

Advances in Animal Fatty Acid Transmembrane Transport Proteins FATP1 and FATP4

Hengyong Xu, Yan Wang and Jiwen Wang
College of Animal Science and Technology, Sichuan Agricultural University,
625014 Ya'an, Sichuan, P.R. China

Abstract: Fat is the main composition of the body, it not only has an important role to maintain the animal's physiological function but also its precipitation and sedimentary parts have become the key factor to influence the carcass and meat quality. As two members of the family of the fatty acid transport proteins, FATP1 and FATP4 involved in the transport of long chain fatty acids across the plasma membrane and the metabolism of fatty acids. They are the key genes which have an influence on fat content and maintain skin phospholipids metabolism. So, in this study, the characteristic of structure, function and variation of the gene *FATP1* and *FATP4* have been summarized. Researchers hope this content can provide certain reference for the researchers in the future.

Key words: FATP1, FATP4, function, polymorphism, plasma membrane, China

INTRODUCTION

Long-Chain Fatty Acids (LCFAs) are not only extremely important in the human diet but they also play an important role in the pathogenesis of fluid accumulation, the production and storage of metabolic energy, the synthesis of the cell membrane and the anchoring functions of proteins. A number of proteins have found to be possibly involved in the uptake of LCFAs. These include Fatty Acid Translocase (FAT/CD36), Fatty Acid Binding Protein (FABP), long-chain Acyl-Coenzyme A Synthetase (ACSL), Acyl-CoA Binding Protein (ACBP) and Fatty Acid Transport Protein (FATP) (Coburn *et al.*, 2001; Chiu *et al.*, 2001; Glatz *et al.*, 2002; Stahl, 2004). Despite the fact that all these proteins have some role in the uptake and metabolism of LCFAs, recent studies have found that FATPs play any important role in the uptake and metabolism of fatty acids or in the maintenance of epidermal phospholipid metabolism and composition. In this project, researchers performed a comprehensive overview of two main members of the FATPs family: FATP1 and FATP4. This study provides information that may promote in-depth understanding of the characteristics and mechanisms of FATP1 and FATP4 and the development of strategies for treating fat metabolism related diseases.

AN OVERVIEW OF THE FATPS FAMILY

The FATP family also known as Solute carrier family 27 (Slc27) includes six different members, each with

different expression patterns in different tissues though, most of them affect fatty acid. FATP1, a major family member of FATP in the adipose tissue (Schaffer and Lodish, 1994) is expressed in the heart and skeletal muscle (Schaffer and Lodish, 1994; Binnert *et al.*, 2000; Pohl *et al.*, 2000). Stahl *et al.* (2002) found that insulin could induce mouse FATP1 to move from the cell kernel cavity to the plasma membrane and the degree of insulin-induced FATP1 translocation was consistent with increases of LCFAs uptake. This indicated that hormone-regulated FATP1 activity may play a very important role the maintenance of a stable energy state within the internal environment. FATP4 which is closely related to FATP1 is highly expressed in the small intestine, adipose tissue, skin, heart and liver (Stahl *et al.*, 1999, 2001; Herrmann *et al.*, 2001, 2003). It is also the only member of the FATP family that is expressed in the small intestine. By analysis of the effects of FATP4 overexpression in cells and of the isolated intestinal epithelial cells, it has been found that FATP4 plays an important role in the absorption of LCFAs by the intestinal epithelial cells. FATP2, FATP3, FATP5 and FATP6 are more distantly related to FATP1 and show little or no expression in fat tissue and muscle. Briefly, FATP2 is highly expressed in the liver and kidneys (Hirsch *et al.*, 1998). FATP3 is expressed in the liver, pancreas and lungs (Stahl *et al.*, 2001). FATP5 is expressed only in the liver (Hirsch *et al.*, 1998). FATP6 is mainly expressed in the heart (Gimeno *et al.*, 2003a, b).

FATP LOCATION AND STRUCTURE

FATP1 gene location and structure: FATP is an integral membrane protein. Analysis of the membrane topology of FATP1 has shown that a short N-terminal fragment is located outside of the cell membrane bilayer and the C-terminus is located in the cell cytoplasm. All members of the FATP family have an AMP-binding protein sequence located within the first 300 amino acids of the FATP signal sequence. Researchers initially analyzed the hydrophobicity of amino acid sequence of the *FATP* gene using the Kyte-Doolittle Method and proposed that this gene might have six transmembrane regions (Lewis *et al.*, 2001). In 2000, the human *FATP1* gene was cloned. It is located on Chromosome 19 and it consists of 12 exons and 11 introns. It contains six hydrophobic transmembrane domains and three potential glycosylation sites (Martin *et al.*, 2000). Song *et al.* (2008) successfully cloned the fowl *FATP1* gene which has an open reading frame of 1941 bp encoding 646 amino acids. Currently, most of the mammalian *FATP1* genes have been cloned. Amino acid sequence analysis has revealed >87% of homology between mammalian FATPs but only 71-74% homology between poultry and mammalian FATPs.

FATP4 location and structure: FATP4 is also a member of the FATP family. It is closely related to FATP1. It was not investigated in depth until it was found to be involved in the fatty acid uptake and transport and to play an important role in lipid metabolism. Thomas *et al.* (2001) cloned the mouse *FATP4* gene which is located on Chromosome 2 using an open reading frame of 1929 bp, encoding 643 amino acids. The human *FATP4* gene contains 12 exons, located on Chromosome 9, encoding a protein of 71 kDa in molecular weight (Herrmann *et al.*, 2001). The protein contains a conserved AMP-binding protein sequence which is common to all FATPs family members.

The mRNA sequences of many animal *FATP4* genes have been reported, most of them in mammals including humans, cattle, rattus and brown rats. Sequence alignment analyses have shown >85% of sequence homology between mammals. However, the mRNA and protein sequences of the non-mammalian *FATP4* genes from such animals as fish and poultry have not yet been recorded in public gene databases.

MECHANISMS OF FATPS

The mechanisms underlying the actions of FATP-family proteins in the uptake of LCFAs remain unknown. There is also, a question of whether FATP1 plays the role

of transporter or promotes the accumulation of fatty acids in cells by preventing the release of LCFAs. Four controversial models of the mechanisms of FATP have been proposed (Doege and Stahl, 2005). The first model assumes that FATPs are the only transmembrane transport proteins that indirectly regulate the absorption of LCFAs and that they are associated with other proteins such as ACSLs (Richards *et al.*, 2006). The second model assumes that the FATPs themselves are the long-chain, membrane-bound proteins and act as long-chain acyl-coenzyme A synthetases causing LCFAs to become involved in cell manipulation and diffuse through the cell membrane. The third model assumes that FATPs act either through a transport role, through an acyl-coenzyme A role or through a combination of the two. The fourth model assumes that FATPs are multifunctional proteins that can indirectly regulate the absorption of LCFAs rather than rely on their esterification role. The results of Schaffer and Lodish (1994) support the first model. This study confirmed that FATP1 forms ACSL heterodimers in adipose cells (Richards *et al.*, 2006). Schaffer and Lodish (1994) deem that the intake of LCFAs is required for all of the proteins. This model is consistent with the results obtained from a study of the uptake of fatty acids in an *E. coli* system using a combination of the transport protein FadL, Fad and acyl coenzyme A synthetase (DiRusso and Black, 1999; DiRusso *et al.*, 2005). However, supporters of the second model deem that the purified FATPs have ACSL activity. FATP-mediated absorption of LCFA may be driven by increasing the transformation of the fatty acids located within the bilayer leaflets to fatty acyl coenzyme A. Yeast expression experiments have confirmed the third model. Specific mutations in the *FATP* gene can identify the input of fatty acids in acyl-coenzyme A synthetase activity indicating that this protein has two independent functions in yeast. Because these existing mechanisms are still controversial, further investigation into the structure and function of FATP is required before a more accurate transport mechanism model can be proposed.

Role of FATP1: To date, many studies have shown that FATP1 plays an important role in the transport and metabolism of fatty acids *in vivo*. Over expression of FATP1 in the cardiocytes of transgenic mice can increase the uptake of myocardial fatty acids and the accumulation of free fatty acids but cannot change the triglyceride level. This causes lipotoxicity cardiomyopathy by increasing the oxidation of palmitic acid (Kim *et al.*, 2004; Chiu *et al.*, 2005). Over expression of FATP1 in skeletal muscle can increase the transport of LCFAs and induce the oxidation of these lipids but it does not affect the accumulation of

intramuscular fat (Holloway *et al.*, 2011). Wu *et al.* (2006a) found that insulin cannot effectively stimulate the adipocytes lacking FATP1 to absorb fatty acids. However, the absorption of basal LCFAs remained unaffected by the absence of FATP1. Schaffer and Lodish (1994) reported that the mouse 3T3-L1 fat precursor cells stably transfected with the *FATP1* gene could transport oleic acid 4-5 times faster than the control group. In a study of the FATP1 protein function Lobo *et al.* (2007) found that knocking out the *FATP1* gene could significantly reduce the deposition of the triglyceride in adipocytes. Garcia-Martinez *et al.* (2005) found that overexpression of FATP1 in the sarcolemma of human skeletal muscle could promote fatty acid uptake and storage but would not be oxidized. In addition, FATP1 has been shown to be involved in the process of hormone-regulated fatty acid uptake and response to insulin in adipose cells and original skeletal muscle cells by changing the plasma and other components (Stahl *et al.*, 2002; Wu *et al.*, 2006b).

FATP1 has specific selectivity in the transport of LCFAs. Shaffer showed that FATP1 mainly mediated the transmembrane transport of fatty acids whose backbone lengths are >10 carbon atoms such as myristic acid (C14:0), palmitic acid (C16:0) and oleic acid (C18:0) but had no effect on the transmembrane transport of short-chain fatty acids (Schaffer and Lodish, 1994).

FATP1 also, affects energy metabolism. Wu *et al.* (2006a, b) found that FATP1 played a role in nonshivering thermogenesis in adipose tissue. In 2009, Mitchell and Guitart independently found that FATP1 could regulate the re-synthesis of cardiolipin and increase pyruvate dehydrogenase activity (Guitart *et al.*, 2009; Mitchell and Hatch, 2009). Recently, Sebastian *et al.* (2009) found that the use of adenovirus to overexpress FATP1 in the L6E9 sarcolemma could increase the oxidation of fatty acids and the esterification of palmitate in triglycerides indicating that FATP1 might be associated with mitochondria and fatty acid oxidation therein (Mitchell and Hatch, 2009).

Role of FATP4: FATP4 (SLC27A4) is the only member of the FATP family that is expressed in the small intestine, mainly in the brush edge of intestinal epithelial cells (Stahl *et al.*, 1999). It has a very important role in the transmembrane transport of LCFAs. Because FATP4 is involved in dietary lipid intake, it may be a suitable anti-obesity target.

In addition, FATP4 plays an important role in the maintenance of the epidermal phospholipid component, phospholipid metabolism and maintenance of normal skin function Moulson *et al.* (2003) and Herrmann *et al.* (2003)

found that newborn mice with homozygous deletions of FATP4 had similar phenotypes, specifically tight, thick skin, broken skin barriers, facial deformities and dyspnea but their ability to absorb LCFAs was unaffected (Herrmann *et al.*, 2003; Moulson *et al.*, 2003). Gimeno *et al.* (2003a) found that the deletion of exons 1 and 2 in the mouse *FATP4* gene would result in embryo lethality within the first 9.5 days of pregnancy (Gimeno *et al.*, 2003b). The deletion of exon 3 in the mouse *FATP4* gene however, resulted in hyperkeratotic skin and damage to epidermal barrier function (Jia *et al.*, 2007). However, increased expression of FATP1 in the dermis and FATP6 and CD36 in the epidermis was found to promote the repair of the permeability barrier.

POLYMORPHISMS OF FATPS

Association of the FATP1 and FATP4 polymorphisms with human obesity and related conditions: The polymorphisms of FATP1 are mainly associated with dyslipidemia. Meirhaeghe *et al.* (2000) identified three Single Nucleotide Polymorphisms (SNPs) in introns 8 and 9 in 1144 French individuals. This was the first study to indicate an association between *FATP* gene polymorphisms and changes in the internal human lipid internal environment. These results suggested that FATP1 polymorphisms might affect lipid metabolism. Gertow *et al.* (2003) analyzed the same A/G polymorphism in intron 8 in 856 Swedish individuals. In contrast to the results of the Meirhaeghe study, no significant association was found between FATP1 polymorphism and fasting plasma Triglyceride (TG) content. However, the associations between FATP1 polymorphism and postprandial cholesterol and the distribution of particle sizes of low-density lipoproteins were found to be significant.

FATP4 is also associated with human pathophysiology. Bower found that in obese African American and Caucasian women increased mRNA and protein levels of FATP4 in adipose cells were associated with a higher LCFA absorption. Gertow *et al.* (2004) investigated 17 pairs of monozygotic twins with different Body Mass Indexes (BMI) and different levels of obesity and insulin resistance and found that there was more FATP4 mRNA expression in patients with more pronounced obesity and that this was not associated with genetic factors. Gertow found that the polymorphism in exon 3 of FATP4 resulted in an amino acid exchange within the FATP4 protein and caused insulin resistance syndrome. These findings suggest that these allele carriers have lower metabolic parameters including BMI, TG insulin levels and systolic blood pressure than carriers with homozygous normal G/G alleles.

Association between FATP1 polymorphisms and economically relevant traits in livestock and poultry:

There are only a limited number of studies on *FATP1* gene polymorphisms in livestock and poultry. Using DNA sequencing and Restriction Fragment Length Polymorphism (RFLP), Hua *et al.* (2011) identified two mutations (g.586T>C and g.601A>G) in the pig *FATP1* gene. The g.586T>C genotype was found to be significantly associated with shoulder and back fat thickness, average back fat thickness and leaf fat weight indicating that this mutation might affect pig fatty deposition. Ordovas *et al.* (2008) identified 14 SNPs in Holsteins using minisequencing. However, these SNPs did not significantly affect the level of cow milk fat, suggesting that FATP1 has little or nothing to do with milk fat content. Lv *et al.* (2010) reported that the C allele of T112C in the Holstein *FATP1* gene had a positive effect on milk production, suggesting that FATP1 may affect the milk yield of the Chinese Holstein. Using the PCR-SSCP Method, Wang *et al.* (2010) identified five mutations in the chicken *FATP1* gene. These mutations were significantly associated with some carcass traits and abdominal fat weight indicating that the *FATP1* gene has some impact on abdominal fatty deposition in chickens.

CONCLUSION

FATP1 and FAPT4 are two important members of the FATPs family, each of which has specific, important biological functions. FATP1 is mainly involved in fatty acid and energy metabolism. It also has an important role in the treatment of fatty deposition, obesity and Type II diabetes. FATP4 mediates keratinocyte uptake of LCFAs and provides the keratinocytes with raw materials for synthesis of phospholipids. FATP4 also has an important role in the treatment of skin diseases. Although, preliminary studies have been performed for some members of the FATP family, there are only a few reports on the effects of FATP1 and FATP4 on the quality of animal meat and on fat and skin diseases. This is why it is important to study the associations between genetic variations of FATP1 and meat quality and fatty deposition and the role of FATP4 in the skin under normal physiological and pathological conditions.

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REFERENCES

- Binnert, C., H.A. Koistinen, G. Martin, F. Andreelli and P. Beetling *et al.*, 2000. Fatty acid transport protein-1 mRNA expression in skeletal muscle and in adipose tissue in humans. *Am. J. Physiol. Endocrinol. Metabol.*, 279: 1072-1079.
- Chiu, H.C., A. Kovacs, D.A. Ford, F.F. Hsu and R. Garcia *et al.*, 2001. A novel mouse model of lipotoxic cardiomyopathy. *J. Clin. Invest.*, 107: 813-822.
- Chiu, H.C., A. Kovacs, R.M. Blanton, X.L. Han and M. Courtois *et al.*, 2005. Transgenic expression of fatty acid transport protein 1 in the heart cause lipotoxic cardiomyopathy. *Circulation Res.*, 96: 225-233.
- Coburn, C.T., T. Hajri, A. Ibrahim, N.A. Abumrad, 2001. Role of CD36 in membrane transport and utilization of long-chain fatty acids by different tissues. *J. Mol. Neurosci.*, 16: 117-121.
- DiRusso, C. and P. Black, 1999. Long-chain fatty acid transport in bacteria and yeast. Paradigms for defining the mechanism underlying this protein-mediated process. *Mol. Cell. Biochem.*, 192: 41-52.
- DiRusso, C.C., H. Li, D. Darwis, P.A. Watkins, J. Berger and P.N. Black, 2005. Comparative biochemical studies of the murine fatty acid transport proteins (FATP) expressed in yeast. *J. Biol. Chem.*, 280: 16829-16837.
- Doerge, H. and A. Stahl, 2005. Protein-mediated fatty acid uptake: Novel insights from *in vivo* model. *Physiology*, 21: 259-268.
- Garcia-Martinez, C., M. Marotta, R. Moore-Carrasco, M. Guitart and M. Camps *et al.*, 2005. Impact on fatty acid metabolism and differential localization of FATP1 and FAT/CD36 proteins delivered in cultured human muscle cells. *Am. J. Physiol. Cell. Physiol.*, 288: C1264-C1272.
- Gertow, K., C. Skoglund-Andersson, P. Eriksson, S. Boquist and K. Orth-Gomer *et al.*, 2003. A common polymorphism in the fatty acid transport protein-1 gene associated with elevated post-prandial lipaemia and alterations in LDL particle size distribution. *Atherosclerosis*, 167: 265-273.
- Gertow, K., K.H. Pietilainen, H. Yki-Jarvinen, J. Kaprio and A. Rissanen *et al.*, 2004. Expression of fatty-acid-handling proteins in human adipose tissue in relation to obesity and insulin resistance. *Diabetologia*, 47: 1118-1125.
- Gimeno, R.E., A.M. Ortegon, S. Patel, S. Punreddy and P. Ge *et al.*, 2003a. Characterization of a heart-specific fatty acid transport protein. *J. Biol. Chem.*, 278: 16039-16044.

- Gimeno, R.E., D.J. Hirsch, S. Punreddy, Y. Sun and A.M. Ortegon *et al.*, 2003b. Targeted deletion of fatty acid transport protein-4 results in early embryonic lethality. *J. Biol. Chem.*, 278: 49512-49516.
- Glatz, J.F., J.J. Luiken, M. van Bilsen and G.J. van der Vusse, 2002. Cellular lipid binding proteins as facilitators and regulators of lipid metabolism. *Mol. Cell. Biochem.*, 239: 3-7.
- Guitart, M., A.L. Andreu, E. Garcia-Arumi, P. Briones and E. Quintana *et al.*, 2009. FATP1 localizes to mitochondria and enhances pyruvate dehydrogenase activity in skeletal myotubes. *Mitochondrion*, 9: 266-272.
- Herrmann, T., F. Buchkremer, I. Gosch, A.M. Hall and D.A. Bernlohr *et al.*, 2001. Mouse fatty acid transport protein 4 (FATP4): Characterization of the gene and functional assessment as a very long chain acyl-CoA synthetase. *Gene*, 270: 31-40.
- Herrmann, T., F. van der Hoeven, H.J. Gro ne, A.F. Stewart and L. Langbein *et al.*, 2003. Mice with targeted disruption of the fatty acid transport protein 4 (Fatp 4, Slc27a4) gene show features of lethal restrictive dermatopathy. *J. Cell Biol.*, 161: 1105-1115.
- Hirsch, D., A. Stahl and H.F. Lodish, 1998. A family of fatty acid transporters conserved from mycobacterium to man. *Proc. Natl Acad. Sci. USA.*, 95: 8625-8629.
- Holloway, G.P., C.J. Chou, J. Lally, T. Stellingwerff and A.C. Maher *et al.*, 2011. Increasing skeletal muscle fatty acid transport protein 1 (FATP1) targets fatty acids to oxidation and does not predispose mice to diet-induced insulin resistance. *Diabetologia*, 54: 1457-1467.
- Hua, X.C., L.F. Zhang, Z.W. Cai, X.L. Jiang, N.Y. Xu and J.Z. Zhang, 2011. Polymorphism in 5' regulatory region of porcine FATP1 gene and its association with fat traits. *Jiangsu J. Agric. Sci.*, 27: 89-93.
- Jia, Z., C.L. Moulson, Z.T. Pei, J.F. Miner and P.A. Watkins *et al.*, 2007. FATP4 is the principal very long chain fatty acyl-CoA synthetase in skin fibroblasts. *J. Biol. Chem.*, 282: 20573-20583.
- Kim, J.K., R.E. Gimeno, T. Higashimori, H.J. Kim and H. Choi *et al.*, 2004. Inactivation of fatty acid transport protein 1 prevents fat-induced insulin resistance in skeletal muscle. *J. Clin. Invest.*, 113: 756-763.
- Lewis, S.E., L.L. Listenberger, D.S. Ory and J.E. Schaffer, 2001. Membrane topology of the murine fatty acid transport protein. *J. Biol. Chem.*, 276: 37042-37050.
- Lobo, S., B.M. Wiczer, A.J. Smith, A.M. Hall and D.A. Bernlohr, 2007. Fatty acid metabolism in adipocytes: Functional analysis of fatty acid transport proteins 1 and 4. *J. Lipid Res.*, 48: 609-620.
- Lv, Y.F., C.H. Wei, L. Zhang, G.B. Lu and K.D. Liu *et al.*, 2010. Identification of SNPs in Bovine SLC27A1 gene and its effects on milk production traits in Chinese Holstein cattle, 38: 12471-12475.
- Martin, G., M. Nemoto, L. Gelman, S. Geffroy and J. Najib *et al.*, 2000. The human fatty acid transport protein-1 (SLC27A1; FATP-1) cDNA and gene: Organization, chromosomal localization and expression. *Genomics*, 66: 296-304.
- Meirhaeghe, A., G. Martin, M. Nemoto, S. Deeb and D. Cattel *et al.*, 2000. Intronic polymorphism in the fatty acid transport protein 1 gene is associated with increased plasma triglyceride levels in a French population. *Arterioscler Thromb. Vasc. Biol.*, 20: 1330-1334.
- Mitchell, R.W. and G.M. Hatch, 2009. Regulation of cardiolipin biosynthesis by fatty acid transport protein 1 In HEK 293 cells. *Biochim. Biophys. Acta (BBA)-Biomembr.*, 1788: 2015-2021.
- Moulson, C.L., D.R. Martin, J.J. Lugas, J.E. Schaffer and A.C. Lind *et al.*, 2003. Cloning of wrinkle-free, a previously uncharacterized mouse mutation reveals crucial roles for fatty acid transport protein 4 in skin and hair development. *Proc. Natl. Acad. Sci. USA.*, 100: 5274-5279.
- Ordovas, L., P. Zaragoza and J. Altarriba, 2008. Identification of 14 new single polymorphisms in the bovine SLC27A1 gene and evaluation of their association with milk fat content. *J. Dairy Res.*, 75: 129-134.
- Pohl, J., B.A. Fitscher, A. Ring, R. Ihl-Vahl, R.H. Strasser and W. Stremmel, 2000. Fatty acid transporters in plasma membranes of cardiomyocytes in patients with dilated cardiomyopathy. *Eur. J. Med. Res.*, 5: 438-442.
- Richards, M.R., J.D. Harp, D.S. Ory, J.E. Schaffer, 2006. Fatty acid transport protein 1 and long-chain acyl coenzyme A synthetase 1 interact in adipocytes. *J. Lipid Res.*, 47: 665-672.
- Schaffer, J.E. and H.F. Lodish, 1994. Expression cloning and characterization of a novel adipocyte long chain fatty acid transport protein. *Cell*, 79: 427-436.
- Sebastian, D., M. Guitart, C. Garcia-Martinez, C. Mauvezin and J.M. Orellana-Gavalda *et al.*, 2009. Novel role of FATP1 in mitochondrial fatty acid oxidation in skeletal muscle cells. *J. Lipid Res.*, 50: 1789-1799.
- Song, Y.Z., J.Y. Feng, L.H. Zhou, G. Shu and X.T. Zhu *et al.*, 2008. Molecular cloning and ontogenesis expression of fatty acid transport protein-1 in yellow-feathered broilers. *J. Genet. Genomics*, 35: 327-333.
- Stahl, A., 2004. A current review of fatty acid transport proteins (SLC27). *Pflugers. Arch.*, 447: 722-727.

- Stahl, A., D.J. Hirsch, R.E. Gimeno, S. Punreddy and P. Ge *et al.*, 1999. Identification of the major intestinal fatty acid transport protein. *Mol. Cell*, 3: 299-308.
- Stahl, A., J.G. Evans, S. Pattel, D. Hirsch and H.F. Lodish, 2002. Insulin causes fatty acid transport protein translocation and enhanced fatty acid uptake in adipocytes. *Dev. Cell*, 2: 477-488.
- Stahl, A., R.E. Gimeno, L.A. Tartagli and H.F. Lodish, 2001. Fatty acid transport proteins: A current view of a growing family. *Trends Endocrinol. Metab.*, 12: 269-273.
- Thomas, C.E., E. Abordo-Adesida, T.C. Maleniak, D. Stone, C.A. Gerdes and P.R. Lowenstein, 2001. Gene transfer into rat brain using adenoviral vectors. *Curr. Protoc. Neurosci.*, 4: 24-24.
- Wang, Y., Q. Zhu, X.L. Zhao, Y.G. Yao and Y.P. Liu, 2010. Association of FATP1 gene polymorphisms with chicken carcass traits in Chinese meat-type quality chicken populations. *Mol. Biol. Rep.*, 37: 3683-3690.
- Wu, Q., A.M. Ortegon, B. Tsang, H. Doege, K.R. Feingold and A. Stahl, 2006b. FATP1 is an insulin-sensitive fatty acid transporter involved in diet-induced obesity. *Mol. Cell. Biol.*, 26: 3455-3467.
- Wu, Q., M. Kazantzis and H. Doege, 2006a. Fatty acid transport protein 1 is required for nonshivering thermogenesis in brown adipose tissue. *Diabetes*, 55: 3229-3237.