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Original article

SNP mapping of QTL affecting growth and fatness on chicken GGA1

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Abstract – An F2 chicken population was established from a crossbreeding between a Xinghua line and a White Recessive Rock line. A total of 502 F2 chickens in 17 full-sib families from six hatches was obtained, and phenotypic data of 488 individuals were available for analysis. A total of 46 SNP on GGA1 was initially selected based on the average physical distance using the dbSNP database of NCBI. After the polymorphism levels in all F0 individuals (26 individuals) and part of the F1 individuals (22 individuals) were verified, 30 informative SNP were potentially available to genotype all F2 individuals. The linkage map was constructed using Cri-Map. Interval mapping QTL analyses were carried out. QTL for body weight (BW) of 35 d and 42 d, 49 d and 70 d were identified on GGA1 at 351–353 cM and 360 cM, respectively. QTL for abdominal fat weight was on GGA1 at 205 cM, and for abdominal fat rate at 221 cM. Two novel QTL for fat thickness under skin and fat width were detected at 265 cM and 72 cM, respectively.

QTL / chicken / growth / fatness / single nucleotide polymorphisms

1. INTRODUCTION

A number of tools for genome analyses developed during the last ten years has allowed the identification of the genes and gene polymorphisms controlling complex traits. This has opened perspectives for predictive medicine in humans and marker-assisted selection (MAS) in plants and animals of economic interest [12, 13, 16, 18]. Understanding the QTL regulating economically important traits can increase the response of breeding programs, especially for those that are difficult to improve by traditional selection. As an economical animal and a model animal, QTL study in the chicken has been widely conducted and great

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advances have been achieved. To date, more than 600 QTL have been identified in the chicken using genome scan with microsatellites [25]. The chicken genome comprises 39 pairs of chromosomes, which are divided into eight pairs of cytologically distinct chromosomes 1–8 (macrochromosomes) along with Z and W sex chromosomes and 30 pairs of microchromosomes. GGA1 is the largest, corresponding to 14.9% of the entire genome [6, 9]. More QTL affecting body weight (BW), growth, feed intake, and weights of breast muscle, thighs, drums, wings and fat deposition have been detected on this chromosome.

Until recently, QTL mapping in chickens was performed mainly by microsatellite linkage analyses. Single nucleotide polymorphisms (SNP) are the most common source of genetic variations in populations. Advances in genome sequencing have led to the discovery of millions of SNP in the chicken genome [26]. Many studies in other species indicated that using the SNP marker is efficient in QTL mapping [4, 14, 17].

In the present study, thirty informative SNP were used to genotype all individuals in an F2 full-sib chicken population established from a crossing between Xinghua (XH) and White Recessive Rock (WRR) chickens. Interval mapping QTL analyses were used to identify QTL associated with growth and fat traits.

2. MATERIALS AND METHODS

2.1. Experimental population

Xinghua and White Recessive Rock lines were selected for crossing. The White Recessive Rock is a fast growing broiler line that has been bred as a meat type. The Xinghua chicken is a Chinese native breed with slow growth, lower reproduction and favourable meat quality. Both were reared at the Guangdong Wens Foodstuff Ltd Company, China, as a closed population. Nine females and nine males from each line were selected for mating on the basis of consistent egg laying and semen production. Each male was paired with a female from the other line. Two each of the XH (♂) × WRR (♀) and WRR (♂) × XH (♀) mating were selected on the basis of satisfactory egg and semen yields to create the F1 generation. At 30 wk of age, 17 F1 males and 17 F1 females were selected to produce the F2 generation. An equal number of spare males and females were kept as replacements for any loss. Each male was mated to a female of the same cross from the alternative family. A total of 502 F2 chickens in 17 full-sib families from six hatches were obtained at two-weekly intervals, and the birds were reared for trait measurement.

2.2. Observations

BW at 7, 14, 21, 28, 35, 42, 49, 56, 63, 70, 77, and 84 d of age were recorded. All F2 chickens were slaughtered at 90 d of age, and fat thickness under skin, fat width, abdominal fat weight, and abdominal fat rate were recorded. Fat width was measured between the leg and breast muscles by vernier caliper. Abdominal fat rate was defined as the abdominal fat weight divided by carcass weight. BW gains per day at 0–4 wk of age (BWG1) and at 5–8 wk of age (BWG2) were defined as BW gain, after being adjusted by the hatch effect, divided by the number of days.

2.3. SNP selection and genotyping

Based on the average physical distance, a total of 46 SNP on GGA1 were initially selected from the dbSNP database of the National Center for Biotechnology Information (NCBI). Thirty informative SNP were potentially available for the genotyping of all F2 individuals, after their polymorphism levels in all F0 individuals (26 individuals) and part of the F1 individuals (22 individuals) were verified. Amongst all 30 SNP, rs15397920 did not follow Mendel Laws, and the polymorphism level of rs14937017 was low in the F2 family. After ruling out these two SNP, 28 informative SNP were available for analysis. In the F2, a genetic map was obtained using the CRI-MAP linkage programme [5]. The functions FLIPS and FIXED were used to evaluate the order of markers along the chromosome and to estimate the map distance between markers. rs1384934 4(M4) and rs15551556 (M28) could not be assigned to the linkage group and were therefore excluded from the QTL analysis. The average marker interval was 21.4 cM, and the average polymorphic information content was 0.3324 (range 0.0997–0.5642). Figure 1 shows the linkage phase of 26 SNP on GGA1.

Based on the sequences provided by NCBI, proper PCR primers for amplifying each SNP were designed (Tab. I). The 25 μL PCR reaction mixture contained 50 ng of chicken genomic DNA, 1 X PCR buffer, 12.5 pmol of each primer, 100 μM dNTP (each), 1.5 mM $MgCl_2$ and 1.0 U Taq DNA polymerase (all reagents were from the Sangon Biological Engineering Technology Company; Shanghai, China). The PCR conditions were 3 min at 94 °C, followed by 35 cycles of 30 s at 94 °C, 45 s at an annealing temperature (ranged from 55 °C to 62 °C according to each primer), 1 min at 72 °C, and a final extension of 5 min at 72 °C in a Mastercycler gradient (Eppendorf Limited, Hamburg, Germany). The PCR products were analysed on a 1% agarose gel to assess

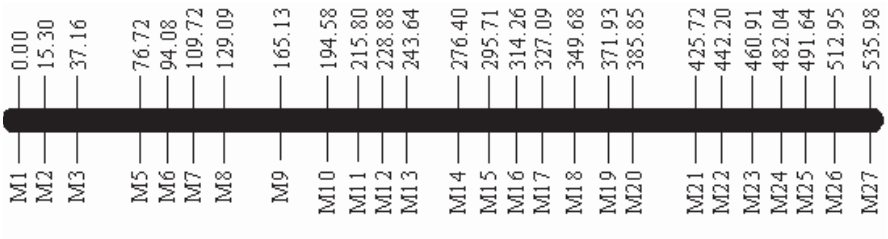

Figure 1. The linkage phase of 26 SNP on GGA1. M1–M27 represents 26 SNP respectively. M4 and M28 could not be assigned to this linkage group. The genetic distance (cM) between markers was estimated by CRI-MAP.

the correct size and quality of the fragments. The RFLP method was utilized in genotyping. The reaction mixture contained 4.0 μL PCR products, 0.5 μL restriction endonucleases, 1.0 μL 10 X PCR buffer, 4.5 μL deionised water. Digestion was carried out at 37 °C overnight. Restriction patterns were visualized by electrophoresis of the digestion product in a 2–3% agarose gel stained with ethidium bromide. Table II shows various restriction endonucleases used in each SNP genotyping.

2.4. QTL analyses

The QTL mapping method proposed by Haley *et al.* [7] was implemented using QTL Express software [19]. A linear model for the additive and dominant effects of a QTL at a given position was analysed by least squares for each trait. The additive effect was defined as half the difference between the two homozygotes and the dominant effect as the difference between the means of the heterozygotes and homozygotes. Phenotypic data from the 17 full-sib families were adjusted for hatch effect and the residuals were used in the QTL analyses. The statistical model included family and sex as fixed effects. In the analysis of abdominal fat weight, the fat thickness under skin and the fat width, a covariate-carcass weight included in the statistical model as another fixed effect. When the analysis demonstrated the existence of one QTL for any trait, the presence of two or more QTL was also tested.

2.5. Significance thresholds and confidence intervals

Significance threshold analyses were conducted using a permutation test [3]. A total of 10 000 permutations were computed to determine the empirical distribution of the statistical test under the null hypothesis of no QTL associated

Table I. PCR primers for the SNP amplification.

Marker no.	SNP	Variations	Primer (5′-3′)	Annealing T (°C)	Product length (bp)	Position of SNP
M1	rs15197960	G/T	TGCAACACAAGATGCTTTCC CATGGATGCTTTCAGCTTCA	56	595	131
M2	rs13835792	T/C	TGGGCAGGTAGAGAGCTGTT CTGCTTTTCCCCTTTCTCCT	58.5	481	182
M3	rs15217588	A/G	GGGGGAAGACTGCTGCTTAT ATGCCAAACCACCATTGACT	55	487	156
M4	rs13849344	A/G	AGGGCTGACAGCTGGTTTTA ACTTCCAACAGCCCATTCTG	60	509	104
M5	rs15245077	T/C	CTGGCTGCAGGAGAGTAAGC AAGCTGCCAAACAAAACCAG	60	489	207
M6	rs13651060	A/G	CTGCTTGCAGACCTCTAGGC ATACAGGCCAAGCACAGGAA	62	439	115
M7	rs15261060	G/T	CTTCCCACCAACGTTCTGTT CCAAAGCTCTGAAAGGCAAG	58	593	238
M8	rs15279778	T/C	AATTCATCCCTCCAGCACAG CTCTCTGCATGCCTTCACTG	56	442	79
M9	rs14837036	A/G	ATCCGTGGTTTGGTATTGGA CCACTTTGCTGCAGTCGTTA	56	561	405
M10	rs15310568	T/C	CACCCAAACAGTCCCATTTT ATTTGCCATGCAGCTTCTTT	56	439	116
M11	rs14848790	T/C	CCAGCAGTGTTCTCACCTCA CTGGATGATCCTGTGGGTCT	60	645	128
M12	rs13896190	A/G	TCAGGACCGTGGAGTTTTTC CCAGCTGAGACAGTTGGACA	60	570	236
M13	rs15343813	C/T	GTCCAAATTCCCCCAGAGAT CGGTTGGACTTGGTGATCTT	60	558	93
M14	rs15361441	T/C	CAATGGAACAGCCTTGAGTG CCAGACTTTGACATGCTGGA	55.8	557	77
M15	rs14870625	A/G	AATCCCTCGTTCATGATGGT TAAGCTAGCAGGGCAGTCGT	55	534	289
M16	rs15389943	A/G	GCTCAGTTTTGGACCTGCTC GGCTTCCTCTGCACAACTTC	56	557	189
M17	rs15397270	G/T	TGTCCGGAAGAGAAGAGGAA AGCCTGGTTCCATGACAAAC	60	400	285
M18	rs14884316	A/G	GTGAGCTTCTGTGGTGCAAA CGAGAACCACTCCCATCTGT	62	468	58
M19	rs14889388	A/G	TGCATGGAGACAACTGGGTA GGGCTCCTGACGTGGTATTA	56	518	121
M20	rs14893213	G/C	TAGCTGCAGGCGTACAAAGA CCGTGCCCTGTACCTGTAGT	56	387	175
M21	rs15462582	T/C	AGGCTGAACAGTCCCAGCTA ATATGGGTGTGTGGCCTTGT	62	597	115
M22	rs15468665	T/C	AAGAAAAGCCGTGTTCTGGA CACTCAGGGCTGTGTCTTGA	60	393	81
M23	rs15481358	C/G	GAGTGTCCCTCTCCCTTTCC GCTTTTAGCCCACTGTGCAT	56	432	214
M24	rs14915286	A/G	TAGCTTTGGCATCCTCACCT AGAAATGTGGATGGGAGCAC	56.7	522	264

Table I. Continued.

Marker no.	SNP	Variations	Primer (5′-3′)	Annealing T (°C)	Product length (bp)	Position of SNP
M25	rs15503250	A/G	AGTGCCTGTGAGGACAAACC CCAATCCACCAAAGATGTCC	58	549	288
M26	rs15520693	A/G	GAGAGAGCCTCCGCTAATGA GGACAATCTCCTCCCTCTCC	60	464	89
M27	rs15538603	A/G	ATGTACTGGGACTGCCTTGG TGCCACTTACACAGGTGCTC	60	598	102
M28	rs15551556	A/T	GTGGGCAAGCTGATGATTTT TGTACCAGTCCCCTCACACA	62	541	248

with the part of the genome under study. Three significance levels were used: suggestive, 5% and 1% genome-wide [13]. An approximate confidence interval for the localization of each of the significant and suggestive QTL was obtained using the bootstrap technique [13, 24] with a total of 10 000 samplings.

3. RESULTS

3.1. QTL for growth traits

The overall means and standard deviations (SD) of 14 growth traits are presented in Table III. Four QTL related to growth were identified. QTL for 35 d BW, 42 d BW, and 70 d BW at a 5% genome-wise level were located at 351 cM, 353 cM, and 360 cM, respectively. QTL for 49 d BW at a suggestive level was located at 360 cM. QTL flanking markers, confidence intervals and the estimated location relative to the first marker on GGA1 are presented in Table IV. Means and standard errors (SE) of estimated additive and dominance effects, as well as each QTL contribution to the phenotypic variance are also presented in Table IV.

3.2. QTL for fat traits

The overall means and standard deviations (SD) of fat traits are presented in Table III. Among all the traits, a QTL for abdominal fat weight at a 1% genome-wise level was located at 205 cM. A QTL for fat thickness under the skin at a suggestive level was located at 265 cM. Two QTL for abdominal fat rate, and fat width at a 5% genome-wise level were located at 221 cM, and 72 cM, respectively. QTL flanking markers, confidence intervals and the estimated location relative to the first marker on GGA1 are presented in Table IV.

Table II. Information of the 28 SNP.

Marker no.	SNP	Genetic marker[1]	Physical distance (Mb)[2]	Genetic distance (cM)[3]	Variations	Reases[4]
M1	rs15197960	ACW0388	10.39	0.00	G/T	TaqI
M2	rs13835792	LEI0209	17.51	15.3	T/C	Hin6I
M3	rs15217588	LEI0194	24.51	37.16	A/G	MSPI
M4	rs13849344	ADL351	31.22		A/G	Eoc721
M5	rs15245077	ADL0019	37.17	76.72	T/C	TaqI
M6	rs13651060	ADL307	42.70	94.08	A/G	TaqI
M7	rs15261060	MCW0365	47.70	109.72	G/T	MSPI
M8	rs15279778	ACW0356	53.89	129.09	T/C	MSPI
M9	rs14837036	MCW0112	61.19	165.13	A/G	MSPI
M10	rs15310568	ACW0067	66.83	194.58	T/C	MSPI
M11	rs14848790	LEI0101	75.21	215.8	T/C	TaqI
M12	rs13896190	ADL251	79.39	228.88	A/G	MSPI
M13	rs15343813	ADL0020	84.11	243.64	C/T	HaeIII
M14	rs15361441	LEI0160	94.58	276.4	T/C	Hin6I
M15	rs14870625	MCW200	100.75	295.71	A/G	TaqI
M16	rs15389943	ADL148	106.68	314.26	A/G	HinPII
M17	rs15397270	ADL313	110.78	327.09	G/T	TaqI
M18	rs14884316	LEI0139	118.00	349.68	A/G	NaeI
M19	rs14889388	ACW0254	125.11	371.93	A/G	TaqI
M20	rs14893213	MCW0049	129.56	385.85	G/C	Hin6I
M21	rs15462582	MCW0102	142.30	425.72	T/C	Hin6I
M22	rs15468665	LEI0084	147.57	442.2	T/C	MSPI
M23	rs15481358	LEI0264	153.55	460.91	C/G	TaqI
M24	rs14915286	RBsts1	160.30	482.04	A/G	AluI
M25	rs15503250	ACW0295	163.37	491.64	A/G	HaeIII
M26	rs15520693	Ros0025	170.18	512.95	A/G	TaqI
M27	rs15538603	ADL001	177.54	535.98	A/G	Hin6I
M28	rs15551556	LEI0331	184.9		A/T	AluI

[1] The most adjacent microsatellite marker or STS marker to this SNP.
[2] The physical distance of this SNP on GGA1.
[3] The genetic distance of this SNP identified by Cri-Map.
[4] Restriction endonucleases.

Means and standard errors (SE) of estimated additive and dominance effects, as well as each QTL contribution to the phenotypic variance are also given in Table IV.

4. DISCUSSION

In the present study, three significant QTL for 35 d BW, 42 d BW and 70 d BW were identified on GGA1, which were located at 351 cM, 353 cM,

Table III. Phenotypic observation and analysis of the F2 population.

		Mean	Max.	Min.	SD[1]
Growth traits	7d BW (g)	58.80	85.11	33.90	8.41
	14 d BW (g)	123.38	178.20	70.90	18.40
	21 d BW (g)	210.83	304.90	101.62	33.77
	28 d BW (g)	311.83	448.80	119.30	50.09
	35 d BW (g)	437.02	628.80	142.00	75.78
	42 d BW (g)	574.06	888.20	214.3	104.71
	49 d BW (g)	708.11	1152.10	268.5	133.24
	56 d BW (g)	864.36	1422.00	430.00	153.23
	63 d BW (g)	1025.3	1572.00	490.50	190.50
	70 d BW (g)	1138.70	1900.00	699.00	214.32
	77 d BW (g)	1333.10	2150.00	797.30	249.24
	84 d BW (g)	1503.17	2800.00	804.00	296.76
	BWG1 (g)	10.06	14.86	3.050	1.80
	BWG2 (g)	19.77	36.14	10.10	4.24
Fat traits	Fat thickness under skin (mm)	3.95	9.00	0.05	1.47
	Fat width (mm)	11.83	22.97	2.00	3.42
	Abdominal fat weight (g)	27.60	94.40	2.60	16.73
	Abdominal fat rate (%)	2.07	6.38	0.18	1.23

[1] n = 488.

and 360 cM, respectively. The contribution of three QTL to phenotype variance ranged from 2.5–7.5%. The contribution of a suggestive QTL for 49 d BW located at 360 cM to phenotype variance was 3.0%. When comparing the test statistics for these BW QTL, we found that two QTL curves for 35 d BW and 42 d BW almost overlapped, and two QTL curves for 49 d BW and 70 d BW almost overlapped too (Fig. 2). The additive effects of these QTL were both positive, and the dominant effects were both negative. This strongly suggests the action of one single QTL affecting growth throughout the growth period. An association test indicates that polymorphism of M19 was associated with 35 d BW ($P = 0.022$) and 42 d BW ($P = 0.0025$), polymorphism of M20 was associated with 70 d BW ($P = 0.0487$). From the analysis of marker genotypes, we could not infer what line the effects of the allele originate from.

Numerous studies demonstrated that QTL displaying significant linkage with BW are located on GGA1 [1,2,11,20,22,23]. Sewalem *et al.* performed a

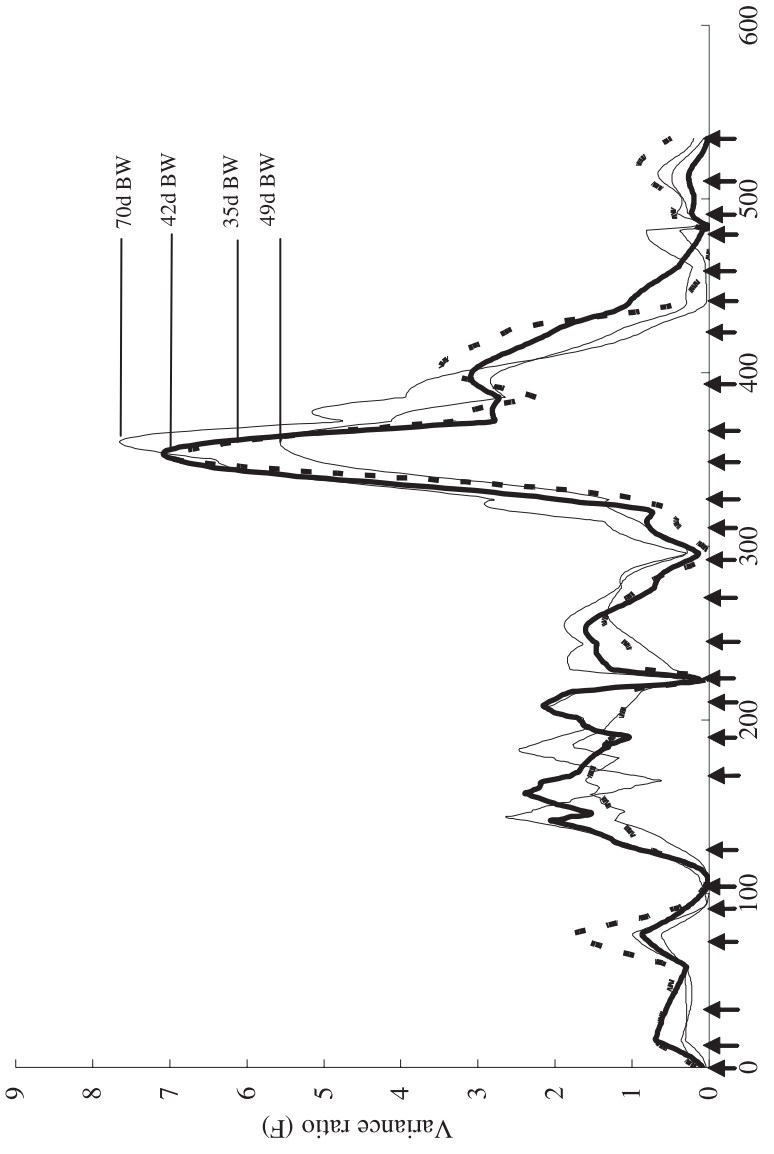

Figure 2. Test statistic values from the GGA1 QTL mapping analysis of body weight at 35 d (35 d BW), 42 d (42 d BW), 49 d (49 d BW) and 70 d (70 d BW) of age. Arrows indicate marker positions.

Table IV. Information of 8 QTL.

Traits	F-ratio[a]	Position (cM)[b]	Flanking-marker	Additive ± SE	Dominance ± SE	95% confidence interval	Effects[c]
35 d BW	6.98*	351	LEI0160-MCW0102	29.36 ± 11.33	–112.36 ± 30.44	288–397	7.5%
42 d BW	7.08*	353	ADL313-MCW0102	23.55 ± 14.10	–152.44 ± 41.76	335–419	2.5%
49 d BW	5.57+	360	ADL148-LEI0084	32.46 ± 13.75	–180.53 ± 60.53	317–430	3.0%
70 d BW	7.65*	360	ADL148-MCW0102	53.16 ± 26.47	–287.60 ± 64.28	319–391	3.1%
Fat thickness under skin	5.05+	265	ACW0388-MCW0102	–0.573 ± 0.54	–0.329 ± 0.15	0–393	7.6%
Fat width	7.44*	72	ACW0388-ADL0020	1.563 ± 0.27	6.26 ± 0.862	0–239	10.4%
Abdominal fat weight	10.74**	205	ACW0356-LEI0160	–3.612 ± 1.22	–14.26 ± 4.351	136–265	2.3%
Abdominal fat rate	8.46*	221	MCW0112-MCW200	–0.426 ± 0.17	–0.77 ± 0.27	168–283	6.0%

[a] + Suggestive linkage; * genome-wise linkage at 5%; ** genome-wise linkage at 1%.
[b] Position of QTL relative to the first marker in the set for this chromosome (Tab. II).
[c] Percentage of total phenotypic variance explained by this QTL.

genome scan for growth using a crossing between a White Leghorn line and a commercial broiler sire line. Two significant QTL for 3 wk-BW were located on GGA1 at 145 cM, and 481 cM, respectively, in which 95% confidence intervals were 113–217 cM, and 441–526 cM, respectively. Another significant QTL for 9 wk-BW was located on GGA1 at 414 cM with 34–419 cM of the 95% confidence interval [20]. Van Kaam *et al.* performed a genome scan for growth and carcass composition using a crossing population between two broiler lines. Only one QTL was up to a genome-wide significant level. This growth QTL was located on GGA1 at 235 cM [23]. Tatsuda *et al.* identified two significant QTL for growth using a crossing population between a Satsumadori line and a White Plymouth Rock line. One QTL identified on GGA1 was located at 220 cM [22]. Kerje *et al.* identified two major QTL for growth, which were located on GGA1 using a crossing population between Red Jungle Fowl (RJF) and White Leghorn. The two major QTL for growth were located around positions 68 cM and 416 cM, which had a large effect on growth from 7 d of age on and during the entire growth period. In addition, this explained more than 20% of the residual phenotypic variance for adult body weight, and about 35% of the difference in adult size between the two populations [11]. Nones *et al.* selected 26 microsatellite markers to conduct a scan on GGA1.

They identified a significant QTL for 35 d BW and 42 d BW, which was located at 332 cM of GGA1 (LEI0079-MCW0145) [15]. The QTL interval almost overlapped a QTL interval (LEI0160-MCW0102) determined in the present study. Interestingly, another significant QTL for 46 d BW, 112 d BW and 200 d BW was reported at a similar position on GGA1 from a crossing population between RJF and White Leghorn, which was located at 337 cM [1].

Fatness is being focused on in the QTL mapping studies of chickens. Iekobi *et al.* scanned the whole chicken genome for QTL controlling fat traits in a resource population derived from a crossing between a broiler line and layer line. Four QTL affecting abdominal fat weight were identified on GGA3, 7, 15, and 28, respectively, and another four QTL for abdominal fat rate were identified on GGA1, 5, 7, and 28, respectively. A QTL for abdominal fat rate identified on GGA1 was located at 126 cM, in which the confidence interval was 100–182 cM. Another QTL for skin fat weight was located at 454 cM of GGA1, in which the confidence interval was 333–487 cM [8]. Jenen *et al.* identified two QTL for fat traits located on GGA1 in a crossing population between two genetically different outcross broiler dam lines, which originated from the White Plymouth Rock breed. They found that a significant QTL controlling abdominal fat rate at 70 d of age was located at 241 cM (MCW0058-MCW0101), and a suggestive QTL for abdominal fat weight at 70 d of age was located at 214 cM (LE10174-ADL0361) [10]. Nones *et al.* [15] also reported a QTL for abdominal fat weight located on GGA1 at 194 cM (ADL0020-LEI0160). In the present study, we identified four QTL for fat traits, a significant QTL at a 1% genome-wise level for abdominal fat weight, two significant QTL at a 5% genome-wise level for abdominal fat rate and fat width, and a suggestive QTL for fat thickness under the skin. Each QTL explained the phenotypic variance with a range of 2.3–10.4%. A QTL for abdominal fat weight and a QTL for abdominal fat rate appear to be very consistent with what has been reported by Jenen *et al*. and Nones *et al*. Fat deposition in chickens has commanded a great deal of interest over the years because of the nutritional significance of fat to humans. Measuring abdominal and skin fat content is expensive and the availability of QTL for use in breeding practice would therefore prove to be of great value.

Confirmation of the presence and location of the QTL of interest can be achieved by comparing the results from different QTL studies. In the study of two distinct layer × layer crossings, Siwek *et al.* validated the presence of a QTL for the primary antibody response to keyhole lympet hemocyanin on GGA14 in both populations [21]. In the present study, we confirmed a QTL for chicken growth, a QTL for abdominal fat weight and a QTL for abdominal

fat rate with SNP mapping. We also detected two novel QTL for fat thickness under the skin, and fat width, respectively. In the above earlier studies, different breeds, different markers and measurements were used. This implies that these regions surely harboured QTL affecting these traits and deserve to be further explored.

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