

## Quantitative trait loci affecting growth performance in F<sub>2</sub> intercross between Golden Montazah and White Leghorn chickens

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### Abstract

Quantitative trait loci (QTL) for body weights (BW) at 4, 8, 12, 16 weeks of age and daily gains (DG) at intervals of 0-4, 4-8, 8-12 and 12-16 weeks were identified in F<sub>2</sub> crossbred population produced by crossing males of Golden Montazah (GM) with females of White Leghorn (WL). Phenotypic data were analyzed using multi-traits animal model including the genetic group, sex and hatch as fixed effects and the additive genetic and common environmental effects as random effects. After parentage checking and F<sub>2</sub> genotyping, data of 1011 chicks of F<sub>2</sub> were genotyped using 43 genetic markers in nine autosomal linkage groups, Z chromosome and the genotypes were used for QTL analysis. A mixed model included the sex and hatch as fixed effects along with the additive and dominance effects of QTL as random effects were used for QTL analysis. The heritability estimates for growth traits were high. The genotypic and phenotypic correlations between growth traits were positive and high. The total map length was 1901 cM (ranging from 25 to 568 cM), with an average spacing of markers of 24.39 cM (ranging from 7.8 to 24.3 cM). A total of 34 QTL were detected for BW traits. These QTL were distributed over five distinct regions on 10 chromosomes, and their effects ranged from 1.2 to 13.8% of the phenotypic variation. A total of 19 significant genome QTL that affected BW traits were located on seven macro-chromosomes (1, 2, 3, 4, 6, 8 and Z) and one micro-chromosome (11). A total of 14 significant QTL were detected for DG traits, distributed over 7 distinct regions on 6 chromosomes, and their effects ranged from 2 to 8.9% of the phenotypic variation. A total of 11 significant genome QTL affecting DG traits were located on five macro-chromosomes (1, 2, 3, 4 and 8) and there was statistical evidence for two QTL on chromosome 4. The proportions of phenotypic variation explained by significant and suggestive QTL for BW traits at 4, 8, 12 and 16 weeks were 21.1, 30.8, 29.3 and 25.4%, respectively. The proportions of phenotypic variation explained by significant and suggestive QTL for DG traits during 0-4, 4-8, 8-12 and 12-16 weeks were 25.9, 29.1, 9.35 and 3.9%, respectively. The largest proportion of the phenotypic variation explained by a QTL was 8.9% for DG4-8 at 428 cM on chromosome 4. The additive effects of QTL on growth traits were positive, while the dominance effects were generally negative or not significant. A QTL for BW at 12 weeks of age segregating on chromosome 4 at 179 cM had the largest additive effect (205.7 ± 22.2 g) and explained 13.8% of the phenotypic variation. The largest dominance effect (-188.1 ± 55.0 g) was for QTL of BW at 16 weeks of age segregation on chromosome 4 at 139 cM and the QTL effect accounted for 6.5% of the phenotypic variation. The total trait variances explained by QTL for each growth trait were 21.1, 30.8, 31.7, 25.4, 25.9, 29.1, 9.35 and 3.9 % in BW4, BW8, BW12, BW16, DG04, DG48, DG812 and DG1216, respectively.

**Keywords:** Chickens, QTL, microsatellite markers, growth traits, additive effects, dominance effects.

### Introduction

Indigenous chickens appear to possess enormous genetic diversity, especially in adaptive traits, and the ability to survive in harsh conditions and under minimum feeding regimens (Qu *et al.*, 2006; Kosba *et al.*, 2009; Eltanany 2011; Ramadan *et al.*, 2012). Comparing the local breeds of chickens with the improved exotic breeds, evidenced that the growth performance of local chicken populations is generally low (Hanafi *et al.*, 1991; Iraqi *et al.*, 2002). Nowadays, we need more workers for crossing Egyptian native breeds with exotic ones to determine the superior breeds, gains in performance from complementary breed effects and heterosis and to develop the superior new breeds through selecting the best combination of several breeds (Iraqi *et al.*, 2013). Results of most crossbreeding experiments that had been carried out in Egypt showed that

crossing between local breeds or strains of chickens with other local ones was generally associated with an existence of considerable heterotic effects on growth performance (Ezzeldin and El-Labban, 1989; Khalil *et al.*, 1991; Nawar and Bahie El-Deen, 2000).

Body weight is a complex quantitative trait resulting from various developmental processes (Brockmann *et al.*, 1998; Ankra-Badu *et al.*, 2010). Such quantitative trait is controlled by the additive effect of multiple genes. In QTL study, it is aimed to determine the most effective genes and chromosomal regions for the quantitative trait and to use this information in genomic selection. Many molecular markers have become excellent means for the study of genetic variation (Chen *et al.*, 2003; Chang *et al.*, 2005), such as random amplified polymorphic DNA (RAPD), amplified fragment length polymorphisms (AFLP), microsatellite DNA, and sequence-related

amplified polymorphism (SRAP) (Zietkiewicz *et al.*, 1994; Li and Quiros 2001). Microsatellites are tandem repeat loci with a core motif of 1 to 6 bp repeated several times. They are highly polymorphic and considered to be evenly distributed in the genome. They can be used in marker-assisted selection programs for improving the growth (Liu *et al.*, 2007).

The identification and utilization of QTL provide the potential for more rapid genetic improvement in selection programs, especially for traits that are difficult to improve with traditional selection (Ikeobi *et al.*, 2002). Van der Beek and Van Arendonk (1996) indicated additional selection responses of 6 to 13% using marker assisted selection (MAS) by incorporating a marker-linked QTL in a simulation study after five generations of selection. Based on chicken linkage maps and data from a variety of populations, several studies have reported many QTL for body weight in chickens (Tatsuda and Fujinaka, 2001; Sewalem *et al.*, 2002; Li *et al.*, 2003; Sasaki *et al.*, 2004; Schreiweis *et al.*, 2005; Gao *et al.*, 2006; McElroy *et al.*, 2006; Nones *et al.*, 2006; Zhou *et al.*, 2006; Liu *et al.*, 2007 & 2008; Ambo *et al.*, 2009; Ankra-Badu *et al.*, 2010; Wang *et al.*, 2012). A whole genome scan for QTL affecting body weight and growth in a 3-generation population generated from two broiler lines genetically different was conducted by van Kaam *et al.* (1998 and 1999). The identification and use of QTL in selection programs, therefore, will offer the potential for more rapid genetic improvement.

In the last 15 years, several experimental chicken populations (F<sub>0</sub>, F<sub>1</sub>, F<sub>2</sub> and F<sub>3</sub>) have been constructed from different breeds for use in gene and QTL mapping studies (Jacobsson, 2005; Liu *et al.*, 2008; Bulut *et al.*, 2013). Furthermore, chromosomal scanning studies have been conducted. To exemplify, the chromosomal regions affecting phenotypic traits including body weight have been investigated in different chicken breeds (Van Kaam *et al.*, 1999; Tatsuda and Fujinaka 2001; Sewalem *et al.*, 2002; Carlborg *et al.*, 2003; Kerje *et al.*, 2003; Li *et al.*, 2003; Zhu *et al.*, 2003; Sasaki *et al.*, 2004; Siwek *et al.*, 2004; Gao *et al.*, 2006; Nones *et al.*, 2006). These studies are ongoing on the identification of the quantitative trait genes (QTGs) and quantitative trait nucleotide (QTNs) controlling these traits.

The resource populations used in the present study were generated by crossing Golden Montazah males (GM) with White Leghorn females (WL). The main objectives were: (1) to phenotyping growth traits of body weights and daily body gains in the parental and F<sub>2</sub> generations in such crossbreeding program, (2) to localize QTL affecting these growth traits in the F<sub>2</sub> population using specific microsatellite markers, (3) to detect the chromosome group, number of informative microsatellite markers, chromosome map length (cM) and average marker interval by the chromosome (cM), (4) to estimate QTL at chromosome-wise level along with the proportion of phenotypic variance explained by each QTL, (5) to quantify the additive and dominance effects for QTL, (6) to explain the total variances attributable to QTL for each growth trait.

## Materials and Methods

### Breeding plan and experimental populations

Chicks of F<sub>2</sub> population were produced by crossing males of Golden Montazah (GM) with females of White Leghorn (WL). A total number of 18 and 8 cockerels and 64 and 51 pullets were chosen randomly from the WL and GM strains, respectively. Each cock was mated with 10 hens housed in separately breeding pen to produce F<sub>1</sub> crossbred chicks ( $\frac{1}{2}$ GM $\frac{1}{2}$ WL), then inter-se matings were practiced to produce F<sub>2</sub> chicks with the genetic structure of ( $\frac{1}{2}$ GM $\frac{1}{2}$ WL)<sup>2</sup>. Also, purebreds from the two strains were produced. The breeding plan permitted to produce four genetic groups as presented in Table 1. Pedigreed eggs from each individual breeding pen were collected from the four mating groups. On the hatching day, chicks of all genetic groups were wing banded, brooded on the floor and were grown in open houses up to 16 weeks of age.

All the chicks were vaccinated against common diseases and they were subject to the same managerial, hygienic and climatic conditions. During the growing and rearing periods, all the chicks were fed *ad libitum* a diet containing 23% crude protein and 3200 kcal ME /kg during the period from hatching to 6 weeks and a diet containing 23% crude protein and 2900 kcal ME /kg during 6 to 16 weeks of age.

**Table 1.** Number of sires, dams and chicks for genetic groups used in the experimental work

Generation	Sire group	Dam group	Genetic group <sup>+</sup>	No. of sires	No. of dams	No. of Hatched chicks
Parental	WL	WL	WL × WL	18	64	1002
Parental	GM	GM	GM × GM	8	51	775
F <sub>1</sub>	GM	WL	( $\frac{1}{2}$ GM $\frac{1}{2}$ WL)	18	103	1343
F <sub>2</sub>	F <sub>1</sub> or $\frac{1}{2}$ GM $\frac{1}{2}$ WL	F <sub>1</sub> or $\frac{1}{2}$ GM $\frac{1}{2}$ WL	( $\frac{1}{2}$ GM $\frac{1}{2}$ WL) <sup>2</sup>	18	106	1011
			Total	62	324	4131

<sup>+</sup> WL and GM = White Leghorn and Golden Montazah strains, respectively; the first letter denoted to the sire group.

The detailed breeding plan and management of the experimental populations are presented by **Iraqi et al. (2013)** and **khalil et al. (2013)**.

### The phenotypic measurements

Individual body weights (BW) of 4131 chicks were recorded at hatch and at 4, 8, 12 and 16 weeks of age. The daily gains in weight for these chicks were calculated during the period interval of 0-4 (DG4), 4-8 (DG8), 8-12 (DG12) and 12-16 (DG16) weeks of age.

### Statistical analysis of phenotypic data

The phenotypic data set was firstly analyzed using SAS program (**SAS, 2004**) to estimate the starting values of additive and residual variances to be used as prior values in the animal model analysis. The differences between means of the genetic groups were tested ( $P < 0.05$ ) using **Duncan test (1955)**. Then, the data set was analyzed using multi-traits animal model of VCE6 program (**Groeneveld et al., 2010**). The animal model used in matrix notation was as follows:

$$y = Xb + Zaua + Zcuc + e \quad (\text{Model 1})$$

Where:  $y = n \times 1$  vector of observation of the bird,  $n =$  number of records;  $X =$  design matrix of order  $n \times p$ , which is related to the fixed effects of genetic group (four levels), year of birth (three levels), hatch (two levels) and sex (two levels);  $b = p \times 1$  vector of the fixed effects of genetic group, year, hatch and sex;  $Za =$  the incidence matrix relating records to the additive genetic effect of the bird;  $ua =$  the vector of random additive genetic of the bird;  $Zc =$  the incidence matrix relating records to random common environmental effect of the bird;  $uc =$  the vector of random common environmental effect of the birds; and  $e = n \times 1$  vector of random residual effects, NID ( $0, \sigma^2e$ ).

### Estimation of heritability:

The heritability was estimated using the following equation:

$$h_a^2 = \frac{\sigma_a^2}{\sigma_a^2 + \sigma_c^2 + \sigma_e^2}$$

Where:  $\sigma_a^2$ ,  $\sigma_c^2$  and  $\sigma_e^2$  are variances due to the effects of direct additive genetic, common environmental effect and random error, respectively.

### Estimation of correlations:

The additive genetic correlation ( $r_g$ ) between the traits were estimated according to the formula of **Quaas et al. (1984)**:

$$r = \frac{\text{Cov}(X)_{ij}}{\sqrt{\text{Var}(X_{ii}) \cdot \text{Var}(X_{jj})}}$$

Where:  $\text{Cov}(X)_{ij} =$  the covariance between additive genetic effects on body weight and daily gain;  $X_{ii} =$  the additive genetic (a) variance of body weight;  $X_{jj} =$  the additive genetic (a) variance of daily gain.

### Genotyping

#### Blood sampling and DNA isolation:

Blood samples (10 ml) were collected from the wing vein at 24 weeks of age from relevant mating birds of  $F_0$  parents,  $F_1$  and  $F_2$  to be included in the genotyping panel. Blood samples were collected in vacuum tubes containing EDTA and stored at  $-20^\circ\text{C}$  until DNA extraction. Genomic DNA was extracted using the Maxwell® 16 blood DNA purification kit according to kit manual, designed specifically for the optimal automated extraction of DNA from whole blood samples on the Maxwell® 16 SEV Instrument. The quality and concentration of extracted DNA was examined spectrophotometrically.

#### Markers selected:

A total of 43 microsatellite markers covering nine autosomal linkage groups and the sex Z chromosome were considered in genotyping fifty  $F_0$  grandparents, twenty  $F_1$  and two hundreds  $F_2$  offspring (Table 2). These markers were selected based on the degree of polymorphism and the genome coverage recommended in the molecular genetic characterization of animal genetic resources (**FAO, 2011**). Detailed information about selected microsatellites are available at the FAO website ([www.dad.fao.org/en/refer/library/guidelin/marker.pdf](http://www.dad.fao.org/en/refer/library/guidelin/marker.pdf)). The assessment of markers was based on their positions on the consensus map. A target for marker spacing of 10 cM was used to test markers across the genome (<http://www.ncbi.nlm.nih.gov/mapview> and <http://www.thearkdb.org>).

#### PCR amplification:

The PCR amplification was performed on a 25- $\mu\text{l}$  reaction mixture (ready to use Master Mix Promega) containing 100–200 ng DNA template, 15 pM of each primer, 200 IM each dNTP, 1 U Taq DNA polymerase, and an optimized quantity of  $\text{MgCl}_2$ . The reaction was carried out by initial denaturation at  $94^\circ\text{C}$  for 2 minute, and then denaturation at  $94^\circ\text{C}$  for 30 second, annealing at the temperature optimized for each primer pair for 30 second and extending at  $72^\circ\text{C}$  for 30 second for 35 cycles, followed by a final extension step at  $72^\circ\text{C}$  for 5 minute. The optimum annealing temperatures for the best amplification are presented in Table 2. Amplified products were electrophoresed at Metaphor gel (**Muhammad et al., 2008**). The gel was run with puc19 DNA marker at 120 V for 2 h in 1X TBE and stained with Ethidium Bromide. The gel was visualized and documented under a white light gel documentation system.

**Table 2.** Microsatellite markers used in genotyping birds of F<sub>0</sub>, F<sub>1</sub> and F<sub>2</sub>

Microsatellite marker (Locus)	Forward primer sequence	Reverse primer sequence	SSR (bp)*	T <sub>m</sub> **
ADL0114	GGCTCATAACTACCTTTTT	GCTCTACATTCCCTCAGTCA	185	45
ADL0142	CAGCCAATAGGGATAAAAAGC	CTGTAGATGCCAAGGAGTGC	231	52
ADL0166	TGCCAGCCCGTAATCATAGG	AAGCACCACGACCCAATCTA	135	47
ADL0183	TTGTGAAGTGGATAAGATGA	ACAGAAATGGAAAGCGAGAC	102	47
ADL0188	CACTTCCAGTATTAACGTGA	GTGGACACAATGAGTCCCTC	129	47
ADL0225	CCAAAAAGCTGTATCACCTT	GCCTGTTGTAACCACCTGA	149	48
ADL0236	CTGGTTGTCAAGTGAAGGAC	ATAAGGTGGTGAGCAGCACT	132	51
ADL0237	GCTTGTGCCTAAGAATGAAC	TGTATGGAGTCTCAGCAAAT	148	50
ADL0238	AAACCCAAACAAAAGCAGAC	GCTCCTCATAAGCAAAATGC	160	53
ADL0241	AAAAATAGCATGGCAAATCAT	CAGATGCATCAGCACAGAAA	216	51
ADL0255	GGGTATTGGTCTTCAAAATG	GTAAAGGCCTTCCTCTTCTT	110	47
ADL0258	TCATTTTCAGCTCACATTTTA	TTTTTCAGGTTGTCTGGTTGC	168	48
ADL0266	GTGGCATTTCAGGCAGAGCAG	AATGCATTGCAGGATGTATG	113	50
ADL0267	AAACCTCGATCAGGAAGCAT	GTTATTCAAAGCCCCACCAC	117	55
ADL0280	CCCCTATGACACAGCAGTCC	GGAACTCAGCCTTGACATT	172	56
ADL0317	AGTTGGTTTCAGCCATCCAT	CCCAGAGCACACTGTCCTG	199	51
LEI0073	TTGAGAGCAGTGAAGGCAAACG	TGGTGGAACTGGAAGAAGAGG	217	65
LEI0075	TTTCACATCCAGTGCCTGTCTG	GGGCAGAGAAAGACGAAATTGG	188	65
LEI0083	AACCCTCACACACCATTGCC	CACTCGCCTGTAATTTCTTGTGG	259	65
LEI0106	TGTGGGTTGTAATCCCTTCACC	CTCCCAAAAACCTTCAAATGG	295	59
LEI0110	GGGACCCAAGGCACACACTA	ATCCTCTATGAGGAAGGGAAGTGA	231	63
LEI0111	CCCACAAAAGAGACACCGTGG	CCTGTTTGCCGTACACTTGGC	116	65
LEI0161	CAGCCTTTTCAAGCTTGCTGC	GTTCACCTTAGACATGAATCGG	100	54
LEI0166	AAGCAAGTGCCTGGCTGTGCTC	TCCTGCCCTTAGCTACGCAC	267	54
LEI0254	AGACCACTGGATCCAATC	GTCTGGAACCTCATCCCTTCATC	95	55
MCW0010	CTGTAGAATTACAGAAATACA	TAGTACAAGAATCTAGTGTTAAAA	93	45
MCW0040	ACTCAAAAATGTGGTAGAATATAG	ACCGAAATTGAGCAGAAGTTA	143	55
MCW0080	CCGTGCATTCTTAATTGACAG	GAAATGGTACAGTGCAGTTGG	280	55
MCW0083	TACATTTTCAAGGAATGTTGC	GCCTTTCACCCATCTTACTGT	90	54
MCW0097	GGAGAGCATCTGCCTTCCTAG	TGGTCTTCCAGTCTATGGTAG	309	56
MCW0100	GATCTAAACAAAACAGACACA	TGTAGGCGATTAACATACTTC	90	55
MCW0107	GAACAGAACTCTGTTACTG	TCTGCTTACCTCAACTGACA	121	56
MCW0135	ATATGCTGCAGAGGGCAGTA	CATGTTCTGCATTATTGCTCC	150	57
MCW0169	GATCCCCTTGTAAAGAAAGTG	CCTGACCTTACTGAGCTTGGGA	96	58
MCW0180	GATCACATCACGTTAATTTT	GGTGGAGAAAAGTGAAAGAC	88	55
MCW0295	ATCACTACAGAACCCCTCTC	TATGTATGCACGCAGATATCC	99	55
MCW0305	TCAGAAACAAAGCAGGAGCTG	TGACATCTTCAAACGAGACC	259	55
MCW0340	ATTATCTGATGCATCAGCTGG	CACCATTGTAGCGGAACATC	174	55
ROS0003	GCAAAGTTATTTCAGGAACTTGC	AAGTGGTCCCCTGATTTAACA	250	56
ROS0025	AGATTGCTGGGGGAAAAGT	ACTGAAAACCTGAACAGAAGGC	210	58
ROS0030	CGGAGAGCATGGTTTCAAGT	CTCTGTGAGCTCCCCATCTC	240	58
ROS0074	AGCACTTTTGGTGTACCAGG	CAGCTGATGCTTCCACAGAA	320	58
ROS0075	CAGCTCCGTGCTCCTCTC	TTTTCAACCCGTTGTTTCAGG	216	58

\* SSR = Simple Sequence Repeats; \*\* T<sub>m</sub> = annealing temperature

### Linkage analysis and QTL mapping

A linkage map was generated using Map Manager QTX version b20 software program (Manly *et al.*, 2001). After parentage checking, data of 1011 chicks from F<sub>2</sub> individuals were genotyped using 43 microsatellite markers in nine autosomal linkage groups and Z chromosome and these genotypes were available for QTL analysis. Markers that did not meet the criteria of polymorphism were avoided from the analysis. The linkage map analysis was used to get the best order of the markers, and to detect the map distance among the markers. The maps were then used for QTL detection on the autosomes, linkage groups, and the Z chromosome. Data of F<sub>2</sub> was used for analyzing the additive (a) and dominance effects (d) of QTL at a given position

for each trait where the additive effect was defined as half the difference between the two homozygotes and the dominance effect as the difference between the means of the heterozygotes and homozygotes. Data of F<sub>2</sub> cross was analyzed using the following mixed model including the fixed effects of hatch and sex along with the additive and dominance effects of QTL as random effects (Haley *et al.*, 1994; Manly *et al.*, 2001):

$$y_{ij} = X_{ij}b + Z_a a + Z_d d + e_i \text{ (Model 2)}$$

Where:  $y_{ij}$  is the phenotype of F<sub>2</sub> birds,  $X_{ij}$  is the designed matrix, and  $b$  is the vector of coefficients for sex and hatch as fixed effects,  $a$  is the vector of additive effect of the QTL,  $d$  is the vector of dominance effect of the QTL,  $Z_a$  the probability of one homozygous type at the putative QTL locus

given the marker information minus the probability of the other homozygous type at the locus given the marker information for the bird  $i$ ,  $Z_d$  is the probability of being heterozygous at the putative QTL locus given marker genotypes for the bird  $i$ , and  $e_i$  is the random error, typically assumed to be normally distributed as  $N(0, \sigma^2)$  (Haley and Knott, 1992). Detection of QTL was based on an F-statistic that was computed from sums of squares explained by the additive and dominance coefficients for the QTL. Additive and dominance effects were estimated for each putative QTL. The informativeness of the markers was assessed at each location as described by Knott *et al.* (1998). Significance thresholds at 1% and 5% levels, and confidence intervals were determined by Map Manager QTX software. Significant and suggestive QTL were defined by test statistics exceeding the 5% significance thresholds. The 5% chromosome-wise level threshold was used as suggestive QTL, and the 5% genome-wise level threshold was used as significant QTL, namely,  $P_{\text{genome}} = \alpha/n$ , where  $\alpha = 0.05$ ,  $n$  was the total number of tests (traits x chromosome).

Percentage of  $F_2$  phenotypic variance explained by the model was calculated as:

**Phenotypic variance percentage = 100 x (RMS – FMS)/RMS**

Where: RMS = the residual mean square from the reduced model, omitting QTL but including all fixed effects, and FMS = the residual mean square from the full model, including QTL and all fixed effects.

The Likelihood ratio test was performed as:

$$n \log_e \left( \frac{\text{residual sum of squares reduced model}}{\text{residual sum of squares full model}} \right)$$

Where:  $n$  is the number of observations. This test statistics distributed approximately as a chi-square with degrees of freedom equal to the number of parameters included in the full model (i.e., estimating the QTL effects) but omitted from the reduced model (i.e., omitting QTL).

## Results and Discussion

### Phenotypic means of genetic groups:

Means presented in Table 3 showed that GM strain was significantly better ( $P < 0.05$ ) in most of the body weight and daily gain traits compared to WL breed. But, WL strain was higher than GM strain in BW0 and DG8-12. This superiority may be due to the genetic makeup of GM strain and the genotype-environment interaction that favours for GM strain over WL breed (El-Labban, 2000).

Crossbred chicks were superior ( $P < 0.05$ ) for most growth traits, probably due to genetic and non-genetic additive effects of genes. Afifi *et al.* (2002), Iraqi *et al.* (2002), Khalil and Al-Homiadan (2003), Iraqi *et al.* (2013) and Mahmoud and El-Full (2014) found that crossbreeds were significantly ( $P < 0.01$ ) superior in growth traits compared to the foundations. In general, the overall performances of the crossbred chickens of  $(1/2GM1/2WL)$  and  $(1/2GM1/2WL)^2$  were found to be better than those for local chickens of GM (Galal *et al.*, 2007; Iraqi *et al.*, 2013).

**Table 3.** Means and standard errors (SE) for growth traits in Golden Montazah (GM), White Leghorn (WL) and their crosses of chickens

Trait	Symbol	Genetic group			
		GM Mean $\pm$ S.E (N= 775)	WL Mean $\pm$ S.E (N= 1002)	$1/2GM1/2WL$ Mean $\pm$ S.E (N= 1343)	$(1/2GM1/2WL)^2$ Mean $\pm$ S.E (N= 1011)
<b>Body weight traits (g):</b>					
0 week	BW0	33.3 $\pm$ 0.13 <sup>b</sup>	34.1 $\pm$ 0.12 <sup>a</sup>	29.6 $\pm$ 0.10 <sup>d</sup>	32.3 $\pm$ 0.12 <sup>c</sup>
4 weeks	BW4	221.4 $\pm$ 1.92 <sup>c</sup>	216.7 $\pm$ 1.67 <sup>c</sup>	250.8 $\pm$ 1.47 <sup>a</sup>	234.9 $\pm$ 1.68 <sup>b</sup>
8 weeks	BW8	601.6 $\pm$ 4.90 <sup>b</sup>	515.2 $\pm$ 4.23 <sup>d</sup>	640.9 $\pm$ 3.74 <sup>a</sup>	554.2 $\pm$ 4.32 <sup>c</sup>
12 weeks	BW12	977.3 $\pm$ 8.25 <sup>bc</sup>	914.4 $\pm$ 7.13 <sup>d</sup>	1121 $\pm$ 6.25 <sup>a</sup>	992.4 $\pm$ 7.29 <sup>b</sup>
16 weeks	BW16	1347 $\pm$ 11.90 <sup>d</sup>	1279 $\pm$ 10.27 <sup>e</sup>	1517 $\pm$ 8.98 <sup>a</sup>	1490 $\pm$ 10.46 <sup>b</sup>
<b>Daily gain traits (g):</b>					
0-4 weeks	DG04	6.71 $\pm$ 0.06 <sup>d</sup>	5.51 $\pm$ 0.05 <sup>e</sup>	7.90 $\pm$ 0.05 <sup>a</sup>	7.23 $\pm$ 0.06 <sup>b</sup>
4-8 weeks	DG48	13.52 $\pm$ 0.14 <sup>b</sup>	10.65 $\pm$ 0.12 <sup>d</sup>	13.92 $\pm$ 0.10 <sup>a</sup>	11.34 $\pm$ 0.12 <sup>c</sup>
8-12 weeks	DG812	13.26 $\pm$ 0.17 <sup>c</sup>	14.14 $\pm$ 0.15 <sup>d</sup>	17.06 $\pm$ 0.13 <sup>a</sup>	15.43 $\pm$ 0.15 <sup>b</sup>
12-16 weeks	DG1216	13.26 $\pm$ 0.21 <sup>d</sup>	13.11 $\pm$ 0.18 <sup>d</sup>	14.23 $\pm$ 0.16 <sup>c</sup>	17.78 $\pm$ 0.19 <sup>a</sup>

<sup>a-e</sup> Means with the same letters within each row of the trait are non-significantly different ( $P \leq 0.05$ ).

### Heritability

Estimates of heritability ( $h^2$ ) for growth traits in genetic group of  $(1/2GM1/2WL)^2$  are presented in Table 4. The estimates showed that these growth traits are highly heritable; the estimates ranging from 0.43 to 0.52. Thus, we would recommend the selection for growth in these strains at early ages, so time and

efforts can be saved. Estimates of  $h^2$  in the present study were generally within the range of those estimates obtained for the same strains by Khalil *et al.* (1991) and Iraqi *et al.* (2000).

### The genetic and phenotypic correlations

The genetic and phenotypic correlations among growth traits in the  $F_2$  population are presented in

Table 4. As expected, there were moderate or high positive correlations between the growth traits studied.

**Table 4.** Heritabilities (diagonals), genetic (above diagonals), and phenotypic (below diagonals) correlations of investigated traits

Trait <sup>+</sup>	BW4	BW8	BW12	BW16	DG04	DG48	DG812	DG1216
BW4	<b>0.51</b>	0.25	0.22	0.11	0.20	0.18	0.11	0.10
BW8	0.25	<b>0.45</b>	0.64	0.58	0.20	1.00	0.29	0.29
BW12	0.19	0.59	<b>0.52</b>	0.75	0.54	0.63	0.92	0.23
BW16	0.17	0.62	0.75	<b>0.43</b>	0.50	0.58	0.63	0.73
DG04	0.20	0.25	0.53	0.53	<b>0.46</b>	0.19	0.59	0.31
DG48	0.18	1.00	0.58	0.62	0.24	<b>0.45</b>	0.29	0.29
DG812	0.12	0.19	0.90	0.57	0.48	0.18	<b>0.51</b>	0.11
DG1216	0.09	0.33	0.24	0.76	0.38	0.33	0.08	<b>0.47</b>

<sup>+</sup> Traits as defined in Table 3.

### Chromosomal linkage analysis

The chromosome group, number of informative microsatellite markers, chromosome map length (cM), average marker interval by the chromosome (cM) and the first marker on each chromosome that was used for a whole genome scan in F<sub>2</sub> cross are presented in Table 5. Ultimately, nine autosomal linkage groups, and the Z chromosome containing 47 microsatellite markers in the F<sub>2</sub> cross were used for linkage analysis.

The total chromosomal map length was 1901 cM ranging from 25 cM on chromosome 11 to 568 cM on chromosome 1, with an average marker spacing of 14.48 cM and that ranging from 7.8 cM on chromosome 8 to 24.3 cM on chromosome 1. Map lengths for these chromosomes were considerably similar to those cited in the chicken consensus map reported by Zhou *et al.* (2006). Ikeobi *et al.* (2002) stated that the total map length was 2923 cM or about 75% of the consensus linkage map and the average marker interval was 40 cM. Zhou *et al.* (2006) in F<sub>2</sub> population of broiler-Leghorn cross and broiler-Fayoumi cross reported that the QTL covered a 20 to 30 cM chromosome region and this size region may contain many candidate genes. The same authors concluded that chromosome 1 had potential positional candidate genes like growth hormone 1, lysosomal associated membrane protein 1, and uncoupling protein 2. The potential candidate genes mapped in the region on chromosome 2 are transforming growth factor- $\beta$  (TGFB) type I receptor and pituitary adenylate cyclase-activating

polypeptide 1. The TGFB type II receptor is mapped on chromosome 4 nearby QTL affecting growth traits. A potential candidate gene on chromosome 10 is insulin-like growth factor type 1 receptor. Growth hormone gene has been associated with growth in chickens (Kuhn *et al.*, 2002). The insulin-like growth factor and TGFB family genes have previously shown associations with growth-related traits in chickens (Amills *et al.*, 2003; Li *et al.*, 2003; Zhou *et al.*, 2005). So far, no association has been found for the genes above with growth-related traits in chickens. Nassar *et al.* (2012) found that the most genomic region affecting body weight was mapped on chromosome 4 at 155 cM.

### Estimates of QTL mapping

The flanking markers, position of QTL relative to the first marker (cM), F-ratio and significant for each QTL at chromosome-wise level along with the proportion of phenotypic variance explained by each QTL for body weights and daily gains in weight are presented in Tables 6 and 7. The results in the current study lay the foundations for fine mapping of the traits in the advanced intercross lines and provide a start point for identifying the causative genes responsible for growth traits in chickens. In Brazil, a layer (CC) and a broiler (TT) lines were crossbred to generate two F<sub>2</sub> reciprocal populations (TCTC and CTCT) to map QTL (Nones *et al.*, 2006; Ambo *et al.*, 2009; Campos *et al.*, 2009; Baron *et al.*, 2011; Nones *et al.*, 2012; Boschiero *et al.*, 2013).

**Table 5.** Chromosome (linkage) group, number of microsatellite markers, map length (cM), marker intervals and the first marker on each chromosome that was used for a whole genome scan of F<sub>2</sub> cross

Chromosome	Number of microsatellite markers	Chromosome map length (cM)	Average marker spacing by the chromosome (cM)	First marker on each chromosome
1	10	568	24.3	ROS0003
2	8	298	18.7	LEI0073

3	2	273	11.6	MCW0169
4	7	198	17.6	ADL0317
6	4	111	10.4	ADL0280
8	3	97	7.8	MCW0080
9	1	123	20.1	ROS0074
11	5	25	8.3	LEI0110
13	2	71	14.5	MCW0340
Z	5	137	11.5	LEI0075
Total	47	1901	14.48	

**Table 6.** Flanking markers, position of QTL relative to the first marker (cM), F-ratios and significance of QTL at chromosome-wise level confidence interval at 95% ( $cM$ ) for body weights at 4, 8, 12 and 16 weeks of age in phenotypic population of chickens along with the percentage of F<sub>2</sub> variance explained by each QTL

Trait / Chromosome	Flanking markers	Position of QTL relative to the first marker (cM)	F-ratio for each QTL at chromosomal wise level	Confidence interval at 95% (cM)	Proportion of phenotypic variance explained by each QTL
<b>4-weeks weight:</b>					
1	ADL0183-ROS0025	502	4.6†	74-615	2.4
2	ADL0236-ROS0074	292	16.1**	43-367	5.8
4	ADL0266-LEI0073	145	8.8*	12-183	3.1
6	ROS0003 - ADL0142	29	9.6*	0-42	2.6
8	MCW0100- ROS0075	62	7.6†	1-87	2.1
11	LEI0110 - MCW0097	0	12.5**	0-10	1.2
13	LEI0083 - MCW0080	50	5.6†	9-71	1.6
Z	LEI0111 - LEI0075	125	6.9†	0-125	2.3
<b>8-weeks weight:</b>					
1	MCW0010-ADL0188	128	17.0**	76-219	4.9
2	ADL0236-ROS0074	150	5.1†	34-370	1.3
3	LEI0161-ADL0280	49	11.4*	14-219	3.0
3	MCW0040-LEI0166	233	5.4†	12-266	1.5
4	ADL0317 - MCW0295	0	8.2*	0-69	2.5
4	ADL0266-LEI0073	159	23.5**	140-183	7.0
8	MCW0100-ROS0075	67	7.5†	0-87	2.5
11	LEI0110-MCW0097	0	12.1**	0-57	3.5
13	MCW0340-ADL0225	44	5.6†	12-71	1.6
Z	LEI0111-LEI0075	117	9.6**	14-127	3.0
<b>12-weeks weight:</b>					
1	MCW0010-ADL0188	133	11.9**	67-227	3.3
3	ADL0237-ADL0166	37	10.0*	155-183	3.0
4	ADL0317-MCW0295	0	8.4*	0-177	2.4
4	ADL0266-LEI0073	179	44.5**	155-183	13.8
8	MCW0100- ROS0075	59	13.2**	12	1.4
9	MCW0135- ROS0030	90	5.0†	0	1.3
13	MCW0340-ADL0225	8	5.1†	0-71	1.4
Z	LEI0111-LEI0075	120	8.9*	8-127	2.7
<b>16-weeks weight</b>					
1	MCW0010-ADL0188	129	6.4†	109-543	2.5
1	ADL0183-ROS0025	555	5.3†	96-598	1.6
2	ADL0236-ROS0074	277	5.7†	0-297	1.9
4	ADL0241-MCW0180	139	16.9**	19-169	6.5
8	MCW0305-ADL0258	12	11.5**	0-86	4.2
8	MCW0100-ROS0075	87	6.2†	14-87	2.3
13	MCW0340-ADL0255	69	7.0†	2.0-71.0	2.8
Z	LEI0111-LEI0075	125	9.3**	0-125	3.6

Total QTL detected = 34.

† Suggestive linkage; \*significant linkage at  $P \leq 0.05$  and \*\* significant linkage at  $P \leq 0.01$ .

**Table 7.** Flanking markers, position of QTL relative to the first marker (cM), F-ratios and significance of QTL at chromosome-wise level confidence interval at 95% (cM) for daily gain at 0-4, 4-8, 8-12 and 12-16 weeks of age in F<sub>2</sub> population of chickens along with the percentage of phenotypic variance explained by each QTL

Trait / Chromosome	Flanking markers	Position of QTL relative to the first marker (cM)	F-ratio for each QTL at chromosomal wise level	Confidence interval at 95% (cM)	Proportion of phenotypic variance explained by each QTL
Daily gain 0-4 week:					
1	ROS0025-ADL0238	452	9.15*	69-437	4.99
2	ADL0267-ADL0236	239	12.88**	80-504	6.89
4	ADL0317-MCW0295	398	10.87**	104-310	5.95
4	ADL0241-MCW0180	418	11.66**	154-208	6.03
13	MCW0340-ADL0225	67	5.82†	32-165	2.04
Daily gain 4-8 week:					
1	ADL0183-LEI0106	0	7.61†	0-37	4.19
2	ROS0074-ADL0114	248	9.80**	15-384	4.81
4	ADL0317-MCW0295	428	16.88**	65-540	8.88
4	ADL0241-MCW0180	436	15.46**	98-506	7.68
8	ROS0026-MCW0305	22	5.56†	0-32	3.54
Daily gain 8-12 week:					
1	ADL0183-MCW0107	512	9.83**	106-584	3.05
3	MCW0169-MCW0083	26	10.02**	0-186	4.12
4	ADL0241-MCW0180	168	18.99**	138-198	2.18
Daily gain 12-16 week:					
8	ROS0025-MCW0305	17	9.76**	0-158	3.9

Total QTL detected = 14.

† Suggestive linkage; \*significant linkage at  $P \leq 0.05$  and \*\* significant linkage at  $P \leq 0.01$ .

For daily body gains (DG), a total of 14 QTL were detected (Table 7). These QTL were distributed over 7 distinct regions on 6 chromosomes. A total of 11 genome significant QTL that affected daily gain were located on five macro-chromosomes (1, 2, 3, 4 and 8). There was statistical evidence for two QTL on chromosome 4 for daily gains at 0-4, 4-8 and 8-12 weeks of age. A further three suggestive QTL were identified for daily gain at DG4-8 and DG0-4 on chromosomes 1, 8 and 13. Similar results were obtained by **Carlborg et al. (2003)**, **Jennen et al. (2004)**, **McElroy et al. (2006)** and **Rosario et al. (2014)**.

The position of QTL relative to the first marker (cM) given in Table 6 indicated that QTL were located in the region of 0 to 502 cM, 0 to 233 cM, 0 to 179 cM and 12 to 555 cM for body weights at 4, 8, 12 and 16 weeks of age, respectively. For daily gains, the position of QTL relative to the first marker (cM) given in Table 7 indicated that QTL were located in the region of 67 to 452 cM, 0 to 436 cM, 26 to 512 cM and 17 cM for daily gain intervals at 0-4, 4-8, 8-12 and 12-16 weeks, respectively. **Wang et al. (2012)** stated that the QTL for body weight at 2 to 5 and 8 to 10 week of age were located in the region

of 89 to 104 cM and the QTL for body weight at 6, 7, 10 to 12 week of age located in the region of 246 to 248 cM.

For body weights evaluated in F<sub>2</sub> cross, a total of 34 QTL were detected and these QTL were distributed over five distinct regions on 10 chromosomes (Table 6). A total of 19 genome significant QTL that affecting body weight were located on seven macro-chromosomes (chromosomes 1, 2, 3, 4, 6, 8 and Z) and one micro-chromosome (chromosome 11). There was statistical evidence for two QTL on chromosome 4 for body weight at 8 and 12 weeks of age. A further 15 suggestive QTL were identified for body weight at different ages on chromosomes 2, 6, 9 and 13.

Previous QTL mapping indicated that chromosome 3 harboured QTL regions are responsible for body weight at different ages (**Ikeobi et al., 2002**; **Wardecka et al., 2002**; **Kerje et al., 2003**; **Siwek et al., 2004**; **Tuiskula-Haavisto et al., 2004**; **Zhou et al., 2006**). **Siwek et al. (2004)** using 174 microsatellite markers detected QTL for body weights at 4, 6, 8, 12, and 18 week of age in an experimental F<sub>2</sub> cross of layers applying two genetic models in the QTL analysis: a half-sib model and a



line-cross model. In the half-sib model, three QTL were detected for body weight at the 4<sup>th</sup> week of age on chromosomes 2, 3, and 9; three QTL for body weight at the 6<sup>th</sup> week of age on chromosomes 2, 3, and 6; one QTL for body weight at the 8<sup>th</sup> week of age on chromosome 7, and one QTL for body weights at 12 and 18 weeks of age on chromosome Z. With the line-cross analysis model, one QTL was detected on chromosome 7 for body weight at the 4<sup>th</sup> week of age, two QTL on chromosomes 3 and 7 for body weight at the 6<sup>th</sup> week of age, and one QTL on chromosome 3 for body weights at 8 and 12 weeks of age, and there was no QTL for body weight at 18 weeks of age. **Rosario et al. (2014)** detected five QTL on chromosomes 1, 3 and 4 for body weight at 35 days of age, five QTL for body weight at 41 days of age on chromosomes 1, 3 and 4. Three QTL for body weight at 35 days and two QTL for body weight at 41 days of age were identified on chromosome 4. **De Koning et al. (2003 and 2004)** validated the presence of QTL for body weight in a commercial broiler line. **Zhu et al. (2003)** detected potential QTL for growth to be located on chromosomes 1, 6, and 8.

The QTLs detected in F<sub>2</sub> population in the present study are similar to those obtained by **Sewalem et al. (2002)**, in which a F<sub>2</sub> population was generated from a commercial broiler line and White Leghorn line. More QTL were detected by **Sewalem et al. (2002)** for body weights at 3, 6, and 9 weeks of age on chromosomes 4, 8, and 13. In this study, one out of 4 QTL on chromosome 3 was suggestive (Tables 6 & 7). **Carlborg et al. (2003); Jennen et al. (2004) and McElroy et al. (2006)** reported that QTL for growth was detected on chromosome 3. The QTL detected for growth on chromosomes 1, 2, 3, 4, 6, 8, 11 and Z in the present study were also found in F<sub>3</sub> population generated from crossing two White Plymouth Rock broilers (**Jennen et al., 2004**) and in F<sub>2</sub> population generated by Red Jungle Fowl and White Leghorn line (**Carlborg et al., 2003**). Several QTL for growth traits on chromosomes 11, 12, and 15 were reported in other studies (**Carlborg et al., 2003; Kerje et al., 2003**). **Carlborg et al. (2003)** and **McElroy et al. (2006)** detected QTL for growth on chromosomes 20 and 26. **Zhou et al. (2006)** reported that most of the QTL for growth traits were detected in chromosomes 1, 2, 4, 7, and 14 for the broiler-Leghorn cross and chromosomes 1, 2, 4, 5, 8, and 13 for the broiler-Fayoumi cross, i.e. majority of the QTL detected for growth traits were similar between the two line crosses. Moreover, they mentioned that there were no QTL affecting growth-related traits detected on chromosomes 11, 12, 13, 15, 17, 27, and Z in the broiler-Leghorn cross, and there were no QTL detected on chromosomes 10, 11, 12, 15, 17, 18, 24, 27, E46, E47, and Z in the broiler-Fayoumi cross. **Bulut et al. (2013)** using Denizli X White Leghorn F<sub>2</sub> populations and a total of 113 microsatellite markers, demonstrated that QTL regions associated with body weight at different age periods were

located on chromosomes 1, 2, 4, 8 and Z and the distances between the QTL regions were wide (>30 cM). Therefore, the relevant QTL intervals should be narrowed by the use of new markers.

The F-ratios for each QTL at chromosome-wise level illustrated in Table 6 for different body weights showed that 19 out of 34 QTL were significant ( $P < 0.05$  or  $P < 0.01$ ). **Schreiweis et al. (2005)** reported that five QTL influencing body weight at 35 or 55 week of age were identified on chromosomes 4, 12, and 27, and four of them were located on chromosomes 4 and 27 and surpassed a 1% genome-wise significance threshold. Each of the significant QTL is associated with an increase in body weight from the broiler allele, while the suggestive QTL is primarily associated with dominant gene action. While, **Liu et al. (2007)** reported 10 QTL identified at the 1% chromosome wide level, two QTL identified at the 5% chromosome wide level, and five QTL identified at the suggestive level for body weight. **Wang et al. (2012)** found on chromosome 3 that three QTL were identified at the 5% chromosome-wide level and 10 QTL were suggestive.

#### Confidence intervals

For confidence intervals of 4-week body weight, four significant QTL were located on chromosomes 2, 4, 6 and 11 at position of 292, 145, 29 and 0 cM, respectively, in which 95% confidence intervals were 43–367, 12–183, 0–42 and 0–10 cM, respectively. For 8-week body weight, another significant QTL was located on chromosomes 1, 3, 4, 11 and Z sex chromosome at position of 128, 48, 0, 159, 0 and 117 cM, respectively with 76–219, 14–219, 0–69, 140–183, 0–57 and 14–127 cM of the 95% confidence interval. For 12-week body weight, six significant QTL were located on chromosomes 1, 3, 4, 8 and Z at positions of 133, 37, 0, 179, 59 and 120 cM respectively, in which 95% confidence intervals were 67–227, 155–183, 0–177, 155–183, 12 and 8–127 cM, respectively. Moreover, at 16-week body weight, significant QTL was located on chromosomes 4, 8 and Z at position of 139, 12, and 125 cM, respectively, with 19–169, 0–86 and 0–125 cM of the 95% confidence intervals. **Soller et al. (2006)** reported that fine-mapping of QTL and the identification of causal gene and underlying genes still remains one of the major challenging tasks because the confidence interval of most reported QTL covers more than 20 cM.

**Van Kaam et al. (1999)** 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 chromosome one at 235 cM. **Tatsuda and Fujinaka (2001)** identified two significant QTL for growth using a crossing population between a Satsumadori line and a White Plymouth Rock line. One QTL identified on

chromosome one was located at 220 cM. **Sewalem et al. (2002)** performed a genome scan for growth using a crossing between a White Leghorn line and a commercial broiler sire line. Two significant QTL of 145 and 481 cM for 3-week body weight were located on chromosome one, in which 95% confidence intervals were 113–217, and 441–526 cM. Another significant QTL for 9-week body weight was located on chromosome one at 414 cM with 34–419 cM of the 95% confidence interval. Also, **Kerje et al. (2003)** identified two major QTL for growth, which were located on chromosome one using a crossing population between Red Jungle Fowl and White Leghorn. The two major QTL for growth were located around positions of 68 and 416 cM.

The effects of QTL expressed as the percentage of phenotypic variance explained by each QTL were mostly of considerable importance ranging from 1.2 to 13.8 % of the phenotypic variation for body weights and from 2.04 to 8.9 % for daily gains in weight (Tables 6 & 7). The largest proportion of the phenotypic variation explained by a QTL was 13.8% for 12-week body weight at 179 cM on chromosome 4 (Table 6). The proportions of phenotypic variation explained by significant and suggestive QTL for body weight at 4, 8, 12 and 16 weeks were 21.1, 30.8, 29.3 and 25.4%, respectively. The proportions explained by significant and suggestive QTL for daily gain 0-4, 4-8, 8-12 and 12-16 weeks were 25.9, 29.1, 9.35 and 3.9%, respectively (Table 7). The largest proportion of the phenotypic variation explained by a QTL was 8.88% for DG 4-8 week at 428 cM on chromosome 4. **Zhou et al. (2006)** found that the phenotypic trait variances explained by QTL ranged from 2.24 to 10.12% in the broiler-Leghorn cross and from 2.94 to 9.14% in the broiler-Fayoumi cross. **Rosario et al. (2014)** reported that the phenotypic variance attributable by each QTL for body weight at 35 and 41 days of age were 10.76 and 10.75 %, respectively.

In general, results of QTL mapping of the present study are in agreement with the previous studies that have identified numerous QTL affecting body weights at different ages in chickens (**Tatsuda and Fujinaka 2001; Deeb and Lamont 2002; Sewalem et al., 2002; Kerje et al., 2003; Siwek et al., 2004; Jacobsson et al., 2005; Zhou et al., 2006; Atzmon et al., 2007, 2008; Ambo et al., 2009; Wahlberg et al., 2009; Goraga et al., 2011; Bulut et al., 2013**).

#### Additive and dominance effects for QTL

**Table 8.** Estimates of additive and dominance effects (g) attributable to QTL and their standard errors for body weights at 4, 8, 12 and 16 weeks of age in F<sub>2</sub> population of chickens.

Trait / Chromosome	Additive effect, g	SE	VP <sub>a</sub> (%) <sup>+</sup>	Dominance effect, g	SE	VP <sub>d</sub> (%) <sup>++</sup>
4-weeks weight (overall mean ± SE = 234.9 ± 1.68)						
1	11.6	4.8	4.9	13.4	12.8	5.7

Details of the additive and dominance effects of the 19 significant QTL for body weights are presented in Table 8. The additive effects were positive, while the dominance effects were generally negative or not significant with the exception of body weight at 4, 8, 12 and 16 weeks of age (QTL on chromosomes 2, 3, 4, 8, 11 and Z). **Wang et al. (2012)** found that positive additive effects, indicating that increasing body weight allele was inherited from the broiler line in F<sub>2</sub> population cross of broiler sire with Bair layer dams (Chinese local breed). Using 174 microsatellite markers, **Siwek et al. (2004)** found that additive effects for QTL detected for body weight at 4, 6, 8, 12, and 18 week of age in F<sub>2</sub> cross were positive on chromosome 7, while the negative additive effects for QTL were detected on chromosome 3. **Zhou et al. (2006)** with a broiler-Leghorn cross and a broiler-Fayoumi cross found that most of the additive effects explained by QTL detected in the study were positive in the broiler-Leghorn cross, and negative in the broiler-Fayoumi cross, which means that alleles of broiler-Leghorn cross and broiler-Fayoumi cross were generally superior in weight and growth relative to both Leghorn and Fayoumi alleles. In F<sub>2</sub> population obtained by crossing males from a layer line (CC) and females from a broiler line (TT), **Rosario et al. (2014)** cited that most QTL presented negative additive effects. These results indicated that the alleles that increase body weights came from broiler line on chromosome 4, while most of the dominance effects were negative except for body weight at 35 days of age, indicating that heterozygotes were heavier than mid-parents.

The estimates of the additive effects attributable to QTL were of considerable importance and ranged from 11.1 to 25.8 g, 18.5 to 94.5 g, 25.8 to 205.7 g and 63.2 to 369.2 g for body weights at 4, 8, 12 and 16 weeks of age, respectively (Table 8). Also, the dominance effects attributable magnitude ranging from -18.6 to 16.4 g, -34.9 to 33.0 g, 127.2 to 155.7 g and -188.1 to 110.1 g for body weights at 4, 8, 12 and 16 weeks of age, respectively (Table 8). The largest additive effect (369.6 ± 64.6 g) was for QTL of body weight at 16 weeks of age on chromosome 4 at 179 cM (Table 6). The largest dominance effect (-188.1 ± 55.0 g) was for a QTL of body weight at 16 weeks on chromosome 4 at 139 cM (Table 8).

The percentage of additive variance explained by each QTL for body weights were mostly moderate and ranged from 2.6% to 24.8%, and the percentage of dominance variance ranged from -2.8 % to 15.7%.

2	13.9	3.2	5.9	16.4	5.5	7.0
4	25.8	6.7	11.0	-6.5	23.8	-2.8
6	11.1	3.1	4.7	-11.1	4.9	-4.7
8	13.6	4.1	5.8	15.4	8.5	6.6
11	13.2	2.7	5.6	7.3	3.9	3.1
13	13.9	4.8	5.9	-18.6	11.8	-7.9
Z	12.8	4.0	5.4	3.3	4.0	1.4
8-weeks weight (overall mean $\pm$ SE = 554.2 $\pm$ 4.3)						
1	43.4	7.5	7.8	-2.8	11.9	-0.5
2	42.1	14.7	7.6	-34.9	43.4	-6.3
3	48.7	10.2	8.8	11.8	17.4	2.1
3	18.5	8.2	3.3	33.0	12.0	6.0
4	33.1	7.4	6.0	-1.5	10.9	-0.3
4	94.5	14.6	17.1	6.0	40.7	1.1
8	43.2	11.5	7.8	25.3	23.0	4.6
11	32.3	7.4	5.8	13.4	10.6	2.4
13	47.2	14.0	8.5	-6.8	37.7	-1.2
Z	52.8	12.2	9.5	19.8	13.2	3.6
12-weeks weight (overall mean $\pm$ SE = 992.4 $\pm$ 10.5)						
1	85.5	16.9	8.6	-5.5	26.3	-0.6
3	90.1	20.1	9.1	-5.7	35.1	-0.6
4	63.0	15.2	6.3	-4.0	22.6	-0.4
4	205.7	22.2	20.7	15.6	44.4	1.6
8	72.0	23.1	7.3	155.7	46.0	15.7
9	25.8	21.9	2.6	-127.2	43.9	-12.8
13	48.6	18.4	4.9	54.0	32.5	5.4
Z	112.0	25.5	11.3	32.2	27.9	3.2
16-weeks weight (overall mean $\pm$ SE = 1490 $\pm$ 10)						
1	90.9	26.1	6.1	26.2	36.9	1.8
1	93.1	34.2	6.2	91.0	66.8	6.1
2	93.9	27.3	6.3	-6.0	44.4	-0.4
4	369.6	64.6	24.8	-188.1	55.0	-12.6
8	107.3	25.4	7.2	105.3	39.1	7.1
8	108.2	32.0	7.3	-72.6	48.8	-4.9
13	63.2	31.1	4.2	-155.5	47.7	-10.4
Z	137.7	35.5	9.2	110.1	38.1	7.4

<sup>+</sup>VP<sub>a</sub> (%) = Percentage of additive variance explained by each QTL.

<sup>++</sup>VP<sub>d</sub> (%) = Percentage of dominance variance explained by each QTL.

As for body weights, all the additive effects detected in daily gains were also positive, and most of the dominance effects were negative (Table 9). The estimates of the additive effects explained by QTL were positive and of moderate magnitude ranging from 1.20 g on chromosome 2 to 1.77 g on chromosome 4 for DG 0-4 weeks, from 1.39 g on chromosome 1 to 3.89 g on chromosome 4 for DG 4-8 weeks, from 1.38 g on chromosome 2 to 3.84 g on chromosome 4 for DG 8-12 weeks and 1.21 g on chromosome 8 for DG 12-16 weeks. On the other hand, the estimates of dominance effects attributable

to QTL were mostly negative, i.e. nine estimates out of 14 QTL were negative. The smallest dominance effect was recorded on chromosome 3 for DG 8-12 week (-2.09 g), while the largest dominance effect was recorded on chromosome 4 for DG 4-8 week (1.44 g).

The percentage of additive variance explained by each QTL for daily gains were moderate and ranged at different intervals from 6.8% to 34.3%, while, the percentages of dominance variance ranged from -14.8% to 12.7%.

**Table 9.** Estimates of additive and dominance effects (g) attributable to QTL and their standard errors for daily gains at 0-4, 4-8, 8-12 and 12-16 weeks of age in F<sub>2</sub> population of chickens

Trait / Chromosome	Additive effect, g	SE	VP <sub>a</sub> (%) <sup>+</sup>	Dominance effect, g	SE	VP <sub>d</sub> (%) <sup>++</sup>
Daily gain 0-4 week (overall mean $\pm$ SE = 7.23 $\pm$ 0.06)						

1	1.30	0.30	18.0	-0.20	0.43	-2.8
2	1.20	0.24	16.6	-0.57	0.44	-7.9
4	1.27	0.25	17.6	-0.29	0.32	-4.0
4	1.77	0.39	24.5	0.62	0.74	8.6
13	1.42	0.42	19.6	-0.52	0.88	-7.2
Daily gain 4-8 week (overall mean $\pm$ SE = 11.34 $\pm$ 0.12)						
1	1.39	0.58	12.3	-1.68	0.73	-14.8
2	1.86	0.48	16.4	0.45	0.80	4.0
4	3.18	0.59	28.0	1.44	0.73	12.7
4	3.89	0.81	34.3	0.87	1.02	7.7
8	3.22	1.25	28.4	-0.33	1.15	-2.9
Daily gain 8-12 week (overall mean $\pm$ SE = 15.4 $\pm$ 0.15)						
1	1.65	0.57	10.7	-1.99	0.98	-12.9
3	1.38	0.31	9.0	-2.09	0.88	-13.6
4	3.84	0.44	24.9	0.08	0.66	0.5
Daily gain 12-16 week (overall mean $\pm$ SE = 17.8 $\pm$ 0.2)						
8	1.21	0.32	6.8	-1.18	0.36	-6.6

<sup>+</sup>VP<sub>a</sub> (%) = Percentage of additive variance explained by each QTL.

<sup>++</sup>VP<sub>d</sub> (%) = Percentage of dominance variance explained by each QTL.

### Total variances explained by QTL for each growth trait

The total variances explained by QTL for each growth trait were 21.1, 30.8, 31.7, 25.4, 25.9, 29.1, 9.35 and 3.9 % in BW4, BW8, BW12, BW16, DG04, DG48, DG812 and DG1216, respectively (Table 10). Across the traits studied, a total of 18 significant QTL were detected at a 5 % chromosome-wise significance level, while a total of 8 and 22 significant QTL were detected at a 5 % and 1 % genomic-wise significance level, respectively. In F<sub>2</sub> population of a broiler-Leghorn cross and a broiler-Fayoumi cross, **Zhou et al. (2006)** found that a total

of 52 and 38 QTL were detected at the 5% chromosome-wise level for the traits evaluated in the broiler-Leghorn cross and the broiler-Fayoumi cross, respectively. Of the 52 suggestive QTL in the broiler-Leghorn cross, 17 QTL were significant at the 5% genome-wise level, while of the 38 suggestive QTL in the broiler-Fayoumi cross, 10 QTL were significant at the 5% genome-wise level. A total of 18 and 13 significant QTL were detected at a 1% chromosome-wise significance level for the 8 growth traits studied, of which 17 and 10 were significant at the 5% genome-wise level, respectively.

**Table 10.** Number of significant QTL at the 5 and 1% chromosome-wise levels and genome-wise level for each trait in F<sub>2</sub> cross.

Trait	Chromosome-wise level		Genome-wise level		Variance (%) <sup>+</sup>
	5%	1%	5%	1%	
BW4	4	-	2	2	21.1
BW8	4	-	2	4	30.8
BW12	2	-	3	3	31.7
BW16	5	-	-	3	25.4
DG04	1	-	1	3	25.9
DG48	2	-	-	3	29.1
DG812	-	-	-	3	9.35
DG1216	-	-	-	1	3.9
Total	18	-	8	22	-

<sup>+</sup> The sum of the total variances explained by each QTL.

Potential candidate genes within the QTL region for growth traits at 1% chromosome-wise significance level were of considerable importance. In F<sub>2</sub> population of broiler sire with Bair layer dams (Chinese local breed) cross, **Wang et al. (2012)** cited that three QTL at 5 % chromosome-wise and 10 QTL at suggestive level on chromosome 3; on chromosome 5, there were four QTL identified at 5% genome-wise level, eight QTL at 5% chromosome-wise level and one at suggestive level. On chromosome 7, there were five QTL identified at 5%

genome-wise level, four QTL at the 5% chromosome-wise level and four QTL at suggestive level.

### Conclusions

- 1) Significant QTL for body weight detected on chromosomes 1, 2, 3, 4, 6, 8, 11 and Z concluded that there are different sets of genes affecting early and late body weights.
- 2) The present genome wide QTL mapping in F<sub>2</sub> populations lays the foundation for identifying

the DNA variants causally responsible for variation in growth traits in chickens. To utilize these results for further identifying causative functional genes or using marker assisted selection (MAS) in poultry improvement program, the detection of fine-mapping QTL is required or the segregation of QTL within commercial population is being to be verified before further efforts are made.

- 3) A single-QTL model was used to detect QTL for growth traits in chickens. Different QTL locations in the same chromosome were observed on several chromosomes. Further analysis with multi-trait QTL model might confirm these multiple QTL. Further studies with this approach might be able to obtain more understanding of the complex genetic architecture underlying quantitative trait variation for growth in chickens.
- 4) It is not very easy at this moment to look for candidate genes in the regions with QTL. The most important reason is that the QTL regions are still too large. The confidence intervals for all of the significant QTL have to be reduced by fine mapping with larger numbers of DNA markers.

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