Genetics and Molecular Biology, 34, 1, 56-61 (2011)

www.sbg.org.br

Research Article

Association of *PPARγ2* polymorphisms with carcass and meat quality traits in a Pietrain x Jinhua F2 population

Zhe Chen[1], Xiaofeng Zhao[2], Xiaoling Jiang[1], Xiaoling Guo[1], Zunzhou Lv[1], Hongmei Zhou[1] and Ningying Xu[1]

[1]*College of Animal Science, Zhejiang University, Zhejiang Province, P.R. China.*
[2]*College of Life and Environmental Sciences, Hangzhou Normal University, Zhejiang Province, P.R. China.*

Abstract

The *PPARγ2* gene is a key regulator of both proliferation and preadipocyte differentiation in mammals. Herein its genotype and allele frequencies were analyzed using PCR-SSCP in eight pig breeds (N = 416). Two kinds of polymorphisms of the *PPARγ2* gene were detected, including a previously reported shift SNP A177G (Met59Val) in exon 1 and a novel silent mutation G876A in exon 5. The results revealed that European pig breeds carry a higher allele A frequency at the A177G locus and a fixed GG genotype at the G876A locus. Allele A at the G876A locus was only found in Jinhua pigs. The association between haplotype (A177G/G876A) and carcass and meat quality traits was analyzed in a Pietrain x Jinhua F2 population (N = 248). The *PPARγ2* gene was found to be significantly associated with backfat thickness at the shoulder ($p < 0.05$), 6-7th ribs ($p < 0.01$), last rib ($p < 0.01$), gluteus medius ($p < 0.05$) and ham weight ($p < 0.01$). Significant effects of different haplotypes on ham weight and backfat thickness at the 6-7th ribs, last rib, and gluteus medius were also observed.

Key words: PPARγ2 gene, haplotype, carcass and meat quality, pigs.

Received: April 23, 2010; Accepted: October 20, 2010.

Introduction

Genetic effects on porcine meat quality have historically been widely recognized and marker-assisted selection has been used by animal breeders to improve the quality and performance of livestock (Van der Steen *et al.*, 2005). Reducing body fat, increasing lean percentage, and improving meat quality are critical issues for the pig breeding industry. Thus, there is great interest in having comprehensive molecular marker information for improvement of domestic swine.

The peroxisome proliferator-activated receptor-γ (*PPARγ*) is a member of the nuclear hormone receptor superfamily. It exists as two distinct isoforms (*γ1* and *γ2*) produced from a single gene sequence by alternative splicing, resulting in amino acid differences in the N-termini (Fajas *et al.*, 1997; Omi *et al.*, 2005). *PPARγ1* is expressed in a variety of tissues at a relatively low level, while *PPARγ2* is exclusively expressed in adipose tissue and at ten times higher transcriptional activity (Tontonoz *et al.*, 1994a; Werman *et al.*, 1997). *PPARγ2* is known as a key regulator of adipogenesis involved in the regulation of fatty acid uptake and storage (Rosen *et al.*, 1999). It participates in the transcriptional activation of several adipogenic transcription factors and lipogenic genes (Wu *et al.*, 1999; Pasceri *et al.*, 2000; Rosen *et al.*, 2002; Arimura *et al.*, 2004), and directs the differentiation of preadipocytes to adipocytes. Interestingly, overexpression of *PPARγ2* in fibroblast cell lines using retroviral vectors efficiently converts them to adipocytes (Tontonoz *et al.*, 1994b). Previous research also supported this view, indicating a dual role for *PPARγ2* in the regulation of both proliferation and preadipocyte differentiation (Fajas *et al.*, 2002; Samulin *et al.*, 2008). Therefore, *PPARγ2* appeared to be a promising candidate gene for improving carcass and meat quality traits. However, very little work has yet been conducted to investigate the role of the porcine *PPARγ2* gene in carcass and meat quality.

Emnett *et al.* (2000) found a signicant association between the *PPARγ2* Met59Val polymorphism, meat quality and backfat thickness in Berkshire, Duroc, Hampshire and Landrace swine breeds, while Grindflek *et al.* (2004) found a significant association between the Met59Val/A220G/G324A haplotype and ham weight in a Norwegian pig population, but they did not denote a major effect on backfat thickness.

The current study was designed to detect novel single nucleotide polymorphisms (SNPs) of the porcine *PPARγ2*

Send correspondence to Ningying Xu. College of Animal Science, Zhejiang University, 310029 Hangzhou, Zhejiang Province, P.R. China. E-mail: nyxu@zju.edu.cn.

gene using polymerase chain reaction-single strand conformation polymorphism (PCR-SSCP) and DNA sequencing, and to further investigate the relationship between these SNPs and carcass and meat quality in a Pietrain x Jinhua F2 crossbred population.

Material and Methods

Animal and data collection

The association between the SNPs and recorded traits were analyzed in a Pietrain x Jinhua F2 crossbred swine population. The Pietrain x Jinhua resource population was constructed from purebred Pietrain sires and Jinhua dams, including 6 F1 boars, 20 F1 sows and 248 F2 progeny. The 248 PJF2 offspring were raised under standard conditions at The Experimental Pig Farm of Zhejiang University and slaughtered at a commercial abattoir. In addition, DNA samples of eight pig breeds were collected, consisting of 96 Jinhua, 36 Jiaxing Black, 54 Bihu, 66 Shengxian Hua, 37 Landrace, 67 Yorkshire, 30 Duroc and 30 Pietrain pigs (Table 1). The sampled pigs were from sources that were unrelated throughout the last three generations.

PJF2 animals were slaughtered (following electric shock) at 219 days of age (SD = 31.4 days, live weight 81.3 ± 10.58 kg). After slaughter, backfat thickness (BFT) was measured on the left side at four locations (shoulder, 6-7th ribs, last rib, and gluteus medius). At 45 min post-mortem, muscle pH and temperature measurements were taken on the longissimus dorsi (LD) muscle using a Thermo Scientific Orion pH meter (Model 230A Plus, USA) and a digital thermometer (Ama-digit 14 TH 35° C-500 °C Amarell electronic, Kreuzwertheim, Germany), respectively. Loin eye area (LEA) of the LD muscle was traced on an acetate film between the 13/14th ribs and quantitated by planimetry. Samples of the longissimus dorsi muscle were taken at the 13th rib for the determination of water-holding capacity (WHC), intramuscular fat (IMF), protein (IMP) and water (IMW) content. WHC was determined with the filter paper press method (Wierbicki and Deatherage, 1958), while intramuscular fat, protein and water content were determined using a Meat Analyzer (Antaris II FT-NIR analyzer, Thermo Electric company, USA). Additionally, the ham was cut from the last lumbar vertebra and the feet were removed.

Primers, amplification conditions and PCR-SSCP analysis

Two pairs of primers were designed based on the porcine *PPARγ2* gene mRNA sequence (GenBank, accession number NM_214379) and human *PPARγ2* gene chromosomal sequence (GenBank, accession number NC_000003). For amplification of the PPARγ*2* gene, the *PPARγ2*-P1 primer (5' TGACACCGAGATGCCGTT 3' and 5' TGGAGCTTCAGGTCGTACTT 3') were used. This primer combination generates a 204 base pair (bp) amplicon from exon 1. The second primer pair named *PPARγ2*-P2 (5'CTTGCAGCCATTTGTCATC3' and 5'AGGGCTTTCTCAGGCTCTT3') amplifies a 350 bp fragment from exon 5.

PCR amplifications were carried out in 25 μL reaction volumes containing at the following final concentrations: 50 ng of template DNA, 400 μM of dNTPs (Sangon, China), 0.25 μM of each primer and 1 unit (U) of Taq polymerase (TaKaRa, Japan). The PCR protocol consisted of an initial denaturation at 94 °C for 5 min, followed by 36 cycles of denaturation at 94 °C for 30 s, annealing at 55 °C for *PPARγ2*-P1 or 51 °C for *PPARγ2*-P2 for 30 s, and extension at 72 °C for 30 s, with a final extension at 72 °C for 10 min.

Table 1 - Genotype and allele frequencies of SNPs A177G and G876A in eight pig breeds.

Breed	N	SNP A177G genotype frequency			Allele frequency	χ^2	SNP G876A genotype frequency			Allele frequency	χ^2
		AA	AG	GG	A (%)		GG	GA	AA	G (%)	
Jinhua	96	37	45	14	0.62	0.24	24	67	5	0.60	20.73**
Jiaxing Black	36	15	18	3	0.67	0.90	36	0	0	1.00	[1]
Bihu	54	16	36	2	0.63	11.48**	54	0	0	1.00	[1]
Shengxian Hua	66	8	44	14	0.45	8.57**	66	0	0	1.00	[1]
Pietrain	30	30	0	0	1.00	[1]	30	0	0	1.00	[1]
Landrace	37	24	11	2	0.80	0.14	37	0	0	1.00	[1]
Duroc	30	27	3	0	0.95	4.46*	30	0	0	1.00	[1]
Yorkshire	67	38	25	4	0.75	0.06	67	0	0	1.00	[1]

Source of breeds: Jinhua (Zhejiang Jiahua Pig Breeding Co., LTD), Jiaxing Black (Shuangqiao Pig Breeding Farm), Bihu (Lishui Pig Breeding Farm), Shengxian Hua (Shangyu Shenghua Pig Breeding Co., LTD), Yorkshire and Pietrain (The Experimental Pig Farm of Zhejiang University), Landrece (Zhejiang Dengta Pig Breeding Farm), Duroc (Xiaoshan Duroc Pig Breeding Farm).

[1]genotype is fixed, the corresponding breed is not in Hardy-Weinberg equilibrium.

χ^2 Test for Hardy-Weinberg equilibrium, $^{*}p < 0.05$, $^{**}p < 0.01$.

The porcine *PPARγ2* alleles were detected using PCR-SSCP. A 5 μL aliquot of each amplicon was diluted in denaturing solution (98% formamide, 10 mM EDTA, 0.025% bromophenol blue, 0.025% xylene-cyanol) denatured at 95 °C for 5 min, rapidly cooled on ice and resolved in acrylamide:bisacrylamide (29:1) (Bio-Rad) gels at 400 V for 4 h at 4 °C, in 1 x TBE buffer (89 mM Tris base, 89 mM boric acid, 2 mM EDTA, pH 8.0). The gels were silver-stained according to the method of Bassam *et al.* (1991).

Sequence analysis

Nucleotide sequence translation and comparisons were carried out using the DNAMAN software. The BLAST algorithm of NCBI was used to search homologous sequences in the NCBI GenBank database.

Statistical analysis

Linkage disequilibria between all pairs of biallelic loci were calculated using measures of Lewontin's D' and r^2 (Pritchard and Przeworski, 2001) in the PJF2 population. Haplotypes were determined by the Linkage algorithm of the HaploView software (V 2.01). The genotype and allele distributions in purebreds were tested for Hardy-Weinberg equilibrium using the χ^2 test.

For studying the association between the *PPARγ2* gene haplotypes and recorded traits in the PJF2 crossbred animals, the following model was used:

$$Y_{ijklm} = \mu + S_i + F_j + G_k + l_l + \beta * X_{ijklm} + e_{ijklm}$$

where Y_{ijklm} is the observation of the trait; μ is the population mean; S_i is the fixed effect of sex; F_j is the fixed effect of the father; G_k is the fixed effect of the k^{th} haplotype; l_l is the random effect of litter; β is the regression coefficient of the slaughter weight; X_{ijklm} is the slaughter weight as covariate, and e_{ijklm} is the random residual error. The least square means method of the GLM (General Linear Model) procedure (SPSS 16.0) was used for association analysis between the *PPARγ2* haplotypes and recorded traits.

Results

Detection of single nucleotide polymorphisms

Two fragments, *PPARγ2*-P1 (204 bp) and *PPARγ2*-P2 (350 bp) were amplified from genomic DNA. These were then assayed by the SSCP technique and products with unique patterns were sequenced to detect any single base substitution. One substitution located in exon 1 at position 177 (c.+177 A > G, GenBank accession No. NM_214379) causes an amino acid change from methionine to valine (Met59Val). This SNP has already been described in previous studies (Emnett *et al.*, 2000; Grindflek *et al.*, 2004). The primer *PPARγ2*-P2 detected a novel polymorphism in exon 5 at position 876 (c.+876 G > A, GenBank accession No. NM_214379) of the porcine *PPARγ2* gene. This SNP represents a silent mutation.

Genotype frequencies in different pig populations

The combined effects of the exon 1 and exon 5 substitutions were estimated as haplotype substitution effects. Genotype and allele frequencies of the SNPs A177G and G876A within pure breeds are shown in Table 1.

At A177G, allele A had a high frequency (> 75%) in European pig breeds, especially fixed in Pietrain swine, and a relatively lower frequency in Chinese native breeds (< 67%). In Jinhua, Jiaxing Black, Landrace and Yorkshire breeds, the alleles were at Hardy-Weinberg equilibrium. Interestingly, at G876A, allele A was found only in Jinhua pigs, whereas allele G was fixed in all other breeds. Therefore, allele A appears to be Jinhua-specific, and, consequently, the breeds were not at Hardy-Weinberg equilibrium with respect to this marker.

Haplotype analysis

Four haplotypes, H1 (A-G), H2 (A-A), H3 (G-G), and H4 (G-A), were found in PJF2 animals and had their frequencies and linkage disequilibria estimated (Table 2). Of the four types, the A-G haplotype (H1) was found to represent the majority. Linkage analysis indicated that there were certain linkage disequilibria between the two SNPs A177G and G876A (D' = 0.504).

Relationship between *PPARγ2* gene haplotypes and recorded traits

The results of the association analysis of *PPARγ2* gene haplotypes and recorded carcass and meat traits within the PJF2 population are shown in Table 3. *PPARγ2* gene haplotypes were found to be significantly associated with BFT at the shoulder and gluteus medius ($p = 0.029$ and 0.042, respectively), with significant effects observed on ham weight and BFT at the 6-7th ribs and last rib ($p = 0.000$, 0.003, and 0.003, respectively). Pigs with the H1H3 genotype had lower ham weight compared to the other genotypes ($p < 0.01$). H2H3 genotype pigs had higher BFT at the 6-7th ribs, last rib and gluteus medius ($p < 0.01$), while for BFT at the shoulder, the H2H3 genotype had relatively higher thickness, just slightly lower than that of the H1H3 genotype.

Table 2 - Haplotype frequency and linkage disequilibrium in a Pietrain x Jinhua F2 (PJF2) population.

Haplotype	Frequency
A-G	0.671
A-A	0.093
G-G	0.147
G-A	0.089
D'	0.504
r^2	0.249

Table 3 - Association of haplotypes with recorded traits.

Traits	Haplotypes (Least square means ± standard deviation)					p-value
	H1H1 (n = 122)	H1H2 (n = 45)	H1H3 (n = 5)	H2H3 (n = 40)	H3H4 (n = 36)	
BFT at shoulder (cm)	4.270 ± 0.883	4.249 ± 0.935	4.720 ± 0.311	4.569 ± 0.809	4.534 ± 0.867	0.029
BFT at 6-7th ribs (cm)	2.964^{B}±0.772	3.019^{AB}±0.832	3.260^{AB}±0.261	3.518^{A}±0.637	3.322^{AB}±0.775	0.003
BFT at last rib (cm)	2.126^{B}±0.578	2.233^{AB}±0.864	2.560^{A}±0.230	2.562^{A}±0.672	2.377^{AB}±0.694	0.003
BFT at gluteus medius (cm)	2.170^{B}±0.624	2.130^{AB}±0.836	2.260^{AB}±0.207	2.540^{A}±0.714	2.330^{AB}±0.578	0.042
Protein content (%)	23.968 ± 0.921	24.233 ± 0.865	23.845 ± 0.408	24.401 ± 0.817	24.243 ± 0.693	0.962
Water content (%)	73.700 ± 1.439	73.514 ± 1.143	74.209 ± 0.672	72.851 ± 1.785	73.813 ± 1.204	0.984
Intramuscular fat content (%)	2.724 ± 1.259	2.544 ± 0.796	2.267 ± 0.349	3.012 ± 1.650	2.365 ± 0.983	0.977
Water-holding capacity	0.424 ± 0.095	0.435 ± 0.081	0.408 ± 0.117	0.420 ± 0.108	0.418 ± 0.153	0.813
Loin eye area (cm^2)	32.993 ± 6.329	32.217 ± 5.971	32.220 ± 4.894	30.537 ± 5.629	31.723 ± 5.924	0.148
LD pH	6.2495 ± 0.344	6.2379 ± 0.289	6.2180 ± 0.135	6.1545 ± 0.410	6.2351 ± 0.288	0.601
LD temperature (°C)	38.699 ± 1.422	38.633 ± 1.579	38.170 ± 1.058	38.810 ± 1.604	38.346 ± 1.488	0.507
Ham weight (kg)	7.541^{B}±1.021	7.528^{B}±1.149	6.680^{A}±2.442	7.671^{B}±0.852	7.569^{B}±0.986	0.000

Means with different superscripts A and B are statistically different at p <0.01.

Discussion

The Jinhua and Pietrain breeds are significantly different in carcass and meat quality phenotypes. As a Chinese domesticated breed, the Jinhua pig has a reputation for its thin skin, fine bones and tender meat, but has higher BFT and IMF content than other breeds. In contrast, the Pietrain type, which has a relatively lower BFT and IMF content, is famous for its high quality and high proportion of lean meat *vs.* fat content, and has been used worldwide as a sire breed. Observed differences in carcass and meat quality phenotypes are probably related to protein accretion and lipid synthesis (Renaudeau and Mourot, 2007). In this regard, it has been suggested that certain genes may play important physiological roles in fat-related pathways (Agarwal and Garg, 2006). Therefore we hypothesized that fat-related genes that are at considerable linkage disequilibrium may produce distinct phenotypes in a Pietrain x Jinhua F2 (PJF2) population. In this study, we found that genotype and allele frequencies for two analyzed polymorphisms differed between the Chinese native pig and the European pig, indicating that the two types of breeds had been subject to different genetic selection regimes.

Lai *et al.* (2008) demonstrated that the region of the *PPARγ2* promoter comprising nucleotide base pair positions -457 to +129 is essential for the CEBPD (CCAAT/Enhancer binding protein D) response which activates *PPARγ2* transcription. They furthermore suggested that the A177G SNP might affect binding efficiency and thus the gene expression level at this site. Although the G876A SNP is a silent mutation, there is substantial evidence indicating that silent mutations may affect mRNA secondary structure and stability (Shen *et al.*, 1999; Duan *et al.*, 2003), mRNA decay rates, and ultimately lead to disease (Capon *et al.*, 2004).

The porcine *PPARγ2* gene was mapped to chromosome 13 (Grindflek *et al.*, 2000) and found to be close to several quantitative trait loci (QTL) and candidate genes for birth weight, daily weight gain and BFT (Andersson *et al.*, 1994; Yu *et al.*, 1995; Evans *et al.*, 2003). Since it plays a key role in proliferation and preadipocyte differentiation, *PPARγ2* is an excellent target for studying growth and fat deposition traits. Experiments with homologous *PPARγ2*-deficient mice showed that genetic ablation of *PPARγ2* decreased adipose tissue expandability and the capacity for buffering toxic lipids in peripheral organs (Medina-Gomez *et al.*, 2007). In humans, several SNPs within the *PPARγ2* locus (Pro12Ala, C1431T, C2821T, A2819G) have been identified in different populations, and their alleles demonstrated to be associated with obesity, type 2 diabetes, increased insulin sensitivity and decreased receptor activity (Deeb *et al.*, 1998; González Sánchez *et al.*, 2002; Meirhaeghe and Amouyel, 2004; Costa *et al.*, 2009).

A total of five mutations have previously been identified in the porcine *PPARγ2* gene promoter and coding regions, these being A177G (M59V), G78A, A42G, A220G and G324A (Emnett *et al.*, 2000; Grindflek *et al.*, 2004). In our study we identified a novel Jinhua-specific SNP (A876G), as well as the previously reported A177G (M59V) polymorphism, and investigated haplotype effects in a PJF2 population.

Haplotypes can be used to correlate a specific phenotype with a specific gene in a small sample population (Drysdale *et al.*, 2000) and can provide more information on the complex relationship between DNA variation and phenotype than single SNPs may afford (Stephens *et al.*,

2001). Thus, we used haplotype information to evaluate the relationship between *PPARγ2* polymorphisms and candidate traits in swine. Our results showed significant effects on ham weight ($p < 0.01$), consistent with previous results obtained by Grindflek *et al.* (2004). We also found in the PJF2 population significant differences between the different haplotypes with respect to BFT at the 6-7th ribs ($p < 0.01$), the last rib ($p < 0.01$) and the gluteus medius ($p < 0.01$). No significant association was found between haplotypes and IMF.

Grant *et al.* (2008) first reported that exposure to the PPARγ agonist troglitazone (TRO) resulted in a 6.4-fold increase in the percentage of differentiated subcutaneous preadipocytes compared to intramuscular preadipocytes, and they conjectured that inherent differences in the capacity for adipose differentiation may exist between bovine subcutaneous and intramuscular preadipocytes. Thus, *PPARγ2* gene regulation is thought to favor subcutaneous fat deposition over intramuscular fat content.

The association observed in our study may arise from linkage between the *PPARγ2* gene and specific polymorphisms that are in linkage disequilibrium and may be located in another region of the *PPARγ2* gene, or by interaction with other quantitative trait loci. Furthermore, the linkage disequilibrium between A177G and G876A in the PJF2 population was unstable ($r^2 = 0.249$). Consequently, additional studies are needed to better assess the real impact of the effects of the *PPARγ2* gene on carcass and meat quality traits.

Acknowledgments

This work was supported by grants from the National Major Basic Research Program of China (973 Program, No. 2006CB102100) and the National High Technology Research and Development Program of China (863 program, No. 2007AA10Z158).

References

Agarwal AK and Garg A (2006) Genetic disorders of adipose tissue development, differentiation, and death. Annu Rev Genomics Hum Genet 7:175-199.

Andersson L, Haley CS, Ellegren H, Knott SA, Johansson M, Andersson K, Andersson-Eklund L, Edfors-Lilja I, Fredholm MA, Hansson I, *et al.* (1994) Genetic mapping of quantitative trait loci for growth and fatness in pigs. Science 263:1771-1774.

Arimura N, Horiba T, Imagawa M, Shimizu M and Sato R (2004) The peroxisome proliferator-activated receptor gamma regulates expression of the perilipin gene in adipocytes. J Biol Chem 279:10070-10076.

Bassam BJ, Caetano-Anollés G and Gresshoff PM (1991) Fast and sensitive silver staining of DNA in polyacrylamide gels. Anal Biochem 196:80-83.

Capon F, Allen MH, Ameen M, Burden AD, Tillman D, Barker JN and Trembath RC (2004) A synonymous SNP of the corneodesmosin gene leads to increased mRNA stability and demonstrates association with psoriasis across diverse ethnic groups. Hum Mol Genet 13:2361-2368.

Costa V, Casamassimi A, Esposito K, Villani A, Capone M, Iannella R, Schisano B, Ciotola M, Di Palo C, Corrado FC, *et al.* (2009) Characterization of a novel polymorphism in PPARG regulatory region associated with type 2 diabetes and diabetic retinopathy in Italy. J Biomed Biotechnol, Epub.

Deeb SS, Fajas L, Nemoto M, Pihlajamäki J, Mykkänen L, Kuusisto J, Laakso M, Fujimoto W and Auwerx J (1998) A Pro12Ala substitution in PPARgamma2 associated with decreased receptor activity, lower body mass index and improved insulin sensitivity. Nat Genet 20:284-287.

Drysdale CM, McGraw DW, Stack CB, Stephens JC, Judson RS, Nandabalan K, Arnold K, Ruano G and Liggett SB (2000) Complex promoter and coding region β_2-adrenergic receptor haplotypes alter receptor expression and predict *in vivo* responsiveness. Proc Natl Acad Sci USA 97:10483-10488.

Duan J, Wainwright MS, Comeron JM, Saitou N, Sanders AR, Gelernter J and Gejman PV (2003) Synonymous mutations in the human *dopamine receptor D2* (*DRD2*) affect mRNA stability and synthesis of the receptor. Hum Mol Genet 12:205-216.

Emnett RS, Grindek E, Rothschild MF, Moeller SJ, Meeker DL and Irvin KM (2000) The effects of porcine peroxisome proliferators-activated receptor gamma (PPARG) in Berkshire, Duroc, Hampshire and Landrace breeds. Proceedings of the International Plant & Animal Genome Conference, San Diego, VIII P396. http://www.intl-pag.org/pag/8/abstracts/pag8141.html.

Evans GJ, Giuffra E, Sanchez A, Kerje S, Davalos G, Vidal O, IIlán S, Noguera JL, Varona L, Velander I, *et al.* (2003) Identification of quantitative traits loci for production traits in commercial pig population. Genetics 164:621-627.

Fajas L, Auboeuf D, Raspé E, Schoonjans K, Lefebvre AM, Saladin R, Najib J, Laville M, Fruchart JC, Deeb S, *et al.* (1997) The organization, promoter analysis and expression of the human PPARgamma gene. J Biol Chem 272:18779-18789.

Fajas L, Landsberg RL, Huss-Garcia Y, Sardet C, Lees JA and Auwerx J (2002) E2Fs regulate adipocyte differentiation. Dev Cell 3:39-49.

González Sánchez JL, Serrano Ríos M, Fernández Perez C, Laakso M and Martínez Larrad MT (2002) Effect of the Pro12Ala polymorphism of the peroxisome proliferators activated receptor gamma-2 gene on adiposity, insulin sensitivity and lipid prole in the Spanish population. Eur J Endocrinol 147:495-501.

Grant AC, Ortiz-Colón G, Doumit ME, Tempelman RJ and Buskirk DD (2008) Differentiation of bovine intramuscular and subcutaneous stromal-vascular cells exposed to dexamethasone and troglitazone. J Anim Sci 86:2531-2538.

Grindek E, Sundvold H, Lien S and Rothschild MF (2000) Physical and genetic mapping of the peroxisome proliferators receptor γ (PPARγ) gene to porcine chromosome 13. J Anim Sci 78:1391-1392.

Grindflek E, Hoen N, Sundvold H, Rothschild MF, Plastow G and Lien S (2004) Investigation of a peroxisome proliferator-activated receptor gamma haplotype effect on meat quality and carcass traits in pigs. Anim Genet 35:238-241.

Lai PH, Wang WL, Ko CY, Lee YC, Yang WM, Shen TW, Chang WC and Wang JM (2008) HDAC1/HDAC3 modulates *PPARG2* transcription through the sumoylated CEBPD in hepatic lipogenesis. Biochim Biophys Acta 1783:1803-1814.

Meirhaeghe A and Amouyel P (2004) Impact of genetic variation of PPARγ in humans. Mol Genet Metab 83:93-102.

Medina-Gomez G, Gray SL, Yetukuri L, Shimomura K, Virtue S, Campbell M, Curtis RK, Jimenez-Linan M, Blount M, Yeo GS, *et al.* (2007) PPAR gamma 2 prevents lipotoxicity by controlling adipose tissue expandability and peripheral lipid metabolism. PLoS Genet 3:e64.

Omi T, Brenig B, Spilar Kramer S, Iwamoto S, Stranzinger G and Neuenschwander S (2005) Identification and characterization of novel peroxisome proliferator-activated receptor-gamma (*PPAR-γ*) transcriptional variants in pig and human. J Anim Breed Genet 122:45-53.

Pasceri V, Wu HD, Willerson JT and Yeh ET (2000) Modulation of vascular inammation *in vitro* and *in vivo* by peroxisome proliferator-activated receptor-gamma activators. Circulation 101:235-238.

Pritchard JK and Przeworski M (2001) Linkage disequilibrium in humans: Models and data. Am J Hum Genet 69:1-14.

Renaudeau D and Mourot J (2007) A comparison of carcass and meat quality characteristics of Creole and Large White pigs slaughtered at 90 kg BW. Meat Sci 76:165-171.

Rosen ED, Sarraf P, Troy AE, Bradwin G, Moore K, Milstone DS, Spiegelman BM and Mortensen RM (1999) PPAR gamma is required for the differentiation of adipose tissue *in vivo* and *in vitro*. Mol Cell 4:611-617.

Rosen ED, Hsu CH, Wang X, Sakai S, Freeman MW, Gonzalez FJ and Spiegelman BM (2002) C/EBPalpha induces adipogenesis through PPARgamma: A unied pathway. Genes Dev 16:22-26.

Samulin J, Berget I, Lien S and Sundvold H (2008) Differential gene expression of fatty acid binding proteins during porcine adipogenesis. Comp Biochem Physiol B Biochem Mol Biol 151:147-152.

Shen LX, Basilion JP and Stanton Jr VP (1999) Single-nucleotide polymorphisms can cause different structural folds of mRNA. Proc Natl Acad Sci USA 96:7871-7876.

Stephens JC, Schneider JA, Tanguay DA, Choi J, Acharya T, Stanley SE, Jiang R, Messer CJ, Chew A, Han JH, *et al.* (2001) Haplotype variation and linkage disequilibrium in 313 human genes. Science 293:489-493.

Tontonoz P, Hu E, Graves RA, Budavari AI and Spiegelman BM (1994a) mPPAR gamma 2: Tissue-specic regulator of an adipocyte enhancer. Genes Dev 8:1224-1234.

Tontonoz P, Hu E and Spiegelman BM (1994b) Stimulation of adipogenesis in fibroblasts by PPAR gamma 2, a lipid-activated transcription factor. Cell 79:1147-1156.

Van der Steen HAM, Prall GFW and Plastow GS (2005) Application of genomics to the pork industry. J Anim Sci 83:E1-E8.

Werman A, Hollenberg A, Solanes G, Bjørbæk C, Vidal-Puig AJ and Flier JS (1997) Ligand-independent activation domain in the N terminus of peroxisome proliferator-activated receptor γ (PPARγ). Differential activity of PPARγ1 and -2 isoforms and inuence of insulin. J Biol Chem 272:20230-20235.

Wierbicki E and Deatherage FE (1958) Water content of meats, determination of water-holding capacity of fresh meats. J Agric Food Chem 6:387-392.

Wu Z, Rosen ED, Brun R, Hauser S, Adelmant G, Troy AE, McKeon C, Darlington GJ and Spiegelman BM (1999) Cross-regulation of C/EBP alpha and PPAR gamma controls the transcriptional pathway of adipogenesis and insulin sensitivity. Mol Cell 3:151-158.

Yu TP, Tuggle CK, Schmitz CB and Rothschild MF (1995) Association of PIT1 polymorphisms with growth and carcass traits in pigs. J Anim Sci 73:1282-1288.

Associate Editor: Alexandre Rodrigues Caetano