



Novel single nucleotide polymorphisms in the 5' regulatory region of the duck *SCD1* gene and their associations with serum biochemical levels and fatty acid composition

W.-G. Li, Y.-Y. Zhang and D.-D. Wang

Key Laboratory of Animal Genetics,
Breeding and Reproduction in the Plateau Mountainous Region,
Ministry of Education, College of Animal Science, Guizhou University,
Guiyang, Guizhou, China

Corresponding author: Y.-Y. Zhang
E-mail: zyy8yyc@163.com

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ABSTRACT. Stearoyl-coenzyme A desaturase 1 (SCD1) is the key limiting enzyme in the synthesis of monounsaturated fatty acids, and plays a crucial role in the regulation of oleic acid. In this study, 165 ten-week-old Cherry Valley ducks were used to investigate single nucleotide polymorphisms (SNPs) in the 5' regulatory region of the *SCD1* gene, and their associations with duck serum biochemical levels and fatty acid composition. Two novel SNPs, g.936516 C > G and g.936551 T > C, were found by polymerase chain reaction-single-strand conformation polymorphism analysis and DNA sequencing methods, exhibiting six genotypes (AA, BB, CC, AB, AC, and BC). The frequency of the dominant genotype BB and allele B was 0.321

and 0.403, respectively. The polymorphism information content value was 0.617, indicating high polymorphism. The chi-square test indicated that the genotype distribution deviated markedly from Hardy-Weinberg equilibrium ($P < 0.01$). The linkage of the two mutant sites in the duck *SCD1* gene had significant effects on the serum albumin, total protein, globulin, triglyceride, total cholesterol, and cholinesterase levels, as well as on 16 kinds of fatty acids except for C14:1 and C20:0 ($P < 0.05$). These results indicated that the C allele might have a positive effect on polyunsaturated fatty acids with potential health benefits. Therefore, the *SCD1*-gene-specific SNPs in the 5' regulatory region may be a useful marker for serum lipid, serum protein, and fatty acid composition in future marker-assisted selection for duck breeding.

Key words: Duck; *SCD1*; SNPs; Serum biochemical levels; Fatty acid composition

INTRODUCTION

Stearoyl-coenzyme A desaturase 1 (*SCD1*) is the critical limiting enzyme in the biosynthesis of monounsaturated fatty acids (MUFAs) from their diet-derived or *de-novo*-synthesized saturated fatty acid precursors, and is a key factor in the metabolism of lipids and in body weight (Ntambi and Miyazaki, 2004; Tan et al., 2014). Therefore, the *SCD1* gene is regarded as an important candidate gene affecting serum biochemical levels and fatty acid composition. *SCD1* introduces a *cis*-double bond at the $\Delta 9,10$ location of palmitoyl-CoA (C16:0) and stearoyl-CoA (C18:0) to form palmitoleoyl-CoA and oleoyl-CoA, respectively (Paton and Ntambi, 2009). The *SCD1* protein includes four transmembrane domains, with both the N- and C-termini oriented to the cytosol, and is rapidly degraded in microsomes (Castro et al., 2011). Expression of the *SCD1* gene is regulated by hormonal, dietary, genetic, and environmental factors. Under a normal diet, *SCD1* mRNA expression is high in brown and white adipose tissues, and in the Harderian, preputial, and meibomian glands (Mauvoisin and Mounier, 2011). *SCD1* gene expression is dramatically decreased by over ≤ 40 -fold in the liver tissue and heart of fasting animals compared with animals on a high-carbohydrate diet. This is due to the insulin-mediated activation of sterol regulatory element-binding protein and the subsequent transcriptional activity of the *SCD1* gene promoter that contains positive or negative transcription-factor-binding sites to regulate the gene's transcription (Hodson and Fielding, 2013; Lu et al., 2014; Dobrzyn et al., 2015; Manor et al., 2015). *SCD1* is one of the target genes of liver X receptor (LXR) activation, which promotes its expression among various tissues. For example, *SCD1* gene expression was increased by more than 10- and 3.5-fold in mouse hepatocytes and human aortic endothelial tissues, respectively, after being treated with T0901317, a synthetic LXR ligand (Zhang et al., 2013a; Yamazaki et al., 2014; Ikeda et al., 2015). In recent years, numerous studies have demonstrated that the accumulation of *SCD1* substrates can promote steatohepatitis, atherosclerosis, inflammation, and pancreatic beta-cell dysfunction in preclinical rodent models (Fujiwara et al., 2015). On the contrary, *SCD1* deficiency can lead to a decrease of body adiposity, an increase of insulin sensitivity, and prevention from diet-induced obesity. Therefore, *SCD1* depressors are considered a new method for treating nonalcoholic steatohepatitis, skin disorders, hepatitis C virus infection,

diet-induced obesity, Alzheimer's disease, insulin resistance, and cancer (Ogasawara et al., 2014). Although the development of SCD1 inhibitors has been a hot research area, the properties and functions of the *SCD1* gene in poultry are still unknown (Zhang et al., 2013b). In this study, we searched for polymorphisms of the *SCD1* gene and detected single nucleotide polymorphisms (SNPs) in its 5' regulatory region in 165 Cherry Valley ducks. By verifying the association of *SCD1* SNPs with serum biochemical levels and fatty acid composition in these ducks, the specific SNP could be used as a valuable marker for marker-assisted selection duck breeding.

MATERIAL AND METHODS

Sample preparation and data collection

Blood samples from the wing vein were taken from 165 healthy female Cherry Valley ducks of parental generation. All animals were maintained in a semi-open house, reared under normal management conditions, fed commercial corn-soybean diets according to National Research Council (NRC) (1994) nutrition standards of duck, and slaughtered with animal welfare methods at 10 weeks at the animal farm of the Institute of Poultry Science, Guizhou University, Guiyang, Guizhou, China.

Genomic DNA samples from each individual were extracted with the TIANamp Blood DNA Extraction Midi Kit (Tiangen Biotech, Beijing, China), and preserved at -20°C. Serum biochemical indexes, including levels of albumin, total protein, globulin, lactate dehydrogenase, alkaline phosphatase, triglyceride, total cholesterol, cholinesterase, and IgA were measured with the AU5800 automatic biochemical analyzer (Beckman Coulter, Brea, CA, USA). The C12:0, C14:0, C14:1, C15:0, C16:0, C16:1, C17:0, C18:0, C18:1, C18:2, C18:3, C20:0, C20:1, C20:2, C20:3, and C20:4 fatty acids in breast muscle of each duck were measured as described by Han et al. (2013).

PCR amplification of *SCD1* gene

Based on the duck *SCD1* gene sequence (GenBank accession No. NW_004677643.1), primers were designed to amplify a 401-bp fragment from g.936399 to g.936799 of the 5' regulatory region of the gene. The primers used were as follows: SCD-1-F: 5'-AAC AAG ACC TTC TCC TTC CTG-3'; and SCD-1-R: 5'-TTT CTG AGT TTC GCC TGA C-3'. The 20- μ L PCR volume included 10 μ L 2X HiFi-PCR Master (PFU), 7 μ L ddH₂O, 1 μ L each primer (10 μ M), and 1 μ L genomic DNA (100 ng/ μ L). The PCR amplification procedure was as follows: an initial 95°C for 10 min, followed by 30 cycles of denaturing for 35 s at 94°C, annealing for 35 s at 56.5°C, extending for 40 s at 72°C, and a final extension for 8 min at 72°C. The amplified products were detected by 1.8% agarose gel electrophoresis.

Polymorphism screening and sequencing

The PCR product (3 μ L) from each individual was mixed with 7 μ L denaturing buffer contained 25 mM EDTA, 95% deionized formamide, 0.025% bromophenol blue, and 0.025% xylene cyanol, and then heated at 98°C for 15 min. The reaction mixture was subsequently immediately cooled on ice and placed in a -20°C freezer for 5 min. The treated PCR products were subjected to 8% acrylamide:bisacrylamide (39:1) electrophoresis in 1X Tris/borate/

EDTA buffer for 8-10 h at a constant 140V. Then, the gel was stained with 0.1% silver nitrate and photographed for determining the genotypes. Three samples from each different genotype were sequenced by two-way dye-terminator sequencing at Sangon Biotech Company (Shanghai, China).

Data statistical analysis

The genotype frequency, allele frequency, heterozygosity (H_E), polymorphism information content (PIC), and Hardy-Weinberg equilibrium were calculated by using the software POP GENE version 1.31. Sequence alignment and analysis were carried out with DNASTar version 7.1. Associations between the linkage genotypes of the *SCDI* gene SNPs and serum biochemical levels and fatty acid composition were analyzed with the least-squares method applied in the general linear model program of SPSS version 19.0, based on the linear equation $Y = \mu + G + e$, where Y is the observed value for each trait, μ is the overall mean for each trait, G is the fixed effect of SNP genotypes, and e is the random error value.

RESULTS

Detection of SNPs and genotyping

The fragment lengths of the PCR amplification products of *SCDI* were consistent with that of the target fragment, showing clear single bands on the gel (Figure 1). Single-strand conformation polymorphism analysis indicated the presence of six genotypes (AA, CC, BB, AC, AB, and BC) and three alleles (A, B, and C) (Figure 2). DNA sequencing and sequence alignment showed two novel variations, corresponding to the G→C and C→T mutations located at positions 936516 and 936551 of the whole duck genome shotgun sequence (NW_004677643.1) (Figure 3), respectively. Two SNPs, g.936516 C > G and g.936551 T > C, were respectively located 1325 and 1290 bp upstream of the translation start point of *SCDI*, which belongs to the 5' regulatory region.

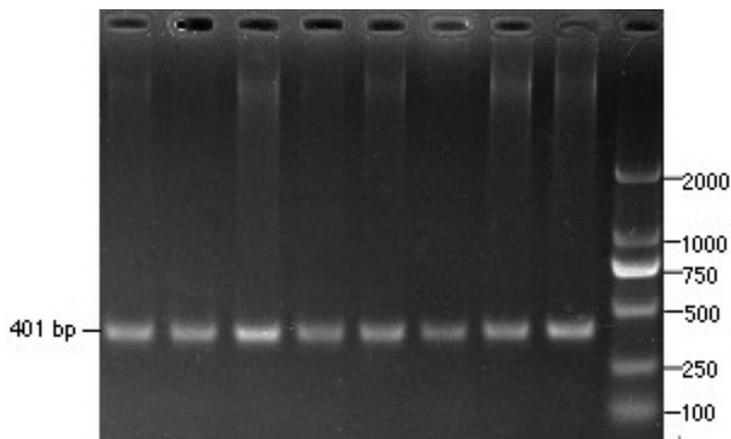


Figure 1. Detection of PCR products.

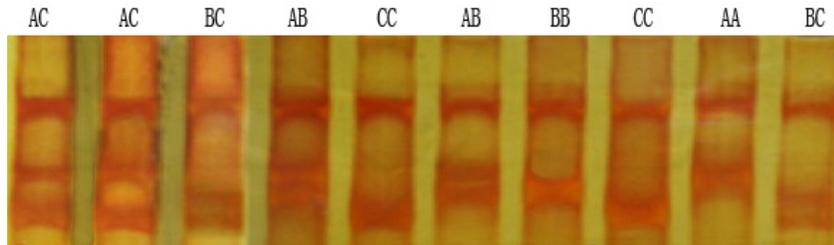


Figure 2. Single-strand conformation polymorphism analysis of different genotypes.

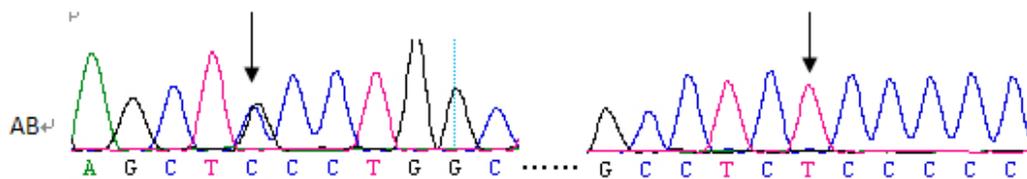


Figure 3. Sequence alignment of the different genotypes.

Genotype and allele frequencies

The genotype and allele frequencies, H_E , effective number of alleles (N_E), PIC, and Hardy-Weinberg equilibrium values from the linkage of the two mutant sites in the 5' regulatory region of the duck *SCD1* gene are shown in Table 1. The linkage of the two SNP sites resulted in six genotypes (AA, BB, CC, AB, AC, and BC) and three alleles (A, B, and C). BB and B were the dominant genotype and allele, respectively. The PIC value of 0.617, being greater than 0.5, indicates high polymorphism. The chi-square test indicated that the genotype distributions were not in Hardy-Weinberg equilibrium ($P < 0.01$).

Table 1. Characteristics from the linkage of two SNP loci in the 5' regulatory region of the *SCD1* gene.

Genotype frequencies						Allele frequencies			H_E	N_E	PIC	χ^2
AA	AB	BB	CC	AC	BC	A	B	C				
0.067	0.073	0.321	0.164	0.284	0.091	0.245	0.403	0.352	0.653	2.881	0.617	75.63

$\chi^2_{0.01} (d.f. = 5) = 15.09$.

Associations of the two mutant sites with serum biochemical levels and fatty acid composition

Comparisons of the least-squares means and standard errors of the serum biochemical levels, in relation to the genotypes from linkage of the two SNPs (g.936516 C > G and g.936551 T > C) in the 5' regulatory region of *SCD1*, are listed in Table 2. For albumin, total protein, triglyceride, total cholesterol, cholinesterase and globulin, ducks with genotype AA had lower than those with the other five genotypes, whereas ducks with genotype BC had higher than others. For albumin, total protein and globulin, ducks with genotype CC had higher than others for triglyceride, total cholesterol and cholinesterase. The data suggested that the SNPs had an effect on serum protein composition and lipid.

Table 2. Association of genotypes, from the linkage of two SNPs, with serum biochemical indexes.

Index	Genotype						P value
	AA(11)	AB(12)	AC(47)	BB(53)	BC(15)	CC(27)	
Albumin (g/L)	15.932 ± 0.226 ^a	18.158 ± 0.876 ^b	18.039 ± 0.153 ^b	19.935 ± 0.208 ^{bd}	26.967 ± 0.488 ^c	21.95 ± 0.345 ^d	0.021
Total protein (g/L)	31.043 ± 0.744 ^a	35.275 ± 1.403 ^b	34.338 ± 2.338 ^b	36.689 ± 0.685 ^{bd}	40.283 ± 1.607 ^c	38.317 ± 1.136 ^{cd}	0.033
Globulin (g/L)	25.111 ± 0.625 ^a	28.117 ± 1.013 ^{bd}	27.299 ± 0.424 ^{ab}	28.755 ± 0.576 ^{bd}	34.317 ± 1.351 ^c	30.367 ± 1.867 ^d	0.042
LDH (U/L)	874.929 ± 63.061	849.500 ± 46.813	941.811 ± 42.724	871.652 ± 58.088	940.500 ± 136.227	989.083 ± 96.327	0.109
ALP (U/L)	248.107 ± 17.798	213.250 ± 12.015	251.934 ± 12.059	282.288 ± 16.395	216.167 ± 38.449	292.583 ± 27.187	0.072
TG (mM)	0.896 ± 0.081 ^a	0.972 ± 0.012 ^a	1.078 ± 0.055 ^a	1.347 ± 0.075 ^b	1.208 ± 0.176 ^b	1.479 ± 0.124 ^b	0.021
TC (mM)	5.026 ± 0.189 ^a	5.470 ± 0.182 ^a	5.408 ± 0.128 ^a	6.514 ± 0.174 ^b	6.412 ± 0.407 ^b	7.345 ± 0.288 ^b	0.011
Cholinesterase (U/L)	1.977 ± 0.119 ^a	2.473 ± 0.069 ^{ab}	2.119 ± 0.081 ^a	2.685 ± 0.11 ^b	2.262 ± 0.257 ^a	2.717 ± 0.182 ^b	0.001
IgA (mg/L)	0.036 ± 0.004	0.039 ± 0.002	0.038 ± 0.003	0.042 ± 0.004	0.033 ± 0.008	0.040 ± 0.006	0.321

Data are reported as least-squares means ± standard errors. Values with different superscript letters in the same line are significantly different (LSD test, $P < 0.05$) in the six genotypes. LDH, lactate dehydrogenase; ALP, alkaline phosphatase; TG, triglyceride; TC, total cholesterol.

The SNPs and their genetic effects on the fatty acid composition in ducks are shown in Table 3. For C12:0, C14:0, C14:1, C15:0, C16:0, C16:1, C18:1, C20:0, UFAs and MUFAs, the birds with genotype AA was the highest among six genotypes, in contrast, genotype BC was the lowest. The birds with genotype BC was the highest among all genotypes for C17:0, C18:0, C18:2, C18:3, C20:1, C20:3, C20:4, PUFA and EFA, however, genotype AA was the lowest. For C20:2, the birds with genotypes BC and CC had higher than genotypes AA, AB, and AC. The results indicated that fatty acid composition was probably influenced by SNPs.

Table 3. Association of genotypes, from the linkage of two SNPs, with fatty acids composition (%).

Index	Genotype						P value
	AA(11)	AB(12)	AC(47)	BB(53)	BC(15)	CC(27)	
C12:0	0.055 ± 0.004 ^a	0.043 ± 0.003 ^b	0.045 ± 0.002 ^b	0.042 ± 0.002 ^b	0.039 ± 0.004 ^b	0.043 ± 0.003 ^b	0.023
C14:0	0.607 ± 0.027 ^a	0.510 ± 0.014 ^b	0.540 ± 0.013 ^b	0.474 ± 0.012 ^c	0.329 ± 0.023 ^d	0.406 ± 0.017 ^c	0.012
C14:1	0.032 ± 0.004	0.027 ± 0.002	0.029 ± 0.002	0.028 ± 0.002	0.023 ± 0.003	0.031 ± 0.003	0.114
C15:0	0.085 ± 0.003 ^a	0.076 ± 0.003 ^b	0.076 ± 0.002 ^b	0.077 ± 0.002 ^b	0.073 ± 0.003 ^b	0.076 ± 0.002 ^b	0.032
C16:0	28.056 ± 0.404 ^a	27.342 ± 0.332 ^{ab}	27.055 ± 0.195 ^{ab}	26.766 ± 0.184 ^{ab}	25.901 ± 0.346 ^b	26.843 ± 0.258 ^{ab}	0.022
C16:1	3.175 ± 0.185 ^a	2.596 ± 0.187 ^b	2.427 ± 0.091 ^b	2.021 ± 0.084 ^b	1.068 ± 0.159 ^c	1.715 ± 0.118 ^{bc}	0.016
C17:0	0.180 ± 0.015 ^a	0.203 ± 0.011 ^a	0.196 ± 0.007 ^a	0.209 ± 0.007 ^{ab}	0.257 ± 0.013 ^b	0.235 ± 0.009 ^b	0.042
C18:0	9.205 ± 0.802 ^a	11.759 ± 0.641 ^b	11.474 ± 1.188 ^b	13.117 ± 1.165 ^{bc}	18.669 ± 1.187 ^d	15.933 ± 0.912 ^{cd}	0.017
C18:1	42.851 ± 1.335 ^a	37.823 ± 1.789 ^{bc}	39.51 ± 1.486 ^{ab}	34.455 ± 1.458 ^{cc}	27.895 ± 1.861 ^d	31.745 ± 2.642 ^{cd}	0.032
C18:2	15.352 ± 0.674 ^a	17.812 ± 0.702 ^{ab}	16.977 ± 0.884 ^{ab}	18.937 ± 0.679 ^b	22.99 ± 0.649 ^c	20.675 ± 0.911 ^{bc}	0.026
C18:3	0.119 ± 0.018 ^a	0.153 ± 0.014 ^{ab}	0.152 ± 0.009 ^{ab}	0.203 ± 0.008 ^{bd}	0.307 ± 0.015 ^c	0.257 ± 0.011 ^{cd}	0.019
C20:0	0.607 ± 0.046	0.585 ± 0.034	0.624 ± 0.022	0.567 ± 0.021	0.558 ± 0.039	0.571 ± 0.029	0.135
C20:1	0.158 ± 0.026 ^a	0.184 ± 0.031 ^{ab}	0.224 ± 0.013 ^{bc}	0.286 ± 0.012 ^c	0.564 ± 0.022 ^d	0.372 ± 0.017 ^c	0.024
C20:2	0.176 ± 0.018 ^a	0.194 ± 0.031 ^a	0.201 ± 0.009 ^a	0.226 ± 0.008 ^{ab}	0.268 ± 0.016 ^b	0.270 ± 0.012 ^b	0.016
C20:3	0.065 ± 0.019 ^a	0.105 ± 0.011 ^a	0.104 ± 0.009 ^a	0.143 ± 0.009 ^a	0.245 ± 0.016 ^b	0.176 ± 0.012 ^{ab}	0.033
C20:4	0.282 ± 0.041 ^a	0.330 ± 0.031 ^{ab}	0.380 ± 0.020 ^{ab}	0.489 ± 0.019 ^b	0.813 ± 0.035 ^c	0.654 ± 0.026 ^d	0.013
UFA	61.211 ± 1.225 ^a	59.224 ± 1.825 ^a	60.004 ± 1.447 ^a	56.788 ± 1.421 ^b	53.867 ± 1.792 ^c	55.638 ± 1.599 ^{ab}	0.025
MUFA	46.216 ± 1.305 ^a	40.630 ± 1.148 ^b	42.190 ± 1.324 ^b	36.790 ± 1.113 ^c	29.551 ± 1.069 ^d	33.863 ± 1.317 ^{cd}	0.018
PUFA	15.994 ± 0.721 ^a	18.594 ± 0.754 ^b	17.814 ± 0.803 ^{ab}	19.998 ± 0.692 ^{bc}	24.316 ± 0.623 ^d	21.775 ± 0.887 ^c	0.039
EFA	15.753 ± 0.195 ^a	18.295 ± 0.132 ^b	17.509 ± 0.094 ^{ab}	19.629 ± 0.089 ^{bc}	24.111 ± 0.167 ^d	21.586 ± 0.125 ^c	0.037

Data are reported as least-squares means ± standard errors. Values with different superscript letters in the same line are significantly different (LSD test, $P < 0.05$) in the six genotypes. MUFA, monounsaturated fatty acid; UFA, unsaturated fatty acid; PUFA, polyunsaturated fatty acid; EFA, essential fatty acid.

DISCUSSION

Studies have demonstrated that serum biochemical indexes play a key role in reflecting the physicochemical properties and revealing the metabolic mechanisms (especially mineral metabolism) of the individual (Xu et al., 2015). In patients with end-stage renal disease, biochemical markers of altered mineral metabolism are verified to be associated with increased mortality (Von Roemeling et al., 2013). The fatty acid composition in ducks has been used as an economically related trait in the duck industry owing to increasing awareness of the consumer preference for a healthy diet. Accumulated evidence suggests that the composition of fatty acids affects human health more greatly than does a diet high in fat alone. It has been well evaluated that the uptake of PUFAs is negatively associated with atherosclerosis, diabetes mellitus, and other cardiovascular diseases. Studies with various animal models indicated that vaccenic acid (11t C18:1) may confer benefit to human health (Liu, 2015). Therefore, increasing the contents of beneficial fatty acids in the daily ration of ducks can add value to the duck products. At present, the improvement of beneficial fatty acids is done mainly through dietary manipulation in ducks. However, even in ducks fed the same diet, the fatty acid composition can vary significantly between individuals, indicating the need for DNA markers to increase the content of beneficial fatty acids. The crucial role of SCD1 in the physiology and biochemistry of lipid metabolism is now being widely studied. SCD1 deficiency has been shown to result in reduced body adiposity, and increased insulin sensitivity and resistance to diet-induced obesity (Yokoyama et al., 2012). In our study, two novel SNPs located in the 5' regulatory region of the *SCD1* gene were identified and analyzed. Sequence analysis indicated that new alleles were produced by G→C and C→T mutations in positions 936516 and 936551 of the duck genome, respectively. Linkage of the two SNP sites resulted in six genotypes (AA, CC, BB, AC, AB, and BC) and three alleles (A, B, and C) in the population. Genotype BB and allele B were dominant, with frequencies of 0.321 and 0.403, respectively. This reflected that there is very high genetic diversity within the *SCD1* gene in Cherry Valley ducks. In group differentiation of breeding, high genetic information is advantageous but not low genetic information (Botstein et al., 1980). The chi-square test indicated that the genotype distribution was markedly inconsistent with Hardy-Weinberg equilibrium ($P < 0.01$), and the genotype frequencies may have been distorted by migration, mutation, selection, or other reasons. In selected breeding populations, a genotype frequency that disagrees with Hardy-Weinberg equilibrium should be expected for SNPs or polymorphic loci, with impact on economic traits under selection (Groeneveld et al., 2010).

We next attempted to correlate the six genotypes with serum biochemical levels and fatty acid composition. The statistical results showed that of the homozygous genotypes, CC was the highest for all serum biochemical indexes, including fatty acids C17:0, C18:0, C18:2, C18:3, C20:1, C20:2, C20:3, C20:4, PUFAs, and EFAs. Homozygous AA was highest for C12:0, C14:0, C14:1, C15:0, C16:0, C16:1, C18:1, C20:0, UFAs, and MUFAs. The linkage of the two mutant sites had significant effects on the levels of serum albumin, total protein, globulin, triglyceride, total cholesterol, cholinesterase, and 16 kinds of fatty acids, except for C14:1 and C20:0, at 10 weeks ($P < 0.05$). Therefore, it is consistent with the function of the *SCD1* gene, which plays a crucial role in regulating lipid metabolism. Results of this study suggest that the *SCD1*-gene-specific SNPs in the 5' regulatory region may be a useful marker for serum lipids, serum proteins, and fatty acid composition in future marker-assisted selection breeding programs for ducks. Moreover, ducks with the CC homozygous genotype may be

predominant in PUFA content. It was suggested that the C allele may have a positive effect on PUFAs of potential health benefits, whereas allele A may have a positive effect on the contents of saturated fatty acids and MUFAs, except for C17:0, C18:0, and C20:1. These SNPs found by us were not in a coding region. Possible reasons for their effect on serum biochemical levels and fatty acid composition could be that they promote the expression of the *SCD1* gene by influencing the 5' regulatory region structure and promoter activity. Since related experiments were not carried out with other duck strains, further studies with other poultry strains are necessary. To date, *SCD1* gene polymorphisms associated with production performance in poultry have not been well documented. This is the first study to analyze the association of the *SCD1* gene with fatty acids and serum biochemical levels in ducks. Similar research has been reported in pigs, where the C/T mutation at position 3101 was detected in exon 2 of the porcine *SCD1* gene and found to be significantly associated with carcass weight (Renaville et al., 2015). The observed effect on carcass weight may result from an effect of SCD on the feed conversion ratio and daily body weight gain, as reported by Bartz et al. (2013), and/or on growth as reported by Chen et al. (2011) in goats. In Chinese goat breeds, three SNPs (IVS3+55A > G in intron 3, and EX3_15G > A and EX3_68A > G in exon 3) were discovered by DNA pooling and PCR-restriction fragment length polymorphism, and the EX3_15G > A missense mutation resulted in an amino acid substitution from Val to Met in the 109 amino acid position of the SCD protein (359 amino acids). The authors described three haplotypes [type A (A-G-A), type C (G-G-A), and type B (A-A-G)] and six genotypes (AA, CC, BB, AC, AB, and BC). Moreover, genotype CC may be considered as a molecular marker for superior body length, body height, and chest circumference (Chen et al., 2011). Three SNPs in the fifth exon of cattle have been detected, where the third SNP of T878C led to a Val/Ala substitution and has been relevant for a higher MUFA percentage and lower melting point of intramuscular fat (Duchemin et al., 2013) as well as for a higher level of SCD activity and MUFA content in dairy cattle (Taniguchi et al., 2004; Mele et al., 2007; Macciotta et al., 2008; Conte et al., 2010). The missense mutation A293V of *SCD1* in summer and winter milk samples was positively correlated with C8:0-C14:0, *cis*-9 C16:1, and C16 and conjugated linoleic acid unsaturation indexes, and negatively correlated to C10:1, *cis*-9 C14:1, C18:0, *trans*-11 C18:1, and C10-C14 unsaturation indexes (Avilés et al., 2015). The *SCD* locus has been suggested for marker-assisted selection breeding for improving milk production in dairy cattle and obtaining a healthier final product (Conte et al., 2010; Matsushashi et al., 2011; Avilés et al., 2013). Taken together, the results indicate that the *SCD1* gene could be used as an experimental candidate gene to influence carcass, meat quality, reproduction, and growth traits in animals. Further studies will be implemented to validate the function of the duck *SCD1* gene polymorphisms, such as the coding region, untranslated region, and promoter, for future application to duck breeding.

CONCLUSION

Two novel SNPs were identified in the duck *SCD1* gene and their associations with duck serum biochemical levels and fatty acid composition were analyzed. Our study provides evidence that the *SCD1* gene might have potential effects on serum biochemical levels and fatty acid composition in ducks. Therefore, in the breeding program for ducks, further research will be carried out by using the SNPs for marker-assisted selection in a larger population. It is also important to investigate whether the *SCD1* gene has a role in the development of production performance and whether it participates in linkage disequilibrium with other regions with mutations.

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