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DETECTION OF QTL FOR PERFORMANCE, FATNESS AND CARCASS TRAITS ON CHICKEN CHROMOSOMES 3 AND 5

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INTRODUCTION
In previous studies we have identified QTLs affecting performance, carcass and fatness traits, and organ weights in chromosomes 1 (Nones et al., 2006), 6 to 8, 11 and 13 (Moura et al., 2006) in a Brazilian F2 chicken resource population. In this report, we focused on chromosomes 3 and 5 for which QTLs for growth related traits have been already mapped in other populations (reviewed by Abasht et al., 2006a). Thus, the objective of this study was to describe QTL for performance, carcass, fatness and organ weights in the Brazilian population.

MATERIAL AND METHODS
Experimental population and data recording
An F2 chicken resource population specially designed for QTL mapping studies was originated from the crossbreeding of seven males from a broiler line and seven females from a layer line at Embrapa Suínos e Aves, Concórdia, Brazil. From a total of 2,063 F2 chickens incubated over a period of 8 months, 544 belonging to six full-sib families were used in this study. F2 chickens were reared as broilers up to 42 d of age. They were individually caged from 35 to 41 d, when weight gain and feed intake were recorded allowing the computation of feed conversion. Body weight was recorded at 1, 35, 41, and 42 d. At the latter age, recording was performed after 6 h fasting and transportation to the slaughterhouse. Carcasses were eviscerated, stored at –4°C for six hours and dissected. Weights of heart, lungs, gizzard, liver, head and feet, as well as the length of intestine were recorded before chilling. Weights of carcass, breast, drums and thighs, wings, residual carcass and abdominal fat were recorded after chilling. Abdominal fat, breast and carcass percentage was computed relative to body weight at 42 d. Blood samples were collected at slaughter for DNA analyses.

Genotyping
Thirteen microsatellite markers covering 84.1% of the consensus map of chromosome 3, and 7 markers covering 75.5% of chromosome 5 (www.thearkdb.org) were used to genotype 12 parental (6 males, 6 females), 9 F1 (3 males, 6 females), and 544 F2 chickens from 4 to 6 informative full-sib families. The first and last markers were LEI0043 and LEI0166 on chromosome 3 and LEI0082 and ADL0298 on chromosome 5. Individual PCR reactions using fluorescent primers were conducted for each marker. PCR products from three to four markers were mixed for allele size determinations in a MegaBACE genotyper (GE Healthcare). Linkage maps were constructed for each chromosome using multipoint linkage analysis (Green et al., 1990).

QTL mapping analyses
Phenotypic data were submitted to a preliminary analysis of variance including effects of hatch, sex, family and their two-way interactions. Adjustments for hatch and significant interactions were then performed and the residuals used in the QTL interval mapping analyses using the regression method (Haley et al., 1994) and the line cross genetic model of the QTL Express software (Seaton et al., 2002). Sex and family effects were included in the model for QTL mapping. Body weight at 35 d was used as covariate in the model for weight gain, feed intake and feed efficiency from 35 to 41 d, whereas body weight at 42 d was used for carcass weight, carcass parts and organ weights. Significance thresholds were computed using a permutation test (Churchill and Doerge, 1994) and probability levels for significant (1 and 5%) and suggestive genome-wise linkage were used (Lander and Kruglyak, 1995). If the test statistics for a QTL exceeded the suggestive threshold.
level, a model including a parent of origin effect (Knott et al., 1998) as well as models including QTL x sex and QTL x family interactions, were tested based on conventional F-tests.

RESULTS AND DISCUSSION

A total of nine QTL surpassed the genome-wide suggestive threshold (Table 1). No interaction with sex were found for any of them (P > 0.05). Four QTL mapped to chromosome 3 had the greatest effects, exceeding the 1% genome-wide threshold. All four showed significant (P < 0.05) QTL x family interactions, suggesting that the QTL alleles for those traits were not fixed in the parental lines. The first two, for closely related traits (i.e. body weight at 35 and 41 d), were likely the same QTL. It acted predominantly in an additive fashion and explained over 4.5% of the phenotypic variance of body weight at the earlier age (Table 2). These results are in agreement with those of two other F2 populations: one derived from a cross of two lines divergently selected for body weight at 56 d (Jacobsson et al., 2005), who found a suggestive QTL for body weight at 28 d in the interval flanked by marker MCW0222 and another from a completely different F2 population derived from a red junglefowl x White Leghorn cross (Kerje et al., 2003), who mapped a suggestive QTL for body weight at 46 d to an interval flanked by ADL0161.

The third QTL, for abdominal fat percentage (Table 1), explained almost 4% of the phenotypic variance. The QTL allele that conferred higher abdominal fat percentage originated from the broiler line (Table 2). Interestingly, a significant (P < 0.01) parent of origin effect was detected for this QTL. The effect was positive, indicating that the broiler allele coming from the male parent increased the trait value. Genomic imprinting may be an explanation for parent of origin effects. De Koning et al. (2002) recommended caution to avoid spurious detection of parent of origin effects when the QTL is segregating in the parental lines, especially for designs in which the number of F1 sires is reduced. There were only three F1 sires involved in the present study and there was evidence of QTL allele segregation in the founder lines, therefore the parent of origin effect detected in this study may not be true. No other fatness QTL with parent of origin effects was reported in chicken (reviewed by Abasht et al., 2006a), but McElroy et al. (2006) found a paternally expressed QTL for white meat percentage, whereas Park et al. (2006) detected a Mendelian QTL for abdominal fat weight at 70 d, both close to position of the QTL reported in this study. Other QTL with parent of origin effects were previously reported for body and carcass weights (McElroy et al., 2006), egg weight (Tuiskula-Haavisto et al., 2004), and disease resistance (Siwek et al., 2003) in other regions of chromosome 3.

The last 1% genome-wise significant QTL for wing’s weight was mapped to the intermediate region of chromosome 3 (Table 1). This QTL showed negative additive effects, indicating that the allele for higher weight, in this case, was coming from the layer line. A suggestive QTL for lung weight (Park et al., 2006) and a significant QTL for skin fatness (Ikeobi et al., 2002) were reported in this region.

Five suggestive QTL were identified: for liver weight on chromosome 3, and for heart and gizzard weights and abdominal fat and carcass percentages on chromosome 5 (Table 1). The abdominal fat percentage QTL, which acted both in an additive and dominant fashion (Table 2), was detected after fitting in a cofactor to account for the background effect of the QTL for the same trait on chromosome 3. Several studies identified QTL related to fatness on chromosome 5, some of them in positions that were close to the QTL mapped in this study (Lagarrigue et al., 2006; Abasht et al., 2006b). The heart and gizzard QTL acted mainly in a dominant fashion, whereas the liver and carcass percentage QTL showed positive additive effects and positive or negative dominance effects.

Identifying chromosome regions associated with fat deposition through QTL mapping studies may lead to the identification of the actual genes controlling the trait, contributing to enhance selection for lean meat yield. Therefore, the two QTL mapped for abdominal fat percentage in this study should be further investigated. Together they explained over 6% of the phenotypic variance of the trait and were mapped to chromosome regions where other independent studies have already detected fatness QTL. Moreover, the abdominal fat percentage QTL on chromosome 3 was located close to a QTL for market age weight, a trait under intense selection in broiler lines. This finding may help to explain the correlated response in fatness to selection for growth rate in broiler lines. As pointed out by Abasht et al. (2006a), due to the large confidence intervals of QTL, higher resolution analysis will be necessary to distinguish a pleiotropic QTL from a closely linked QTL.
Other QTL mapped in this study (e.g. for body and heart weights and for carcass percentage) point out to candidate regions for genes affecting traits of great economic relevance to the poultry industry.

Table 1. QTL that exceeded suggestive linkage

<table>
<thead>
<tr>
<th>Chromosome</th>
<th>Trait</th>
<th>Position (cM)</th>
<th>Flanking markers</th>
<th>F</th>
</tr>
</thead>
<tbody>
<tr>
<td>3</td>
<td>Body weight at 35 d</td>
<td>102</td>
<td>MCW0222 - LEI0161</td>
<td>14.02**</td>
</tr>
<tr>
<td></td>
<td>Body weight at 41 d</td>
<td>102</td>
<td>MCW0222 - LEI0161</td>
<td>11.42**</td>
</tr>
<tr>
<td></td>
<td>Abdominal fat percentage</td>
<td>112</td>
<td>LEI0029 – ADL0371</td>
<td>8.16**</td>
</tr>
<tr>
<td></td>
<td>Wings weight</td>
<td>157</td>
<td>ADL0371 – LEI0118</td>
<td>12.87**</td>
</tr>
<tr>
<td></td>
<td>Liver weight</td>
<td>176</td>
<td>ADL0127 – MCW0224</td>
<td>5.37†</td>
</tr>
<tr>
<td>5</td>
<td>Heart weight</td>
<td>25</td>
<td>MCW0193 – MCW0090</td>
<td>6.80†</td>
</tr>
<tr>
<td></td>
<td>Carcass percentage</td>
<td>97</td>
<td>LEI0149 - ADL0233</td>
<td>5.30†</td>
</tr>
<tr>
<td></td>
<td>Abdominal fat percentage</td>
<td>133</td>
<td>ADL0233 – ADL0298</td>
<td>7.16†</td>
</tr>
<tr>
<td></td>
<td>Gizzard weight</td>
<td>150</td>
<td>ADL0233 – ADL0298</td>
<td>5.55†</td>
</tr>
</tbody>
</table>

A Position from the first marker (LEI0043 for chromosome 3 and LEI0082 for chromosome 5) in the chromosome set. LEI0043 is at 9 cM and LEI0082 is at 32 cM in the consensus map of chromosomes 3 and 5, respectively.

† Significance at the genome-wide suggestive level
** Significance at the 1% genome-wide level

Table 2. Additive and dominance effects (standard errors) and the proportion of the phenotypic variance explained by the QTL

<table>
<thead>
<tr>
<th>Chromosome</th>
<th>Trait</th>
<th>Additive effect</th>
<th>Dominance effect</th>
<th>Phenotypic variance (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>3</td>
<td>Body weight at 35 d (g)</td>
<td>40.64 (7.93)</td>
<td>-17.32 (12.66)</td>
<td>4.74</td>
</tr>
<tr>
<td></td>
<td>Body weight at 41 d (g)</td>
<td>47.21 (10.24)</td>
<td>-21.00 (16.35)</td>
<td>3.83</td>
</tr>
<tr>
<td></td>
<td>Abdominal fat percentage (A) (%)</td>
<td>0.140 (0.035)</td>
<td>-0.053 (0.051)</td>
<td>3.95</td>
</tr>
<tr>
<td></td>
<td>Wings weight (g)</td>
<td>-1.32 (0.26)</td>
<td>-0.18 (0.42)</td>
<td>4.35</td>
</tr>
<tr>
<td></td>
<td>Liver weight (g)</td>
<td>0.53 (0.21)</td>
<td>-0.74 (0.36)</td>
<td>1.65</td>
</tr>
<tr>
<td>5</td>
<td>Heart weight (g)</td>
<td>0.024 (0.074)</td>
<td>0.413 (0.112)</td>
<td>2.17</td>
</tr>
<tr>
<td></td>
<td>Carcass percentage (%)</td>
<td>0.328 (0.126)</td>
<td>0.346 (0.180)</td>
<td>1.62</td>
</tr>
<tr>
<td></td>
<td>Abdominal fat percentage (%)</td>
<td>0.153 (0.049)</td>
<td>-0.194 (0.103)</td>
<td>2.32</td>
</tr>
<tr>
<td></td>
<td>Gizzard weight (g)</td>
<td>0.087 (0.229)</td>
<td>1.254 (0.376)</td>
<td>1.71</td>
</tr>
</tbody>
</table>

A This QTL showed significant (P < 0.01) parent of origin effect = 0.100 (0.035)

CONCLUSION AND FUTURE WORK

The QTL mapped for body weight and abdominal fat percentage on chromosome 3 give support to the results of recently published QTL mapping studies, and also provide strong evidence for candidate regions for genes affecting traits of great economic relevance to the poultry industry. A half-sib analysis should be carried out to investigate the QTL that showed interaction with family. Expression studies should be conducted to search for evidence of imprinting at the molecular level.

Our group is concluding the genotyping of over 400 F2 chickens from five full-sib families with markers from the microchromosomes (9, 10, 12, 14, 15, 18, 19, 23, 24, 26 to 28 and Z) in 2007, to complete the genome scan for growth-related and some metabolic parameters-related traits.

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