Quantitative trait loci affecting fatness in the chicken

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Summary

An F_2 chicken population of 442 individuals from 30 families, obtained by crossing a broiler line with a layer line, was used for detecting and mapping Quantitative Trait Loci (QTL) affecting abdominal fat weight, skin fat weight and fat distribution. Within-family regression analyses using 102 microsatellite markers in 27 linkage groups were carried out with genome-wide significance thresholds. The QTL for abdominal fat weight were found on chromosomes 3, 7, 15 and 28; abdominal fat weight adjusted for carcass weight on chromosomes 1, 5, 7 and 28; skin and subcutaneous fat on chromosomes 3, 7 and 13; skin fat weight adjusted for carcass weight on chromosomes 3 and 28; and skin fat weight adjusted for abdominal fat weight on chromosomes 5, 7 and 15. Interactions of the QTL with sex or family were unimportant and, for each trait, there was no evidence for imprinting or of multiple QTL on any chromosome. Significant dominance effects were obtained for all but one of the significant locations for QTL affecting the weight of abdominal fat, none for skin fat and one of the three QTL affecting fat distribution. The magnitude of each QTL ranged from 3.0 to 5.2% of the residual phenotypic variation or 0.2–0.8 phenotypic standard deviations. The largest additive QTL (on chromosome 7) accounted for more than 20% of the mean weight of abdominal fat. Significant positive and negative QTL were identified from both lines.

Keywords broiler, chicken, fat, fat distribution, layer, quantitative trait loci.

Introduction

The identification and use of quantitative trait loci (QTL) in selection programmes offer the potential for more rapid improvement, particularly in difficult-to-measure traits. To achieve this potential, there is a need to identify loci having large effects on these traits and the origin of potentially beneficial alleles. With this information, operators of commercial breeding programmes may consider the introgression of positive alleles into their commercial lines, or select for increasing frequency of desirable alleles by within-line marker assisted selection (MAS). Further benefits may be obtained by precise identification of the loci and the genes involved.

The search for QTL requires the identification of linkage disequilibrium in the experimental population and this has been accomplished in domestic animal species through the development of crosses between breeds, or through searching within full- and half-sib families. Using crosses between breeds or strains, QTL have been mapped for a wide range of traits in pigs (Knott *et al.* 1998; Walling *et al.* 1998), mice (Flint *et al.* 1995; Brockman *et al.* 1998) and chickens (Vallejo *et al.* 1998; van Kaam *et al.* 1998; van Kaam *et al.* 1999a,b). The use of within-family linkage disequilibrium has also been exploited for mapping QTL for milk traits in commercial populations of dairy cattle (Georges *et al.* 1995; de Koning *et al.* 1998; Wiener *et al.* 2000). The benefits of wide crosses include the power to identify QTL within single populations of manageable size

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and the potential to exploit breed variation through introgression and/or crossing programmes, whereas the benefits of using linkage disequilibrium within breeds is the ready applicability to within-breed selection programmes.

Van Kaam *et al.* (1998, 1999a,b) scanned the whole chicken genome for QTL controlling feed intake, growth, body weight and some carcass traits using a population derived from a cross of two broiler dam lines. Their reports identified QTL for feed intake and growth between 23 and 48 days on linkage group 1, a QTL for feed intake adjusted for body weight on linkage group 2, a QTL for carcass percentage on linkage group 1 and a QTL for meat colour on linkage group 2. Evidence for a QTL controlling chicken body weight at 13 weeks was reported on chromosome 1 by Tatsuda *et al.* 2001. The same authors found evidence of a QTL for abdominal fat deposition on chromosome 7 (Tatsuda & Fujinaka 2001). Yonash *et al.* (2001) used a combination of backcross and F_2 progeny to identify DNA microsatellites linked to the QTL affecting antibody response and survival rate in broiler chickens. Their earlier studies (Yonash *et al.* 1999) reported significant linkage of Marek's disease associated traits to chromosome 1 and other linkage groups in chickens.

Fat deposition in the chicken 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 programmes would therefore prove to be of great value. Abdominal fat weight in broilers has been reported to have a heritability ranging between 0.50 and 0.80 (Chambers 1990; Bihan-Duval *et al.* 1999) indicating that there is a strong genetic basis for the deposition of abdominal fat in broiler chickens. Selection against fatness in broiler chickens on the basis of abdominal fat weight or circulating lipoprotein has been successful in producing fat and lean lines of chickens (Delpech & Ricard 1965; Leclercq 1988; Whitehead 1990). A cross between a broiler sire-line and a layer (White Leghorn) strain that differs substantially for many growth and reproduction traits was used in this experiment which was part of a larger study to map QTL for many biological and economically important traits. The bodies of broilers are both heavier and fatter than layers at the same age and contain as much as three times the weight of abdominal fat in the carcass (Morris & Njuru 1990). In two unpublished experiments, the sex averaged mean of the White Leghorn line used in this cross had 5–6 g/kg abdominal fat at 42 days of age compared with 14–16 g/kg in the broiler line, results that are consistent with Morris's data for commercial hybrids. In contrast to abdominal fat, the sum of the weight of skin from the breast and thigh for the two lines, respectively, were 16 and 23 g/kg at 35 days of age (one experiment).

It is generally assumed that selection against abdominal fat based on sib analysis will also decrease fat in other parts of the carcass. The other major depots are the mesenteric fat, fat surrounding the internal organs and subcutaneous fat, of which the latter is the most important commercially because of its effect on carcass quality. This paper reports on the existence of QTL for abdominal fatness, subcutaneous fat and the distribution of fat between these two depots in a broiler–layer cross using within-family marker regression methods for QTL detection (Haley & Knott 1992; Haley *et al.* 1994).

Materials and methods

Animals and husbandry

The origin, mating structure and husbandry of the birds was presented in an earlier paper (Sewalem *et al.* 2002) and will only be summarized briefly here. A line of White Leghorn egg laying (L) chickens was chosen as one of the foundation lines and the other was a commercial broiler (B) sire-line that had been genetically selected for high growth rates and breast muscle yields. Two males from both lines were each mated to a female from the other line to create four F_1 families. At 30 weeks of age, eight male and 32 female F_1 were selected to produce the F_2 generation. Each male was mated to two females of the same cross from the alternative family and to one female from each family of the opposite cross (e.g. B1♂ × L1♀ males were crossed with two B2♂ × l2♀ females and one each of L1♂ × B1♀ and L2♂ × B2♀) and there were two matings of each type. The females were inseminated weekly and eggs were collected for 7 days before being set. The eggs were incubated and hatched in standard machines.

A total of 546 F_2 chicks in 32 full-sib families from five hatches were obtained for broiler trait measurements. The chicks from each hatch were randomly allocated to one of four floor pens littered with wood shavings. The pens contained a suspended drinker and two tubular feeders and food and water were available *ad libitum*. The wheat and soya bean ration was formulated to contain 280 g crude protein/kg, 13 MJ metabolizable energy/kg, 14 g/kg Ca and 7 g/kg P. A hanging brooding lamp provided local heat for the entire experiment and an ambient temperature of 15–16°C was maintained by controlled ventilation and heating of the poultry house. A photoperiod of 23 h light and 1 h darkness was maintained throughout the experiment.

Observations

The birds were weighed at 3 and 6 weeks of age and at slaughter at 2 kg live weight when they were 9 weeks of age ($n = 510$). The feeders were withdrawn 2 h before the birds

were crated for removal to the processing area. The birds were killed by dislocating their necks. The blood vessels of the neck were cut and the body was suspended by the feet to bleed out. The birds were immersed in a tank of hot water (58°C) for 15 s and immediately plucked in an automatic wet defeathering machine. The neck skin was loosened and the head and neck were removed at the anterior edge of the breast and the feet and shanks were cut off at the hock joint. The crop and respiratory tract were removed and the carcasses were eviscerated by hand. The weight of abdominal fat and fat surrounding the gizzard and proventriculus was recorded. The eviscerated carcasses weight was noted and the carcasses were stored at −20°C. The carcasses were subsequently thawed overnight and dissected according to established guidelines (Jensen 1983). The weights of the breast meat, breast skin, legs, thighs, wings and residual carcass were recorded. The legs and thighs from both sides were dissected into bone, muscle and skin including adhering fat. The weights of skin plus fat from the breast, leg and thigh were summed to form a single variable (skin fat).

Genotyping

Samples of fresh chicken blood were collected at 6 weeks of age by superficial venipuncture of a wing vein and DNA was prepared by standard procedures. A total of 103 microsatellite markers covering 26 autosomal linkage groups and the sex chromosomes were used to genotype the eight F_0 grandparents, 40 F_1 parents and 510 F_2 offspring as described previously (Sewalem *et al.* 2002). Fragment sizes were determined by using GeneScan 3.1 DNA fragment analysis and Genotyper 2.1 software (PE Biosystems, Foster City, USA). All pedigree, marker genotypes and trait data were recorded in resSpecies, a generic resource database (http://www.res Species.org, Law & Archibald 2000). Information on the genetic markers can be viewed at http://www.thearkdb.org/browser?species = chicken (Hu *et al.* 2001). After parentage checking and genotyping edits, data from 442 F_2 individuals from 30 families with genotypes on 101 microsatellite markers in 27 linkage groups were available for analysis (Table 1). The total map length, including an arbitrary 20 cM for the end markers and for each linkage group with a single marker was 2923 cM or about 75% of the consensus linkage map (Schmid *et al.* 2000). The average marker interval was 40 cM and the average polymorphic information content was 0.61 (range 0.19–0.98).

QTL analyses

The QTL mapping method proposed by Haley *et al.* (1994) was implemented using QTL Express software (Seaton *et al.* 2001). The probability of an F_2 offspring being each of the four QTL genotypes (QQ, Qq, qQ, and qq) at each position in the genome at 2 cM intervals was calculated conditionally upon the marker genotype. A linear model for the additive (a) and dominance effects (d) of a QTL at a given position was analysed by least squares 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. The statistical model included family, sex and pen as fixed effects because hatch was confounded with pen. Abdominal fat was also analysed by a model that included carcass weight as a covariate (abdominal fatness). The total weight of skin fat was analysed with carcass weight as a covariate (skin fatness) and with abdominal fat weight as a covariate (fat distribution) in a model that included the fixed effects. The informativeness of the markers was assessed at each location as described by Knott *et al.* (1998).

If the test statistics in the initial analysis exceeded the threshold value, we conducted a series of analyses based on

Table 1 Number of microsatellite markers, chromosome (linkage) group, map length and the first marker on each chromosome that were used for a whole genome scan of a broiler layer cross.

Chromosome	Number of markers used	Map length, cM	First marker
1	24	542	*MCW0168*
2	12	474	*LEI0163*
3	11	282	*ADL0131*
4	4	232	*ADL0317*
5	6	167	*LEI0082*
6	4	89	*ROS0062*
7	3	109	*LEI0064*
8	2	94	*ADL0179*
9	4	132	*ROS0078*
10	1	–	*ADL0209*
11	5	70	*MCW0097*
12	2	33	*ADL0240*
13	3	70	*MCW0340*
14	1	–	*MCW0123*
15	2	45	*LEI0083*
17	1	–	*ADL0199*
18	2	23	*ROS0022*
23	2	1	*LEI0090*
24	1	–	*ROS0113*
E25C31	1	–	*ROS0102*
26	1	–	*ADL0285*
27	1	–	*ROS0071*
28	2	40	*ROS0095*
E32	1	–	*ALVE3*
E38	1	–	*ROS0073*
W25	1	–	*MCW0249*
Z	3	127	*ROS0072*

conventional *F*-tests and appropriate degrees of freedom. A QTL by sex interaction was assessed to investigate whether the effect differed between the two sexes. In order to look for evidence that a QTL was segregating in one or the other line, we also included an analysis of the interaction between the QTL effect and family. A model fitting an imprinting effect (paternal origin of the allele) was evaluated as described by Knott *et al.* (1998). A trait showing evidence for a single QTL was tested for the presence of two or more QTL. The two-QTL model fits two QTL by fixing one of the QTL and searching at 2 cM intervals along the chromosomes before moving the fixed QTL to the next location (also spaced at 2 cM). This model was tested by an *F*-ratio against a model with no QTL and against a model with only one QTL. For each suggestive linkage group found in the initial interval mapping analysis we accounted for background genetic effects of the significant QTL in the other linkage groups by backward elimination and substitution of the putative QTL in the other linkage groups (Jansen 1993; Zeng 1993).

Significance thresholds and confidence intervals

Genome-wide significant linkage thresholds were calculated as described (Sewalem *et al.* 2002). Genome-wide thresholds for significance as defined by Lander & Kruglyak (1995) were $F = 8.2$ for the 5% level of probability, $F = 10.0$ for the 1% level and $F = 5.0$ for suggestive linkage. An approximate confidence interval for the localization of each of the significant and suggestive QTL for fat traits was obtained using the bootstrap technique (Knott *et al.* 1998; Visscher *et al.* 1996) with a total of 500 samples. The 95% intervals presented were of minimum length after removal of background bias associated with marker locations (Walling *et al.* 2002).

Results

Phenotypic means and variation

The overall means and standard deviations (in parentheses) of the traits at a live body weight of 2 kg were 51 (16) g for abdominal fat weight, 94 (19) g for skin fat weight and 1349 (235) g for carcass weight. Abdominal and skin fat, respectively, as a proportion of carcass weight were 0.038 (0.011) and 0.070 (0.010). Males were heavier than females and contained a similar weight of abdominal fat so that abdominal fatness was lower in males compared with females (respectively 0.034 vs. 0.042, SE 0.001). The weight of skin fat was greater in males than females but was similar when expressed as a proportion of carcass weight (0.068 vs. 0.072, SE 0.001). Carcass weight was poorly associated with abdominal and skin fatness ($r = -0.18$ and -0.17) whereas abdominal and skin fat were positively correlated (0.74), as were abdominal and skin fatness (0.39).

Evidence for QTL and their effects

Strong evidence for QTL affecting abdominal fat deposition were found on chromosomes 3, 5, 7, 15 and 28 (Table 2). Adjusting abdominal fat weight for the weight of the carcass led to the detection of a significant linkage on chromosome 1 with the loss of the suggestive QTL on chromosome 13, and the addition of suggestive linkages on chromosomes 9 and 15. The microsatellite markers flanking the QTL and the QTL positions within the flanking region are also shown in Table 2. The additive and dominance effects of the significant QTL are presented in Table 3 with the proportion of phenotypic variance explained by each one. The QTL effects ranged from 3.0 to 5.2% of the phenotypic variation. Evidence for significant ($P < 0.05$) QTL for skin fat were found on chromosomes 3, 7 and 13 and chromosomes 3 and 28 for skin fatness (Table 2). The effects of the QTL ranged from 3.4 to 4.2% of the phenotypic variation (Table 3). Fat distribution (abdominal fat weight adjusted for skin fat weight) showed a strong genome-wide significant ($P < 0.01$) linkage (Table 2) on chromosomes 5, 7 and 15 that explained 3.6–4.4% of the phenotypic variation (Table 3).

Interactions, imprinting and multiple QTL

Interactions of the QTL with sex or family were not statistically significant. There was also no evidence of imprinting and multiple QTL affecting the fat traits on any of the linkage groups. Fitting background effects did not result in any suggestive QTL becoming significant.

Discussion

Quantitative trait loci for fat traits

Significant QTL for fat traits were detected on chromosomes 1, 3, 5, 7, 13, 15 and 28 and suggestive linkages on chromosomes 2, 4, 6, 9 and Z (Table 2). Evidence for two locations, respectively, affecting abdominal fat and skin fat on the two ends of chromosome 3 were obtained. Adding the covariate for carcass weight exposed a further QTL on chromosome 1 and led to the loss of significance of two QTL for abdominal fatness on chromosomes 3 and 15. For skin fat, the covariance analysis resulted in the loss of a QTL on chromosome 7 and the loss of formal significance for a QTL on chromosome 13. The weight of skin fat is closely related to the surface area of the carcass and the correlation

Table 2 QTL for the weight of abdominal fat, abdominal fat weight adjusted for body weight (abdominal fatness), the weight of skin and fat and the weight of skin and fat adjusted for body weight (skin fatness), and the weight of abdominal fat adjusted for the weight of skin and fat in an F_2 population of chickens derived from a broiler × layer cross.

Chromosome	F	Position, cM[1]	Flanking markers (FM)	Position from first FM[2]	95% Confidence interval
Abdominal fat weight, g					
3	8.16*	40	ADL0177-MCW0083	9	0–91
5	11.49**	51	ROS0013-ADL0292	4	12–57
7	13.29**	41	LEI0064-ROS0019	41	14–63
13	5.83†	17	*ADL0147-ADL0225*	17	109–182
15	8.13*	0	LEI0083-MCW0080	0	0–26
28	8.52*	17	ROS0095-ADL0299	17	4–39
Z	5.97†	127	*LEI0111-LEI0075*	40	56–127
Abdominal fatness, g					
1	8.14*	126	ADL0188-LEI0068	17	100–182
3	5.80†	50	*ADL0177-MCW0083*	41	0–63
5	10.89**	50	ROS0013-ADL0292	3	10–56
7	11.50**	39	LEI0064-ROS0019	39	0–50
9	5.03†	127	*MCW0135-ROS0030*	67	106–132
15	5.67†	0	*LEI0083-MCW0080*	0	0–29
28	9.25*	17	ROS0095-ADL0299	17	3–32
Z	6.93†	127	*LEI0111-LEI0075*	40	86–127
Skin fat weight, g					
2	6.24†	238	*ADL0196-LEI0124*	13	106–343
3	9.05*	170	MCW0187-ADL0306	16	140–182
4	7.26†	148	*ADL0266-LEI0073*	22	92–171
5	5.85†	51	*ROS0013-ADL0292*	4	24–54
6	6.28†	45	*ROS0003-ADL0142*	12	0–74
7	8.36*	78	LEI0064-ROS0019	78	32–108
13	8.51*	35	ADL0147-ADL0225	35	9–38
28	6.98†	0	*ROS0095-ADL0299*	0	0–23
Skin fatness, g					
1	7.08†	454	*ADL0183-ROS0025*	49	333–487
2	5.66†	230	*ADL0196-LEI0127*	5	201–344
3	9.35*	166	MCW0187-ADL0306	12	129–184
5	6.18†	45	*SNP047I-ROS0013*	20	0–54
13	6.15†	29	*ADL0147-ADL0225*	29	7–38
28	8.36*	0	ROS0095-ADL0299	0	0–21
Fat distribution, g					
2	5.12†	399	*MCW0056-MCW0157*	39	273–438
5	8.58*	51	ROS0013-ADL0292	4	0–54
7	11.08**	36	LEI0064-ROS0019	36	0–59
15	9.37*	0	LEI0083-MCW0080	0	0–36

*significant linkage at 5%; **significant linkage at 1%; †Suggestive linkage.
[1]Position of QTL relative to the first marker in the set for this chromosome (Table 1).
[2]Position of QTL relative to the first flanking marker, i.e. independent of the marker set.

between carcass weight and skin fat was moderately high. A QTL analysis of body weight at 9 weeks in these same animals (Sewalem *et al.* 2002) located QTL on chromosome 3 with confidence intervals that overlap with those for QTL in this study for abdominal fat weight and skin fat weight; and on chromosome 13 for skin fat weight. There was no correspondence of statistically significant QTL for body weight and fat weight adjusted for carcass weight, suggesting that the QTL we have identified for relative fatness are related to lipid deposition rather than growth *per se*.

The overall mean weight of skin including adhering fat was greater than that for the weight of abdominal fat. Skin fat weight in broiler chickens has not received as much interest as abdominal fat weight as the latter is more easily measured and has been used experimentally and commercially as a selection criterion to decrease carcass fatness. The

Table 3 Mean and standard errors (SE) of additive and dominance effects, effects as a proportion of the phenotypic standard deviation (SD) and the proportion of the residual sum of squares that were removed by fitting the model of QTL. Traits analysed were the weight of abdominal fat, abdominal fat weight adjusted for body weight (abdominal fatness), the weight of skin and fat and the weight of skin and fat adjusted for body weight (skin fatness), and the weight of abdominal fat adjusted for the weight of skin and fat.

	Additive effect			Dominance effect			Phenotypic variance percentage
Chromosome	Mean	SE	SD[1]	Mean	SE	SD[1]	
Abdominal fat weight, g							
3	2.6	1.31	0.18	7.5	2.17	0.51	3.26
5	4.8	1.10	0.33	3.5	1.62	0.23	4.53
7	12.0	2.39	0.78	–5.7	7.56	–0.37	5.24
15	3.5	1.23	0.23	4.5	1.73	0.28	3.04
28	–4.2	1.38	–0.29	6.9	2.42	0.49	3.50
Abdominal fatness, g							
1	–3.1	0.89	–0.23	2.6	1.38	0.20	3.00
5	4.4	1.04	0.32	3.7	1.55	0.27	4.25
7	10.1	2.15	0.72	–4.1	6.67	–0.29	4.51
28	–4.3	1.22	–0.34	5.4	2.15	0.43	3.84
Skin fat weight, g							
3	6.2	1.52	0.44	4.18	3.35	0.30	4.05
7	7.0	1.73	0.45	2.17	3.92	0.14	3.41
13	5.2	1.27	0.35	–2.16	2.04	–0.14	3.59
Skin fatness, g							
3	5.1	1.21	0.43	2.70	2.48	0.23	4.18
28	–3.3	0.94	–0.28	2.72	1.25	0.23	3.64
Fat distribution, g							
5	3.9	1.01	0.30	2.90	1.46	0.22	3.64
7	9.1	2.07	0.66	–9.56	6.23	–0.70	4.40
15	2.7	1.08	0.21	4.93	1.52	0.37	3.75

[1]Additive effect divided by the residual standard deviation.

two fat traits are weakly positively associated and there are different QTL and linkage groups associated with them (Table 2), suggesting that specific attention needs to be given to each of them either singly or in combination to enhance the carcass quality of broiler carcasses. Positive effects of three QTL for fat distribution at the same locations indicate that genes exist that affect the proportion of subcutaneous relative to abdominal fat. These three QTL are at similar locations to three of the five QTL for abdominal fat and we conclude that selection against abdominal fat may not decrease skin fat in proportion to the reduction in abdominal fat.

In contrast to the results for body weight (Sewalem *et al.* 2002), we found both positive and negative additive effects originating from the broiler line. In the present study, dominance effects were significant for six of the nine QTL for abdominal fat weight and abdominal fatness and one of three QTL for fat distribution compared with none for skin fat and skin fatness (Table 3). This compares with body weight where we found evidence for a single QTL for body weight at 3 weeks with a significant dominance effect and none at 6 and 9 weeks of age (Sewalem *et al.* 2002).

The magnitude of the additive effects as a proportion of the phenotypic variation was comparable with those for body weight (Sewalem *et al.* 2002) generally accounting for 0.2–0.8 phenotypic standard deviations or 3–5% of the phenotypic variation. The largest QTL on chromosome 7 acted additively and had a large positive effect on all five traits. This single QTL increased abdominal fat by 12 g (24% of the mean) whereas the magnitude of the additive effects of the remaining QTL was generally one quarter to one half of this. It is not easy to estimate the weight of abdominal fat in the parent lines at an immature body weight of 2 kg because this is similar to the mature body weight of male White Leghorns, but it is possible that this single QTL (i.e. twice the additive effect) explains most of the difference between the lines in the weight of abdominal fat.

The lack of a sex by QTL interaction is interesting as it suggests that QTL for fat act additively, although the females are fatter than the males. However, the difference in fatness in this experiment was not large and the experiment may have been too small to detect interactions of this nature. Analysis of marker-QTL data for fatness at maturity may help to further elucidate the nature of gene action for fatness in the chicken.

The various QTL identified in this study exert strong effects on fat deposition in the chicken and are good candidates for the reduction of fatness in commercial birds by MAS in breeding flocks. Initial studies to determine that segregation occurs at these QTL in commercial flocks are a first step to realizing this goal. Furthermore, fat deposition in humans has strong associations with such conditions as diabetes, hypertension and atherosclerosis and it is therefore expected that the identification of the genes in the QTL in chickens that affect fat deposition may contribute to the efforts to combat these conditions in the human population.

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