) for BW at 2 to 5 weeks and 8 to 10 weeks of age. It was very likely that the same one pleiotropic QTL controlled these traits considering their close positions, positive additive effects and significant phenotypic correlations between BW and AFW (Table 1). This QTL was in the same region as the QTL for AFW identified by Lagarrigue et al. (2006). Park et al. (2006) also identified a QTL for AFW in this region in an intercross between chicken lines divergently selected for growth. Atzmon et al. (2008) previously associated the marker MCW0222 (this marker was just located in this region) with AFW using a multigenerational resource chicken population.

On chromosome 5, only one suggestive QTL for AFW was identified at 82 cM. The flanking markers related to this QTL were ADL0253 and ADL0292. In this region, many QTL for AFW were identified. McElroy et al. (2006) mapped a QTL for AFW flanked by MCW0193 and ADL0292, which was located in the similar genomic region. Marker MCW0193 locating in this region was significantly associated with AFW (Atzmon et al., 2008). Le Mignon et al. (2009) reported the identification of a QTL for AFW in this region, explaining 14% of the phenotypic variance. Nadaf et al. (2009) identified a QTL for AFW, covering this region, in an F_2 intercross between high- and low-growth chicken lines. In addition, this QTL was also confirmed by other independent studies (Ikeobi et al., 2002; Abasht et al., 2006; Lagarrigue et al., 2006).

Two QTL affecting AFW and AFP were detected at the same position, 129 cM, on chromosome 7 in the current study, having positive additive effects. Considering their same position and positive additive effects, it was assumed that the same QTL actually controlled both AFW and AFP in the chicken. The flanking markers associated with the region of this QTL included MCW0183, ADL0180 and ADL0109. In this genomic region, 4 significant and 1 suggestive QTL for AFW were previously identified using differently independent resource populations (Ikeobi et al., 2002; McElroy et al., 2006; Park et al., 2006; Atzmon et al., 2008), which was consistent with our results.

Aside from the aforementioned literatures concerning QTL mapped, a large number of QTL for BW and AFW were also previously reported in other regions on chicken chromosomes 3, 5 and 7 (Abasht et al., 2006; Hu et al., 2007). However, those QTL for BW and AFW were not detected in the present study. These inconsistencies in QTL results among experiments may be attributed to many aspects such as markers used, choice of statistical models, population type and size, genetic background, segregation of specific QTL in specific populations, differences in trait definition or measurement, as well as type I and II errors (Abasht et al., 2006; McElroy et al., 2006).

In summary, commercial breeding programs of broiler chickens have become more complex and challenging in that so many objectives need to be simultaneously considered to maintain health, reduce production costs, and improve product quality. Breeding goals must include increased growth rate, decreased abdominal fat, maintenance of good development and growth of the skeletal system, and overall fitness. The relationships of these traits are complex, and some of the traits are very difficult to measure. Therefore, molecular MAS may be required to improve genetic selection programs (Liu et al., 2007). The population described herein allowed confirmation of several QTL for BW and abdominal fat previously reported, as well as the identification of a previously unreported QTL region for BW on chromosomes 3, 5 and 7 in other studies. These findings have important significances for prospective fine mapping studies and for the identification of underlying genes and causal mutations, which will ultimately contribute to an understanding of the genetic background of growth and fat deposition of chicken.

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