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Association between Polymorphisms of Lipoprotein Lipase Gene and Chicken Fat Deposition*

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ABSTRACT : The objective of this study was to screen single nucleotide polymorphisms (SNPs) of the chicken lipoprotein lipase gene (*LPL*), using 545 F1 hybrids developed from 4×4 diallel crossing of four chicken breeds, and to analyze the associations between polymorphisms of the *LPL* and chicken fat deposition traits. PCR-SSCP was used to detect SNPs in *LPL*. Fifteen sets of primers were designed to amplify DNA fragments covering the 5′flanking and coding regions of *LPL*. It showed that there existed 5 polymorphic loci in the 5′flanking region and coding region, respectively. Association analysis was carried out between 10 polymorphic loci and intermuscular fat width, abdominal fat weight, and thickness of subcutaneous fat using ANCOVA, respectively. The results indicated that, in the 5′flanking region, the loci *d* and *e* significantly affected thickness of subcutaneous fat ($p<0.05$), abdominal fat weight ($p<0.01$) and subcutaneous fat ($p<0.05$), while in the coding region, synonymous mutation in exon 8 was significantly associated with intermuscular fat width ($p<0.05$), however, the non-synonymous mutations in exon 7 and exon 9 did not show statistically significant effects on fat deposition traits in this study. (**Key Words :** Chicken, SNP, *LPL*, Fat Deposition Traits, PCR-SSCP)

INTRODUCTION

Poultry breeding has been making exciting progress for the past several years, e.g. broiler chicken has been improved in many traits such as daily weight gain, feed efficiency and resistance to disease. Basically, chicken production could meet our current consumption needs. The high selection intensity for growth rate, however, has caused many problems, especially the increasing trend for abdominal fat deposition. Excessive fat deposition affects chicken carcass merit and flavors, furthermore it affects feed efficiency and resistance to diseases.

Researchers and producers have been paying more and more attention to chicken fat deposition. Many studies research concerned with chicken fatness, from either nutrition or genetics point view were reported, but as far as known, genetics will be the perfect choice. Some research has found several candidate genes or markers for chicken fat deposition (Meng et al., 2005; Choi et al., 2006; Li et al., 2006; Li et al., 2006).

A little-known fat-builder enzyme called lipoprotein lipase (LPL) is the gatekeeper to fat cells, and is the most important enzyme that functions in the catabolism of triglyceride from plasma lipoprotein. LPL was shown to have a relationship with adipose tissue fat deposition (Hermier et al., 1989; Hermier et al., 1991). Sato et al. (1999) were successful in reducing chicken body fat by injecting antilipoprotein lipase antibody, indicating that LPL plays an important role in fat deposition. Cooper et al (1992) first reported the full sequence of the chicken *LPL* gene which is 17 kb long and composed of a 1,947 bp 5′ flanking region, 10 exons and 9 introns. Many studies have been performed on the effect of polymorphisms of the *LPL* gene on plasma lipoprotein concentration which were associated with a number of pathophysiological conditions, including atherosclerosis, chylomicronaemia, obesity, Alzheimer's disease and dyslipidaemia in humans (James et al., 2002; Merkel et al., 2002). However, few studies have been reported on polymorphism of the *LPL* gene in livestock. To date there appears to be only one study which

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Table 1. Simple statistics of fat deposition traits in different chicken crosses (Mean±SE, Units: mm, mg, and mm)

Crosses	EA	DA	CA	AA	EC	DC	CC	AC
[1]IMFW	1.23±0.04	0.76±0.03	0.98±0.04	0.73±0.06	1.12±0.06	0.81±0.06	0.85±0.05	0.97±0.05
AFW	0.47±0.01	0.36±0.01	0.37±0.01	0.29±0.01	0.51±0.02	0.39±0.02	0.37±0.02	0.41±0.02
TSF	3.80±0.1	2.81±0.14	2.40±0.16	1.33±0.2	3.93±0.13	2.69±0.14	2.38±0.16	2.35±0.16
Crosses	ED	DD	CD	AD	EE	DE	CE	
IMFW	1.04±0.1	0.69±0.04	0.87±0.04	0.95±0.04	1.17±0.08	0.98±0.06	1.27±0.05	
AFW	0.50±0.02	0.37±0.02	0.41±0.02	0.39±0.02	0.41±0.02	0.43±0.02	0.55±0.02	
TSF	3.59±0.18	2.67±0.17	3.35±0.11	2.63±0.16	3.93±0.16	4.12±0.15	4.39±0.09	

[1] IMFW: Intermuscular fat width; AFW: Abdominal fat weight; TSF: Thickness of subcutaneous fat.

Table 2. Primer sequences and their annealing temperatures for 5´flanking and coding regions in chicken *LPL* gene

Locus	Sequence of primers	Locus	Sequences of primers
a	F[1]: CAC TGT CTG GGT TGG GAA TG	Exon 2	F: GCA TCG TAG ACT GTG ACT TC
59°C [3]	R[2]: GGC TCC GTA ACG CAG TCT GT	57°C	R: GCC ACC CTT TTC TTC TTA C
b	F: TGC GTT ACG GAG CCT TAT TT	Exon3	F: CCA TCA CAA CAT AAC AGG TG
55°C	R: GTG TGT GCT CAC TGT TCT G	58°C	R: CGT GAG ACA TAC ATG ACT AC
c	F: TCA GAA CAG TGA GCA CAC	Exon4	F: TAA ACA AAG CTC TCC TCT GC
57°C	F: TGG GGA TTT GGC GGT CTT	63.2°C	R: GGA CTT CCC TCT ACA ACC AC
d	F: TAA GAC CGC CAA ATC CCC	Exon5	F: TAA AGG TCT GGA TCC TGC TG
55°C	R: CAG TGG TTT CTC AGA GTG	62.4°C	R: TCC CTT CCG ATG TTA TAC TG
e	F: CTG CCC TTG AAG TGA ATG	Exon6	F: GAA GAT GTG GAT CAG CT
57°C	R: TGC AAG TTG CTG GAG GCT	58°C	R: GGA GAA CCT ACC TTT G
f	F: CTC CAG CAA CTT GCA CTC	Exon 7	F: CCA GTC TTC CAT TAT CAG G
54°C	R: CTG CCC CCG CTC TTT G	58°C	R: GGC TAG AGT ATG CAG TG
g	F: CAA AGA GCG GGG GCA G	Exon 8	F: CTC TGA TTC CTT ACA GG
Failure	R: CTC CCT CCG AAA CCT AT	58°C	R: CTC AAG GCA ATC AAA GC
		Exon 9	F: CAG GGT GGT ATT CTG TTC TC
		63.5°C	R: CAC TCT TAC CCT CCC CTC TT

[1] F stands for forward primer; [2] R stands for reverse primer; [3] Annealing temperature.

investigated the polymorphism in intron 6 of swine *LPL* gene and its significant association with carcass length and backfat at the last rib (Lei et al., 2004). There have been no studies on the association between polymorphism of the *LPL* gene and fat deposition in chickens. Genetic markers associated with fat deposition are warranted to assist in selection for meat quality, production efficiency, and health in chickens. The *LPL* was therefore chosen in the current study as a candidate gene affecting body fat deposition based on its biological function in lipid metabolism. The objective of this study was to screen the polymorphisms of 5´ flanking and coding regions of the chicken *LPL* gene in a total of 545 individuals developed from a 4×4 diallel crossing of four chicken breeds including White Leghorn (A), Silkies (C), CAU Brown (D), and White Plymouth Rock (E), and to analyze whether there are associations between polymorphisms of the *LPL* gene and chicken fat deposition.

MATERIALS AND METHODS

Animals

A total of 545 male F1 individuals were developed from diallel crossing involving White Leghorn (A), Silkies (C), CAU Brown (D), and White Plymouth Rock (E), which were reared in cages under the same conditions. Randomly selected healthy cocks from these crosses were slaughtered and measured for three fat deposition traits (intermuscular fat width, abdominal fat weight, and thickness of subcutaneous fat) at 14 weeks of age. Sample sizes of each cross exceeded 40 (except EA which was not available). Table 1 presents the simple statistics of these traits derived from the experimental population. Individual blood samples were collected and stored at -20°C for DNA extraction.

PCR-SSCP analysis

Using the software Oligo6.0, seven pairs of primers were designed, named *a, b, c, d, e, f* and *g*, for the 5´ flanking region and another 8 pairs of primers for all exons, except for exon 1 and exon 10 which code for signal peptide and 3´ un-translated region, respectively。 Genomic sequence of the *LPL* gene reported by Cooper et al. (GenBank Accession No: X60547) was referred to and all primer sequences were shown in Table 2.

The primers were annealed at 55-63°C for 40-45 seconds and then extended at 72°C for 30 seconds. PCR products were separated on native polyacrylamide gels and then detected by silver staining. Polymorphisms were detected for each fragment for 545 individuals according to

Table 3. The contrast between normal and mutation

Loci	Normal	Mutation	Cooper (1992)
a	A(-1908), C(-1864), T(-1810), C(-1668)	G(-1908), C(-1864), T(-1810), T(-1668)	A(-1908), /(-1864), T(-1810), C(-1668)
b	C(-1634)	T(-1634)	C(-1634)
d	T(-1086), A(-1078), G(-982)	T(-1086), G(-1078), /(-982)	C(-1086), A(-1078), G(-982)
e	G(-774), C(-526)	C (-774), T(-526)	C(-774), C(-526)
f	T(-435), C(-396), G(-363), A(-341), T(-297), G(-277),C(-276), A(-273)	T(-435), C(-396), A(-363),G(-341), T(-297), G(-277), C(-276), G(-273)	C(-435), T(-396), A(-363), A(-341), C(-297), C(-277), G(-276), A(-273)
Exon 3	C(6731)	T(6731)	T(6731)
Exon 6	A(10407)	G(10407)	A(10407)
Exon 7	C(11382)	G(11382)	G(11382)
Exon 8	C(12315)	T(12315)	C(12315)
Exon 9	A(13644)	G(13644)	A(13644)

Table 4. Allelic frequencies of polymorphism loci located in 5´flanking and coding regions in chicken *LPL* gene

Crosses	*a*	*b*	*d*	*e*	*f*	Exon 3	Exon 6	Exon 7	Exon 8	Exon 9
EA	0.2	0.13	0.49	0.49	0.39	0.21	0.18	0.13	0.48	0.33
DA	0.46	0.0	0.31	0.50	0.05	0.43	0.0	0.39	0.41	0.41
CA	0.49	0.09	0.38	0.43	0.46	0.46	0.18	0.33	0.50	0.45
AA	0.49	0.0	0.50	0.38	0.50	0.06	0.29	0.06	0.47	0.06
EC	0.34	0.40	0.25	0.34	0.44	0.49	0.01	0.43	0.12	0.06
DC	0.41	0.17	0.44	0.50	0.21	0.24	0.20	0.0	0.37	0.06
CC	0.46	0.10	0.44	0.50	0.43	0.50	0.07	0.06	0.50	0.31
AC	0.4	0.06	0.35	0.47	0.44	0.49	0.0	0.49	0.44	0.49
ED	0.46	0.49	0.38	0.49	0.32	0.46	0.47	0.45	0.18	0.12
DD	0.25	0.01	0.37	0.41	0.41	0.09	0.50	0.0	0.49	0.19
CD	0.18	0.09	0.40	0.46	0.06	0.32	0.35	0.0	0.44	0.08
AD	0.30	0.0	0.49	0.42	0.45	0.36	0.03	0.41	0.50	0.30
EE	0.35	0.02	0.44	0.44	0.42	0.15	0.50	0.06	0.27	0.33
DE	0.22	0.49	0.46	0.49	0.20	0.39	0.28	0.38	0.07	0.31
CE	0.40	0.47	0.46	0.48	0.37	0.41	0.25	0.30	0.31	0

This table showed the minor allelic frequency of each given polymorphic loci.

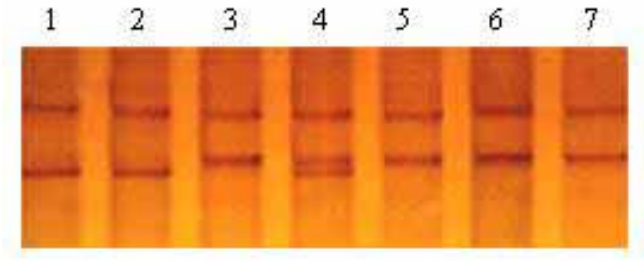

Figure 1. SSCP result of locus *d* in the 5´flanking region in chicken *LPL* gene. Lane 1-2: genotype EE; lane 3, 5-7: genotype FF; lane 4: genotype EF.

the band patterns.

Cloning and sequencing

Once polymorphism was found in each locus, the PCR products from mutated and wild homozygotes were isolated and purified with BioGene Geneclean III Kit (Carlsbad, CA, USA), then ligated into pMD18-T vector and transformed into *E. coli* DH5α. The recombinants were identified by thalline PCR and then sequenced by an ABI 377 DNA sequencer.

Statistics analysis

With the SAS (version 8.02) software, association between SNPs of the *LPL* gene and chicken fat deposition was analyzed using the following single trait model:

$$y = \mu + b \times x + comb + g + e$$

Where y is the phenotypic record of intermuscular fat width, abdominal fat weight, or thickness of subcutaneous fat, μ is overall mean, b is regression coefficient of co-variable *x*, *x* is individual live weight, *comb* is combination effect for the interaction between breed composition and type of reciprocal cross, *g* is the genotypic effect for each locus, and *e* is residual.

RESULTS

Polymorphisms of *LPL* gene

All fragments from 5´ flanking and coding regions were successfully amplified with the exception of the fragment in 5´ flanking region with primer *g* because high content of GC. Polymorphisms were identified in the *a*, *b*, *d*, *e* and *f* sub-regions in the 5´ flanking region and exons 3, 6, 7, 8

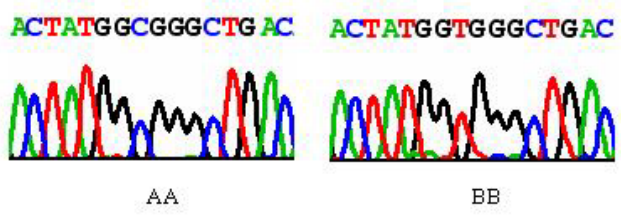

Figure 2. Partial sequences of locus *a* in the 5′flanking region of chicken *LPL* gene. AA showed C at position 1,668; BB showed T at position 1,668.

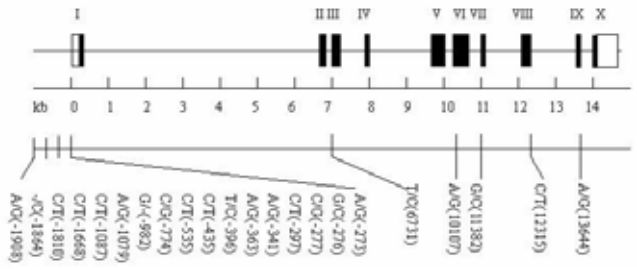

Figure 3. Sketch map of the SNPs in *LPL* detected. Black boxes represent exons, the blank boxes represent the un-translated region and the open boxes represent introns and 5′ flanking region. Positions are given in number of base pairs relative to the transcriptional start of *LPL*.

and 9, and listed in Table 3; allelic frequencies of each polymorphic locus are shown in Table 4. Big variations existed among different crosses for most of the loci. At each polymorphic locus, 3 kinds of genotypes were found. Figure 1, as an example, shows the genotypes of locus *d* in the 5′ flanking region.

By comparing the sequences of each fragment with that described by Cooper et al. (1992) multiple types of base alterations were identified in most of the 5′ flanking sub-regions. In locus *a*, for example, four types of base alteration were found, A transferred into G at -1,908, C inserted at -1,864, C transferred into T at -1,810 and -1,668; however, there was only one type of alteration out of these four existing between individuals: at -1,908 and -1,668, and the type of base alteration of C into T at -1,668 is shown in Figure 2. Another 7 sites of mutation in 5′ flanking sub-regions existed: C-1634T, A-1078G, G-982 delete, C-774G, C-526T, A-341G and A-273G, and 7 sites of base alteration in 5′ flanking sub-regions compared with the reported sequence, namely C-1086T, C-435T, T-396C, A-363G, C-297T, C-277G, and G-276C. One type of base alteration was observed for each exon. The base alteration of G into C at position 11,382 in exon 7 induced an amino acid substitution of Pro with Ala, and the base alteration of A into G in exon 9 led to a substitution of Gly with Asp, all of the other alterations in exon 3, exon 6 and exon 8 were synonymous substitution. Table 3 summarizes the sequences for each SNP, and Figure 3 presents a sketch map of the SNPs in *cLPL* detected in this study.

Association between polymorphisms of *LPL* and fat deposit traits

The association analysis between polymorphic sites within *cLPL* and fat deposition traits is shown in Table 5. In the 5′ flanking region, there were significant association between locus *d* and thickness of subcutaneous fat ($p<0.05$) and abdominal fat weight ($p<0.01$), and between locus *e* and subcutaneous fat ($p<0.05$). In the coding region, exon 8 was significantly associated with intermuscular fat width ($p<0.05$), while no associations between the other exons and fat deposition were found ($p>0.05$).

Least squares analysis was performed for these polymorphism loci (Table 6). At locus *d*, allele F had a negative effect on fat deposition, individuals with genotype FF had significantly lower fat deposition reflected in all three traits than that of individuals with genotype EE. The deviation between genotypes FF and EE was -0.064 mm for intermuscular fat width (IMFW, $p<0.05$), -6.752 g for abdominal fat weight (AFW, $p<0.05$), and -0.022 mm for thickness of subcutaneous fat (TSF, $p<0.05$). The base alteration at exon 8, C converted into T, led to a synonymous substitution. The effect of genotype LL and KK on fat deposition was inconsistent. Genotype LL had a significant effect on increasing TSF, 0.013 mm ($p<0.01$) and 0.029 mm ($p<0.01$), compared to genotype KK and KL, however, genotype LL showed a very significant effect in reducing AFW by -8.733 g ($p<0.01$) compared to genotype KK. This implied that a different set of genes could control fat deposition for IMFW, AFW and TSF and the relationship among the effects of these genes may not be consistent.

Table 5. Association between SNPs within *LPL* and fat deposition traits in chicken

Traits	a	b	d	e	f	Exon 3	Exon 6	Exon 7	Exon 8	Exon 9
IMFW[1]	1.19[2]	0.18	2.44	0.46	0.61	1.60	0.17	0.60	3.45*	0.64
	(0.3064)[3]	(0.8369)	(0.0883)	(0.6305)	(0.5415)	(0.2034)	(0.8467)	(0.5477)	(0.0324)	(0.5283)
AFW	2.08	0.09	3.10*	3.25*	1.37	0.52	0.62	0.74	1.58	0.29
	(0.1256)	(0.9165)	(0.0461)	(0.0397)	(0.2545)	(0.5942)	(0.5380)	(0.4754)	(0.2076)	(0.7496)
TSF	0.00	0.64	6.31**	2.84	0.68	0.23	1.23	1.47	1.01	0.26
	(0.9980)	(0.5297)	(0.002)	(0.0591)	(0.5050)	(0.7968)	(0.2946)	(0.2305)	(0.3633)	(0.7731)

[1] IMFW: Intermuscular fat width, AFW: Abdominal fat weight, TSF: Thickness of subcutaneous fat.
[2] F values. [3] Pvalues. ** Significance level at $p<0.01$. * Significance level at $p<0.05$.

Table 6. Least squares mean (LSM) and standard error (SE) for chicken fat deposition traits of different genotypes at polymorphic sites (Unit: mm, kg, mm)

Locus	Genotype	IMFW[1]		AFW		TSF	
		LSM	SE	LSM	SE	LSM	SE
a	AA	0.922^{a}	0.028	0.414^{A}	0.009	37.812^{A}	2.103
	AB	0.974^{a}	0.025	0.427^{B}	0.008	38.304^{B}	1.827
	BB	0.977^{a}	0.023	0.404^{B}	0.008	35.089^{B}	1.726
b	CC	0.969^{a}	0.021	0.413^{b}	0.007	37.588^{B}	1.547
	CD	0.941^{a}	0.036	0.419^{a}	0.012	35.590^{A}	2.626
	DD	0.931^{a}	0.131	0.407^{ab}	0.423	40.632^{A}	10.529
d	EE	0.999^{a}	0.022	0.429^{a}	0.007	39.262^{a}	1.623
	EF	0.935^{ab}	0.025	0.404^{b}	0.008	38.378^{ab}	1.822
	FF	0.935^{b}	0.025	0.407^{b}	0.008	32.510^{b}	1.865
e	GG	0.977^{a}	0.026	0.430^{a}	0.008	36.865^{b}	1.829
	GH	0.961^{a}	0.023	0.401^{a}	0.007	34.614^{ab}	1.650
	HH	0.943^{a}	0.024	0.417^{a}	0.008	39.816^{a}	1.761
f	II	0.985^{A}	0.027	0.425^{A}	0.009	37.135^{A}	1.962
	IJ	0.957^{B}	0.303	0.403^{B}	0.10	38.591^{B}	2.210
	JJ	0.946^{B}	0.021	0.414^{B}	0.007	36.110^{B}	1.542
Exon 3	AA	0.946^{a}	0.023	0.421^{a}	0.008	38.550^{a}	1.710
	AB	0.949^{a}	0.023	0.411^{a}	0.008	34.712^{a}	1.713
	BB	1.016^{a}	0.034	0.409^{a}	0.011	38.332^{a}	2.530
Exon 6	GG	0.968^{a}	0.022	0.421^{a}	0.007	37.035^{ab}	1.624
	GH	0.951^{a}	0.026	0.407^{a}	0.008	36.820^{a}	1.916
	HH	0.896^{a}	0.183	0.414^{a}	0.059	47.018^{b}	16.427
Exon 7	II	0.977^{cB}	0.030	0.424^{B}	0.010	34.949^{cB}	2.177
	IJ	0.970^{aA}	0.029	0.405^{A}	0.009	34.394^{aA}	2.122
	JJ	0.915^{bB}	0.043	0.414^{B}	0.014	44.536^{bB}	3.153
Exon 8	KK	0.881^{a}	0.039	0.425^{A}	0.013	42.430^{A}	2.845
	KL	0.972^{a}	0.018	0.409^{B}	0.060	35.645^{B}	1.303
	LL	1.079^{a}	0.060	0.438^{B}	0.019	33.597^{B}	4.345
Exon 9	MM	0.969^{B}	0.042	0.422^{B}	0.013	39.009^{B}	3.057
	MN	0.931^{B}	0.029	0.417^{B}	0.009	36.891^{C}	2.143
	NN	0.974^{A}	0.022	0.411^{A}	0.007	36.349^{A}	1.626

Small and capital letters of superscripts means significance at $p<0.05$ and $p<0.01$, respectively.

DISCUSSION

In this study 18 SNPs were detected in the 5′ flanking region of the chicken *LPL* gene. It had been reported previously that removal of the region between -333 bp and -138 bp of the chicken *LPL* gene resulted in a 6-fold increase in gene expression activity (Lu, 1993), which suggested that a negative regulatory element was located in this region. A silence element was located between -263 and -237 by further deletion analysis (Zhang, 1999), and this sequence contained two 10 nucleotides palindromic halves (-263 to -254 and -250 to -241) with a three-nucleotide spacer (-253 to -252), either half being sufficient for full inhibitory function. Among the SNPs detected in our study, the one which was the closest to the silencer, was located at -274 in site *f* of the 5′ flanking region, 10 nucleotides from the palindromic sequence, but the effect of different genotype at site *f* did not reach significance level ($p>0.05$), nevertheless, site *f* (-513 to -253) contained 10 nucleotide which is the palindromic half. We suspect that site *f* may be an important function site and it may function by the silencer, therefore it is worthwhile to extend the length of site *f* in future studies and to further investigate its potential.

Two missense mutations were found in exons 7 and 9, which resulted in a substitution of Pro with Ala, and Gly with Asp, respectively. Ala is the 377th amino acid and Asp is the 447th amino acid coded by the *LPL* gene (Sendak, 1998). To ensure functional activity, the least continuous segment of *LPL* must contain amino acid from 310th to 450th. The missense mutation detected in our study was located in this region, indicating that exons 7 and 9 could be important function sites for *LPL*, and further studies are needed to investigate them.

Association analysis indicated that mutation in exon 8 had a very significant effect on AFW and TSF ($p<0.01$), although this is a synonymous mutation. It is well known that the *cLPL* gene contains 9 introns; given that the pre-mRNAs undergo alternative splicing, it is not surprising that disruption of normal splicing patterns may cause or modify the phenotypic performance of animals. Therefore

this mutation is worth further investigated, such as identification of this mutation in different species or other chicken breeds, in order to confirm how the site is conserved, and then check the mRNA splicing pattern of the *cLPL* gene or investigate the stability of mRNA in different alleles. This research will be carried out later by the authors.

As the main component of gene expression regulation, the 5′ flanking region plays an important role in affecting the phenotypes of traits. Polymorphisms of both site *d* and *e* in the 5′ flanking region had significant effects on TSF. These two loci affected the same trait and their closely adjacent physical location appeared to be one QTL or a closely linked marker that affected TSF.

From a physiological point of view, TSF is related to AFW to some extent, and polymorphic locus *d* in the 5′ flanking region significantly affected both of these traits ($p<0.05$). If locus *d* is considered as a genetic marker in a breeding program for one trait it will bring the correlated genetic progress for the other. Allele L of exon 8, on the other hand, expressed significantly diverse effects of both increasing TSF and decreasing AFW. In conclusion, some important polymorphic loci affecting chicken fat deposition were identified in the present study, which implied that more caution will be necessary for marker assisted selection to decrease fat deposition in chickens in order to fully consider the pleiotropy of gene.

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