Breed difference and regulation of the porcine Sirtuin 1 by insulin

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ABSTRACT: Sirtuin 1 (Sirt1) plays an important role in fat metabolism. In the current study, we examined the breed differences in Sirt1 between Jinhua pigs (a fatty breed of China) and Landrace pigs (a leaner breed). In addition, the effect of insulin on the gene expression of Sirt1 and the major lipase, adipose triglyceride lipase (ATGL), and hormone-sensitive lipase (HSL) in fat metabolism was also studied in vitro. Compared with the Landrace pigs, the BW of Jinhua pigs was less ($P < 0.01$), whereas the body fat content were greater ($P < 0.01$). The protein content and the mRNA abundance of Sirt1 in Jinhua pigs were less ($P < 0.01$) in subcutaneous adipose tissues compared with the Landrace pigs. Likewise, the mRNA abundance of ATGL and HSL were also less ($P < 0.01$) in Jinhua pigs. In vitro, treatment with a different dose of insulin (10, 50 and 100 nM) decreased ($P < 0.01$) glycerol release and the mRNA abundance of Sirt1, ATGL, and HSL in porcine adipocytes. Likewise, treatment with 50 nM insulin for 24 and 48 h also decreased ($P < 0.05$) glycerol release and the expression of Sirt1, ATGL, and HSL in porcine adipocytes. Furthermore, insulin and Sirt1-specific small interfering RNA treatment decreased ($P < 0.01$) the expression of Sirt1, ATGL, and HSL compared with the control or insulin treatment. These results indicate that insulin may regulate transcription of Sirt1, ATGL, and HSL in porcine adipocytes and provide information for manipulating these gene expressions in regulating fat metabolism in pigs.

Key words: adipose triglyceride lipase, hormone sensitive lipase, insulin, pig, Sirtuin 1

INTRODUCTION

Sirtuin 1 (Sirt1), the mammalian homolog of Sir2, is a NAD-dependent deacetylase that plays an important role in a variety of biological processes, including cell proliferation and differentiation (Langley et al., 2002), apoptosis (Kitamura et al., 2005), and metabolism (Rodgers et al., 2005). Sirtuin 1 is important in mammalian energy metabolism by regulating glucose or lipid metabolism (Boily et al., 2008; Liang et al., 2009). The expression and the regulation of Sirt1 by fasting (Rodgers et al., 2005; Rodgers and Puigserver, 2007), calorie restriction (Wolf, 2006; Chen et al., 2008), inhibitors (Araki et al., 2004), and agonists (Howitz et al., 2003) have been studied in humans and mice. In pigs, however, very little is known about the regulation of the Sirt1 gene expression in porcine adipocytes.

Recent studies have shown that Sirt1 also has a positive role in the metabolic pathway through its involvement in insulin signaling (Yang et al., 2006; Liang et al., 2009). It improves insulin sensitivity by silencing the expression of protein tyrosine phosphatase 1B (Sun et al., 2007; Zabolotny and Kim, 2007). However, it is unclear whether insulin can affect transcription of Sirt1 in porcine adipocytes.

Therefore, based on our previous studies (Shan et al., 2008, 2009a), we have examined the differences in porcine Sirt1 between Jinhua pigs (a fatty breed of China) and Landrace pigs (a leaner breed) and studied the effects of insulin alone or in conjunction with Sirt1-specific small interfering RNA (Sirt1-siRNA) on expression of Sirt1 in porcine adipocytes. In addition, because of the important roles of adipose triglyceride lipase (ATGL) and hormone-sensitive lipase (HSL) in fat metabolism, the regulation of ATGL and HSL by insulin in porcine adipocytes was also studied.
MATERIALS AND METHODS

All procedures were approved by the University of Zhejiang Institutional Animal Care and Use Committee.

Animals and Experimental Design

Eight Jinhua male pigs and 8 male Landrace pigs (30 or 180 d of age; 4 pigs per age) were selected randomly and killed to collect samples. Left-half carcasses were weighed after the head, hooves, tail, and viscera (except the kidney) were removed. The omental adipose tissue, subcutaneous adipose tissue (SAT), and peritoneal adipose tissue, and LM in left-half carcasses were collected. The fat deposition was determined by the percent BW according to previously published methods (Shan et al., 2009b). The SAT in right-half carcasses were collected and rapidly frozen in liquid nitrogen, then stored at –80°C until analysis for gene expression was determined by spectrophotometry absorbance measurements at 260 and 280 nm. For each sample, 2 μg of total RNA was used to synthesize the first cDNA according to our previously published procedures (Shan et al., 2008, 2009a).

Cell Culture

The porcine preadipocytes were prepared and counted with a hemocytometer by previously published methods (Shan et al., 2008, 2009a). Briefly, Duroc × Landrace × Yorkshire pigs from 5 to 7 d of age were overdosed with sodium thiopental and exsanguinated. The SAT was removed, and the porcine preadipocytes were prepared and counted (Shan et al., 2008). The cells were seeded on 6-well (35-mm) tissue culture plates (Shanghai Sangon Co. Ltd., Shanghai, China) at a density of 2 × 10^4 cells/cm^2 in complete media, Dulbecco’s modified Eagle medium/HamF12 (DMEM/F12; Invitrogen Life Technologies, Carlsbad, CA) + 10% fetal bovine serum (Invitrogen Life Technologies) + 100 U of penicillin + 100 U of streptomycin. Cells were cultured at 37°C under a humidified atmosphere of 95% air: 5% CO2. Media were changed every 3 d. After 10 d, cultures were washed with DMEM/F12 and then used for experiments to assess the effect of insulin on gene expression of Sirt1, ATGL, and HSL. For insulin regulation experiments, porcine adipocytes were incubated with 0, 10, 50, and 100 nM insulin for 24 h for dose-response experiments, and the optimal concentration of 50 nM of insulin in DMEM/F12 (without fetal bovine serum and phenol red) for 24 and 48 h for time-response experiments.

RNA Interference

In addition, we studied the effect of insulin on the gene expression of Sirt1 and triglyceride lipase after knockdown Sirt1 with the Sirt1-siRNA (Shan et al., 2009c). Cells were plated in 2 mL of growth medium without antibiotics to yield 90% confluent cells at the time of transfection. For Sirt1-siRNA treatment, porcine adipocytes were transfected with Sirt1-siRNA. For insulin and Sirt1-siRNA synergistic regulation, cells were treated with 50 nM insulin in conjunction with transfection Sirt1-siRNA. The control group was transfected with the empty plasmid. Transfections were performed (Lipofectamine 2000, Invitrogen Life Technologies) according to the manufacturer’s protocol. Then, the gene expression was determined by real-time quantitative PCR 48 h after transfection. The glycerol release was determined using a glycerol kit (Applygen Technologies Inc., Beijing, China).

Total RNA Extraction and Reverse Transcription

Total RNA was isolated from the porcine tissues and adipocytes using (Trizol Reagent, Invitrogen Life Technologies) according to the manufacturer’s instructions. The purity and concentration of total RNA were determined using a spectrophotometer absorbance measurements at 260 and 280 nm. For each sample, 2 μg of total RNA was used to synthesize the first cDNA according to our previously published procedures (Shan et al., 2008, 2009a).

Quantitative Real-Time PCR

Quantitative real-time PCR was performed using the real-time PCR system (7500 Fast Real-Time PCR System, Applied Biosystems, Foster City, CA) and a kit (SYBR Premix Ex Taq kit, Takara Biotechnology Co. Ltd., Dalian, China). In this study, 18S rRNA (18S) was used as the reference gene. Oligonucleotide primers (Shanghai Sangon Co. Ltd.) specific for porcine Sirt1, ATGL, HSL, and 18S were based on known sequences deposited in GenBank (Table 1). The PCR system and cycling conditions were the same as those described previously (Shan et al., 2008, 2009a). The 2^ddCt method was used to analyze the relative changes in each gene expression. The measurements were repeated 3 times.

Protein Extraction and Western Blot Analysis

Total protein was isolated from SAT using the protein extraction reagent (Pierce Biotechnology, Rockford, IL). The protein concentration was measured by the method described previously (Bradford, 1976). Proteins in the supernatant were denatured with dithiothreitol for 5 min at 100°C. Proteins were separated by using SDS-PAGE on a 5 to 8% Tris-glycine gel, transferred to a polyvinylidene fluoride membrane (Millipore Corporation, Billerica, MA), and incubated with Sirt1 antibody (sc-19857, 1:400 dilution; Santa Cruz Biotechnology, Santa Cruz, CA) or glyceraldehyde-3-phosphate antibody (sc-48166, 1:400 dilution; Santa Cruz Biotechnology). The secondary antibody (anti-mouse IgG, Santa Cruz Biotechnology) was diluted 2,000-fold. All incubations were performed in Tris-HCl buffer (pH 7.5) with 0.1% Tween 20 and...
5% dry milk. Immunodetection was performed by using enhanced chemiluminescence Western Blotting Substrate (Pierce Biotechnology, Rockford, IL). Signal intensity was determined (Bandscan 4.5, Dulakshi Co., San Ramon, CA), and means were calculated for each breed.

Statistical Analysis

All experimental data are presented as means ± SEM. Comparisons were made by unpaired 2-tailed Student’s t-tests or 1- or 2-way ANOVA, as appropriate. Effects were considered significant at \( P < 0.05 \).

RESULTS

Sirt1 and Triglyceride Lipase Genes mRNA Abundance in Jinhua and Landrace Pigs

At 30 and 180 d, the BW of Jinhua pigs was less (\( P < 0.01 \); Figure 1A) than Landrace pigs, but the body fat content (Figure 1B) of Jinhua pigs was greater (\( P < 0.01 \)) compared with Landrace pigs. At 30 d, the mRNA abundance of Sirt1, ATGL, and HSL in SAT of Jinhua pigs was less (\( P < 0.01 \)) than that in Landrace pigs (Figure 1C). Likewise, at 180 d, the mRNA abundance of Sirt1, ATGL, and HSL (Figure 1D) in SAT of Jinhua pigs were also less (\( P < 0.01 \)) compared with the Landrace pigs.

Sirt1 Protein Abundance in Jinhua and Landrace Pigs

The abundance of Sirt1 protein in SAT at 180 d is presented in Figure 2. Compared with the Landrace pigs, the Sirt1 protein abundance in SAT of Jinhua pigs was less (\( P < 0.01 \)). The greater Sirt1 protein in Landrace pigs was accompanied by an increase in Sirt1 gene expression.

Effect of Insulin on the Gene Expression of Sirt1, ATGL, and HSL

The results showed that insulin treatment decreased (\( P < 0.01 \)) the glycerol release and the expression of Sirt1 in porcine adipocytes (Figures 3 and 4). Compared with the control (0 nM), 10, 50, and 100 nM insulin treatments decreased (\( P < 0.01 \)) the mRNA abundance of Sirt1 by 32.6, 35.1, and 43.9% (Figure 3B), respectively. Likewise, treatments with 10, 50, and 100 nM insulin decreased (\( P < 0.01 \)) the mRNA abundance of ATGL by 23.2, 29.4, and 33.0% (Figure 3C), and the mRNA abundance of HSL by 15.9, 29.3, and 22.7%, respectively (Figure 3D).

Furthermore, treatment with 50 nM insulin for 24 and 48 h also decreased (\( P < 0.01 \)) the mRNA abundance of Sirt1 by 25.4 and 27.3%, respectively (Figure 4B). Likewise, treatment with 50 nM insulin for 24 and 48 h also decreased (\( P < 0.01 \)) the gene expression of ATGL (Figure 4C) and HSL (Figure 4D) in porcine adipocytes.

The Synergistic Regulation of Insulin and Sirt1-siRNA

Compared with the control, transfection of the porcine adipocytes with Sirt1-siRNA decreased (\( P < 0.01 \)) the mRNA abundance of Sirt1 (Figure 5A), ATGL (Figure 5B), and HSL (Figure 5C). Treatment of the porcine adipocytes with insulin decreased the expression of Sirt1 (\( P < 0.05 \); Figure 5D), ATGL (\( P < 0.01 \); Figure 5E), and HSL (\( P < 0.01 \); Figure 5F). Likewise, compared with the controls, insulin + Sirt1-siRNA treatment decreased (\( P < 0.01 \)) the expression of Sirt1 and ATGL by 80.77 and 77.48%, respectively.

Table 1. Specific primers used for real-time PCR verification of the porcine Sirtuin 1 (Sirt1), adipose triglyceride lipase (ATGL), hormone-sensitive lipase (HSL), and 18S rRNA genes

<table>
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<tr>
<th>Gene</th>
<th>Accession number</th>
<th>Primer source</th>
<th>Oligonucleotide sequence</th>
<th>Product length, bp</th>
<th>Reference</th>
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<td>EU030283</td>
<td>Pig</td>
<td>5′- TGGGGTTTCTGTTCCTTGTG - 3′ (sense primer)</td>
<td>98</td>
<td>Shan et al., 2008</td>
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<td></td>
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<td></td>
<td>5′- CTTGAGGATCAGGAGGTC - 3′ (antisense primer)</td>
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<td>ATGL</td>
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<td>Pig</td>
<td>5′- TCACCAACACCACATCCA - 3′ (sense primer)</td>
<td>95</td>
<td>Shan et al., 2009a</td>
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<td>5′- GCCACTCTCTGAGCAACCA - 3′ (antisense primer)</td>
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<td>HSL</td>
<td>AY686758</td>
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<td>Pig</td>
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<td>122</td>
<td>Shan et al., 2009b</td>
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<td></td>
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<td></td>
<td>5′- TTGACGGAAGGACCACCA - 3′ (antisense primer)</td>
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</table>

1From the database of the National Center for Biotechnology Information (Bethesda, MD).
Figure 1. Effect of breed on the BW (A), fat percent (B), and gene expression in subcutaneous adipose tissue (C, D). Gene expression was determined by real-time PCR and was normalized to 18S rRNA and expressed relative to expression in Jinhua pigs. Each column represents the mean of 4 individual pigs ± SEM. **P < 0.01. Sirt1 = Sirtuin 1; ATGL = adipose triglyceride lipase; HSL = hormone-sensitive lipase.

Figure 2. The relative abundance of Sirtuin 1 (Sirt1) protein in subcutaneous adipose tissue. The Sirt1 protein in subcutaneous adipose tissue was examined by Western blotting (A), and the signal intensity was determined (B). Proteins were separated by using SDS-PAGE, transferred to a polyvinylidene fluoride membrane, and incubated with Sirt1 antibody or glyceraldehyde-3-phosphate (GAPDH) antibody, and the secondary antibody (anti-mouse IgG). Immunodetection was performed by enhanced chemiluminescence Western blotting substrate. Signal intensity was determined (Bandscan 4.5, Dulakshi Co., San Ramon, CA), and means were calculated for each breed. Each column represents the mean of 4 individual pigs ± SEM. **P < 0.01.
Figure 3. Effect of different doses of insulin on glycerol release (A) and gene expression of Sirtuin 1 (Sirt1; B), adipose triglyceride lipase (ATGL; C), and hormone-sensitive lipase (HSL; D) in porcine adipocytes. After treatment for 24 h, the glycerol release and gene expression were assessed. Glycerol release was normalized to total cellular protein and expressed relative to the control group (0 nM). Gene expression was determined by real-time quantitative PCR and was normalized to 18S rRNA and expressed relative to the gene expression in the control. Each point represents the means of 3 replicates ± SEM. **P < 0.01.

Figure 4. Time course for glycerol release (A) and the gene expression of Sirtuin 1 (Sirt1; B), adipose triglyceride lipase (ATGL; C), and hormone-sensitive lipase (HSL; D) were generated by incubating porcine adipocytes with 50 nM of insulin for the time indicated. Glycerol release was normalized to total cellular protein and expressed relative to the control group (0 h). Gene expression was determined by real-time quantitative PCR and was normalized to 18S rRNA and expressed relative to gene expression in the control group (0 h). Each point represents the means of 3 replicates ± SEM. *P < 0.05; **P < 0.01.
DISCUSSION

Sirtuin 1, the most intensely studied sirtuin family member, is involved in adipogenesis and seems to regulate lipid metabolism in animals (Picard et al., 2004; Picard and Guarente, 2005). It is an important regulator of energy metabolism through its impact on glucose or lipid metabolism in animals (Boily et al., 2008; Liang et al., 2009). The Sirt1 genotype correlates with visceral obesity traits in an obese individual, and genetic variation in Sirt1 increases the risk for obesity (Peeters et al., 2008). Obese humans have less Sirt1 expression in SAT compared with lean humans (Pedersen et al., 2008). In a model of diabetic nephropathy, Sirt1 was shown to be decreased in the diabetic rat kidney (Tikoo et al., 2007). In the current study, we found that the protein content and mRNA abundance of Sirt1 in SAT of fatty pigs (Jinhua pigs) were less compared with the lean pigs (Landrace pigs). This is consistent with previous studies in humans (Pedersen et al., 2008).

Figure 5. Insulin and Sirtuin 1 (Sirt1)-specific small interfering RNA (Sirt1-siRNA) synergistic regulation of the gene expression of Sirt1, adipose triglyceride lipase (ATGL), and hormone-sensitive lipase (HSL). Small interfering RNA-mediated knockdown of Sirt1 was achieved by transfecting porcine adipocytes with Sirt1-siRNA and further Sirt1-siRNA in conjunction with 50 nM insulin. The control group was transfected with the empty plasmid (Lipofectamine 2000, Invitrogen Life Technologies, Carlsbad, CA). The mRNA expression of Sirt1 (A, D), ATGL (B, E), and HSL (C, F) was determined 48 h after transfection. Gene expression was determined by real-time quantitative PCR and was normalized to 18S rRNA and expressed relative to gene expression in the control. Each column represents the means of 3 replicates ± SEM. *P < 0.05; **P < 0.01.
The Jinhua pig is a type of fatty pig from China. Jinhua pigs are noted for their scarlet muscle, greater intramuscular fat contents, and meat quality. The Landrace pig is a lean breed noted for its lean carcasses. In this study, we found that the BW of Jinhua pigs were less, whereas body fat content was greater compared with Landrace pigs. The current result is consistent with previous findings (Shan et al., 2009b) that the BW of Jinhua pigs were decreased, whereas body fat content was increased compared with Landrace pigs. Furthermore, mRNA abundance of Sirt1, ATGL, and HSL in SAT were associated with the fat percentage (data not shown). This result could provide some information needed for understanding the molecular mechanism underlying the variation between the Jinhua pigs and Landrace pigs.

Adipose tissue is an important source of energy and plays an important role in whole-body energy homeostasis. Dysregulation of lipolysis has been involved in the development of insulin resistance in obese, type 2 diabetes, and related disorders. In adipose tissue, lipolysis is affected by physiological conditions, hormones, and cytokines, such as insulin, IL-6, and adiponectin (Duncan et al., 2007). Insulin, a major regulator of lipolysis, has a vital role in regulating glucose uptake, lipid metabolism, preadipocyte differentiation, and lipogenesis (Boden, 2003). Sirtuin 1 is involved in adipogenesis and could promote fat mobilization in white adipocytes (Picard et al., 2004; Picard and Guarente, 2005). In the mouse and human, Sirt1 gene expression is regulated by nutritional status (Rodgers and Puigserver, 2007; Chen et al., 2008) and by other factors such as nicotinamide (Araki et al., 2004), resveratrol, fisetin, and bultei (Howitz et al., 2003). Furthermore, caloric restriction and fasting increased Sirt1 abundance in tissues such as muscle, brain, liver, and fat (Ramsey et al., 2008). In the current study, we found that insulin decreased the expression of Sirt1 in porcine adipocytes. In addition, the mRNA abundance of ATGL and HSL in porcine adipocytes were also decreased. This regulation was further demonstrated when siRNA against Sirt1 was used in conjunction with insulin. Taken together, these findings indicated that insulin could decrease the mRNA abundance of Sirt1, ATGL, and HSL in porcine adipocytes.

Obesity is associated with insulin resistance and insulin resistance involves decreased glucose transport activity in adipocytes. Sirtuin 1 can regulate insulin secretion, decrease plasma glucose concentrations, and improve insulin sensitivity (Bordone et al., 2006; Sun et al., 2007). Sirtuin 1 directly regulates the insulin action through its actions to control protein tyrosine phosphatase 1B gene expression (Sun et al., 2007). However, information is limited regarding whether insulin could feedback on the expression of Sirt1 in adipocytes. It has been shown that insulin decreases endogenous Sirt1 expression (Cohen et al., 2004). During fasting or caloric restriction, the insulin signaling is decreased (Al-Regaiey et al., 2005). Likewise, Sirt1 abundance in several metabolic tissues including the liver, muscle, and brain are increased during caloric restriction (Nisoli et al., 2005; Civitarese et al., 2007; Rodgers et al., 2008). Based on the previous studies and the results of the current study, it was concluded that insulin decreases expression of Sirt1 in adipocytes. However, the precise mode of insulin action on Sirt1 gene expression needs further study.

Insulin-signaling pathway not only regulates glucose, protein, and lipid metabolism, but also the aging process (Das, 2005). Adipose triglyceride lipase and HSL are major novel triglyceride lipases and coordinate the catabolism of stored triglycerides in adipose tissues of mammals. The expression ATGL and HSL is regulated by fasting, re-feeding, and other factors such as hormones. In mice, the expression of ATGL is upregulated by fasting and downregulated by refeeding (Kershaw et al., 2006). In murine adipocytes, the expression of ATGL genes was downregulated by isoproterenol (Kralisch et al., 2005), tumor necrosis factor α (TNFα; Kralisch et al., 2005; Kim et al., 2006), insulin (Kralisch et al., 2005; Kershaw et al., 2006), and rosiglitazone (Liu et al., 2009). The expression of HSL was also decreased by isoproterenol, TNFα, and insulin (Kralisch et al., 2005). In pigs, previous studies have shown that insulin, forskolin, and TNFα decreased porcine ATGL gene expression (Deiuliis et al., 2008; Shan et al., 2009b), whereas resveratrol treatment increased mRNA abundance of porcine ATGL in porcine adipocytes (Shan et al., 2008). In this study, we also found that the expression of ATGL and HSL was decreased by insulin in porcine adipocytes.

It is well known that lipolysis is regulated by nutritional status, hormones, and other effectors. Hormone sensitive lipase is a major effector of lipolysis. The regulation of HSL by insulin is mediated mainly via cyclic adenosine monophosphate (cAMP), and subsequently cAMP activates protein kinase A, which activates HSL by promoting its phosphorylation (Holm, 2003; Carmen and Víctor, 2006). Adipose triglyceride lipase is a recently discovered triglyceride lipase in animals. Insulin decreases the expression of ATGL, but the precise molecular mechanism of the regulation of ATGL by insulin is unclear. Unlike HSL, ATGL is not phosphorylated by protein kinase A (Zimmermann et al., 2004). Although it has been shown that the expression of ATGL is regulated by PPARγ (Festuccia et al., 2006; Kim et al., 2006; Liu et al., 2009), it is unclear whether PPARγ is responsible for the regulation of ATGL by insulin. Other studies showed that Sirt1 may regulate the mRNA abundance of porcine ATGL in porcine adipocytes (Shan et al., 2008). Recently, it has been shown that ATGL has 2 FoxO1-binding sites, and insulin may inhibit expression of ATGL in adipocytes by restraining the nuclear localization of FoxO1 (Chakrabarti and Kandror, 2009). However, more studies are needed to further evaluate the regulation of ATGL in animals.

In conclusion, we have demonstrated the differences in the expression of Sirt1, ATGL, and HSL in fatty pigs
(Jinhua pigs) and lean pigs (Landrace pigs). Insulin can regulate the lipolysis and transcription of Sirt1, ATGL, and HSL in porcine adipocytes. These results provided some information on the role of these genes in lipid metabolism and may be useful for regulating fat deposition and meat quality in pigs.

LITERATURE CITED


Shan, T., T. Wu, Y. Reng, and Y. Wang. 2009b. Breed difference and regulation of the porcine adipose triglyceride lipase and