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Metabolic Effects of Polyunsaturated Fatty Acids in Chickens: A Review

Review Article

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ABSTRACT

Chicken has been used as a suitable model for lipid metabolism studies, because dietary modifications especially dietary fat type can change chicken body composition. Fats act as a condense source of energy and certain fatty acids such as polyunsaturated fatty acids (PUFAs) are required for both animal and human health. The n-3 PUFAs, especially, eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA) are well known for their favourable effects on human health. The n-3 PUFAs act as feed originated modulators of fatty acid oxidation and are feedback inhibitors of new fatty acid synthesis. This regulatory mechanism not only decreases overall liver lipid content and VLDL secretion, but also removes excessive very long-chain PUFA from cell membranes that may cause oxidant stress or damage membrane integrity. This paper reviews in brief the most important metabolic effects of PUFA in chickens.

KEY WORDS chicken, gene expression, metabolism, polyunsaturated fatty acids.

INTRODUCTION

It is now generally accepted that broilers consume an amount of feed that is adequate to meet their energy demands (NRC, 1994). Modern broiler chickens can reach their high genetic potential for growth, but this appears only after supplying ingredients to support their high energy requirements. Because of the limited capacity of the digestive tract of broiler chickens (Svihus, 2014), the inclusion of fats as condense energy sources in the diets seems to be unavoidable. Animal or vegetable fats or their mixtures are common ingredients in broiler diets. It has been reported that there is a positive correlation between dietary fat unsaturation degree and its absorption in chicken's gastrointestinal tract and there is a possible synergistic effect between saturated and unsaturated fats in diet (Freeman, 1984; Hulan *et al.* 1984; Ketels and DeGroote, 1989). Be-

sides its direct energy contribution, fat is also known to improve feed efficiency through an "extra caloric" effect (Vermeersch and Vanschoubroek, 1968). Specific fatty acids (FAs) such as n-3 polyunsaturated fatty acids (PUFAs) were reported to enhance performance in the growing chicks (Hellerstein *et al.* 1989; Cook *et al.* 1993; Korver and Klasing, 1997). The n-3 PUFAs, especially, eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA), are well known for their favourable effects on human health (Kinsella *et al.* 1990; Knapp, 1991).

Some strains of commercial birds like broiler chickens are at risk of fatness and their liver synthesises and secretes large amount of triglyceride (TG) and lipoproteins (Griffin *et al.* 1991). Consequently, the higher blood concentration of TG in broilers than layer hens will result in higher TG deposition in adipose tissue of broilers. Liver is the main site of lipoenesis in birds and it is suggested that hepatic TG

synthesis in broiler chickens is higher than the amount necessary to meet energy requirements, since the mobilization of adipose tissue TG is less than its synthesis (Newman *et al.* 2002).

According to Jump *et al.* (2005), the n-3 PUFAs act as feed-forward activators of fatty acid oxidation and feedback inhibitors to prevent the production of new FAs, including PUFA. This regulatory scheme not only reduces overall hepatic lipid content and very low density lipoproteins (VLDL) secretion, but also eliminates excessive very long-chain PUFA that may promote oxidant stress or impair membrane integrity. Hence, both enhanced catabolism of TG-rich particles as well as reduced secretion of VLDL particles are mechanisms that contribute to the hypolipidemic effect of (n-3) PUFA (Schoonjans *et al.* 1996). This paper briefly reviews the major metabolic effects of n-3 PUFAs.

Fats in poultry diets

There are many reports on the benefits of supplementing fat to chicken diets. Biely and March (1954) in one of the earliest studies, showed that inclusion of tallow in poultry diets improved nutrient utilization and resulted in a superior growth rate. Besides its' direct energy contributor role, fat is also known to improve feed efficiency through an "extra caloric" effect. This phenomenon has been reported in both chicks (Vermeersch and Vanschoubroek, 1968) and poults (Jensen *et al.* 1970). Improvement in chicken's growth rate and carcass yield, which mainly is achieved by increasing breast yield and reducing abdominal fat deposition, is the main goal of broiler meat production industry (Zerehdaran *et al.* 2004). Young chicks are not able to digest and absorb dietary fat efficiently but this improves with age (Polin and Hussein, 1982). The age dependent variations in fat digestibility are most obvious for more saturated fats containing high levels of C16:0 and C18:0 FAs. The superior fat digestibility in older chickens is in part due to a higher bile salt synthesis and intestinal lipase activity (Krogdahl, 1985; Krogdahl and Sell, 1989). The average digestibility of PUFAs is more than 85% in the chickens (ages more than 10 2 weeks) (Sklan *et al.* 1973; Noy and Sklan, 1995).

Poultry like most other animals have an absolute dietary requirement for linoleic and α-linolenic acids. Dietary deficiency in EFAs can cause lower growth and harmful effects on membrane biology, nervous system, bone formation, visual function and reproduction (Watkins, 1995; Calder, 1997). The NRC (1994) recommends a minimum of 1% of linoleic acid in chicken diets, but there is no recommendation for lenolenic acid requirements of chicken. The PUFAs linoleic (C18:2) and linolenic (C18:3) acids have a higher digestibility than saturated stearic acid (C18:0) (Sklan, 1976). In growing chickens, the reported digestibility of

soybean oil, lard and tallow are 96%, 92% and 67%, respectively (Sklan, 1976). It is well known that because of a synergism effect between saturated and unsaturated fats, mixed dietary fats could result in an improved digestibility of saturated fats in chicken (Doreau and Chilliard, 1997), which in turn could lead to better utilization of metabolisable energy of the mixed fats in poultry.

Metabolic effects of fatty acids

Frequently, FAs effects on cellular homeostasis are mediated through their metabolites. After uptake by cell, an acyl CoA synthetase enzyme rapidly converts the FAs to fatty acyl coenzyme A (CoA) thioesters (Coleman *et al.* 2002). This step is necessary to the further pathways of FAs metabolism, including elongation and desaturation, complex lipid synthesis, ß-oxidation, and producing prostaglandins, thromboxanes, and leukotrienes as the secondary signaling intermediates, which can in turn make possible alterations in synthesis of cellular second messengers such as inositol triphosphate and cyclicAMP (cAMP) (Harini and Ntambi, 2006). The altered FAs through different reactions such as elongation, desaturation, oxidation or peroxidation, enter into phospholipids and complex lipids such as ceramides and sphingolipids, or involve in eicosanoid synthesis (Madsen *et al.* 2005). The acyl CoA synthetase reaction and the different pathways of cellular FAs are quite fast so the free fatty acid content within the cell is usually maintained at very low levels. Therefore the metabolic effects of FAs in cells may be mediated, not only by means of free FAs, but also through fatty acyl CoAs and second messengers (Harini and Ntambi, 2006). The importance of dietary polyunsaturated FAs from the n-3 and n-6 series and their interactions with carbohydrate and lipid metabolism has received much attention. FAs motivate gluconeogenesis and glucose output of liver (Patsouris, 2006). High dietary PUFAs enrich hepatic plasma and microsomal membranes with long-chain PUFAs. This enrichment consequently changes hormone binding to cell-surface receptors and influences signaling mechanisms, which sequentially modify carbohydrate and lipid (Benatti *et al.* 2004). Over the past 25 years, several authors have verified that dietary n-3 and n-6 PUFAs decrease hepatic lipogenesis, while saturated and monounsaturated FAs have no inhibitory effects (Blake and Clarke, 1990). Furthermore, it seems that the lipogenic effects of n-3 and n-6 FAs are liver specific, and can not affect the adipose and lung lipogenic rates (Clarke and Jump, 1993). This fat reducing effect of PUFAs is due to suppressed synthesis of n-9 FAs family. The dietary PUFAs inhibit fatty acid biosynthesis, and consequently decrease the available substrates for Δ9 desaturase (Enser and Roberts, 1982), thus less n-9 FAs will be available to incorporate into plasma membranes.

PUFAs also decrease the activities of microsomal enzymes involved in fatty acid desaturation and TG synthesis (Christianse *et al*. 1991). These mechanisms cause a reduction of substrate availability for TG synthesis and a shift of FAs into the mitochondria and peroxisomal β-oxidative pathways (Flatmark *et al*. 1988). This is important considering that n-3 and n-6 PUFA are not metabolically interconvertible and have different physiological roles. It is demonstrated that 18:2 n-6 fatty acid deficiency adversely affects growth, reproduction and skin function in mammals (Burr, 1942; Holman, 1968; Hansen and Jensen, 1985). The 18:3n-3 fatty acid has a key role to maintaining normal growth and skin function (Burr, 1942; Fu and Sinclair, 2000).

Highly unsaturated long chain FAs (LC-PUFAs) such as 20:4 n-6, 20:5 n-3 and 22:6 n-3 which are derivatives of 18:2 n-6 and 18:3 n-3, are also physiologically considerable. In humans, brain, retina and other neural tissues contain large amounts of 22:6 n-3 and this fatty acid is required for normal cognitive and visual development mainly in fetal and infants (Hornstra *et al*. 1995). The energy and fat balance of animals can be manipulated by changing the dietary polyunsaturated to saturated fatty acid (P/S) ratio, in particular by the addition of the LC-PUFA's (Field *et al*. 1990; Luo *et al*. 1996; Couet *et al*. 1997).

The n-6 PUFA subtypes also inhibit the activity of enzymes associated with hepatic lipogenesis. Allmann and Gibson (1965) first reported in mice, that within two days of inclusion of just 2% linoleic acid (18:2 n-6) to a high-carbohydrate, fat-free diet, the rate of liver fatty acid synthesis and the activities of FAS, glucose-6- phosphate dehydrogenase and malic enzyme were reduced by 70%. On the contrary, dietary palmitic (16:0) or oleic (18:1 n-9) acids did not change hepatic FA synthesis (Clarke and Jump, 1994). The observation that dietary soybean oil reduced liver fat deposit is in agreement with the inhibitory activity reported for the n-6 PUFA family on enzymes of hepatic lipogenesis (Royan *et al*. 2013).

n-3 PUFAs

The n-3 PUFAs, especially EPA and DHA, are well known for their favorable effects on human health (Kinsella *et al*. 1990; Knapp, 1991). It seems that in human rations and most animal feeds there is an unbalanced fatty acid composition, so that the content of n-3 and n-6 FAs has been decreased and increased, respectively. One approach to restore this balance is by supplementing food with fish originated fats, which are rich in long chain n-3 PUFAs (Bezard *et al*. 1994; Tuncer *et al*. 1987; Manilla *et al*. 1999; Lopez Ferrer *et al*. 2001).

Fish oils contain high levels of 20:5 n-3 and 22:6 n-3 and are effective to prevent coronary heart disease (Dyerberg *et al*. 1975; Stansby, 1990). Previous reports indicate that chickens and rats fed diets enriched with fish oil had normal mortality rate, growth rate and feed conversion ratio (Phetteplace and Watkins, 1990; Nash *et al*. 1995).

In another report, fish oil reduced the catabolic response induced by immune stimulation and it can be speculated that part of the better performance observed in chickens fed fish oil containing diets was caused by the improved specific immunity (Chin *et al*. 1994). But, there are some contrasting reports too. Hulan *et al*. (1988) found that dietary fish oil reduced feed intake and body weight and increased feed conversion ratio in broilers, and attributed this unfavorable effect to a lower palatability of fish oil containing diets. In cultured hepatocytes, EPA (20:5 n-3) decreased the activity of acyl-CoA; 1, 2-diacylglycerol O-acyltransferase, the enzyme that catalyses the last step of TG synthesis (Rustan *et al*. 1988). These FAs reduce hepatic lipogenesis by down-regulation fatty acid synthase, spot 14 and stearoyl-CoA desaturase gene expression in liver (Jump *et al*. 1994). Fish oils also decrease the risk of atherosclerotic plaque formation and colon and breast cancer (Bougnoux *et al*. 1994; Caygill *et al*. 1995). Fish oils have also been reported to influence the activity of enzymes involved in hepatic TG synthesis. TG Mikkelsen *et al*. (1993) showed that the greater the degree of unstauration of the fatty acid the more fatty acid synthesis was inhibited; DHA (22:6 n-3) being more potent than EPA (20:5 n-3) or arachidonic (20:4 n-6).

When chickens were fed n-3 PUFA's derived from fish oil, plasma TG concentrations were reduced (Akiba *et al*. 1995). The n-3 PUFAs act as feed derived promoters of fatty acid oxidation and feedback inhibitors to synthesis of new FAs, including PUFA (Jump *et al*. 2005). This regulatory mechanism not only decreases overall liver lipid content and VLDL secretion, but also reduces excessive very LC-PUFAs that may cause oxidant stress or impair membrane integrity (Jump *et al*. 2005). Therefore, the higher degradation of TG-rich particles as well as a lower secretion of VLDL particles is responsible for the hypolipidemic effect of free FAs (Schoonjans *et al*. 1996). The effect of n-3 rich fats especially fish oil on serum TG reduction has been previously reported in chicks (Akiba *et al*. 1995). In the study of Phetteplace and Watkins (1989) the chickens fed menhaden oil had lower plasma TGslevels compared with values for those fed chicken fat; they concluded that a decrease in TGs (TG) synthesis by the liver could result in lower amounts of TG in the VLDL + LDL fraction. Daggy *et al*. (1987) and Royan *et al*. (2013) found that experimental birds fed fish oil exhibited decreased VLDL production. According to Jump *et al*. (2005), the n-3 PUFAs act as feed-forward activators of fatty acid oxidation and feedback inhibitors to prevent the production of new FAs, including

PUFA. This regulatory scheme not only reduces overall hepatic lipid content and VLDL secretion, but also eliminates excessive very LC-PUFA that may promote oxidant stress or impair membrane integrity. Hence, both enhanced catabolism of TG-rich particles as well as reduced secretion of VLDL particles are mechanisms that contribute to the hypolipidemic effect of n-3.

Eicosanoids

The essential FAs, linoleic acid (18:2 n-6) and linolenic acid (18:3 n-3) and their longer chain polyunsaturated derivatives are precursors for the synthesis of the eicosanoids, which have a regulatory role in many physiological processes (Smith and Marnett, 1991). Eicosanoids are short half-life biologically active agents, which are not stored in the cells, but their synthesis and release in response to a variety of hormones or cytokines is rather fast (within 5-60 s) (Madsen *et al*. 2005). The stored n-6 and n-3 FAs in phospholipid fractions of cell membranes and in glycerides and phospholipids of lipid bodies are released by phospholipase A2 and further converted to eicosanoids (Schmitz and Ecker, 2008).

Arachidonic acid is the primary metabolite of linoleic acid, which is generated by the action of Δ-6 and Δ-5 desaturase and elongase enzymes. Arachidonic acid is a major part of cell membranes, although phospholipase A2 mobilize and convert it to eicosanoids, such as thromboxane A2, prostacyclin, and leukotriene B4. Arachidonic acid derived eicosanoids are typically pro-active (arrhythmic, platelate activator and inflammatory stimulatory effects) whereas EPA derived such as 3-series-prostaglandins (PGI3, PGE3 and TXA3), and the 5-series leukotrienes have inhibitory roles. An increase in EPA intake and, thus a lower AA/EPA ratio in dietary lipids, cause the synthesis of anti-inflammatory (PGE3), or even less inflammatory compounds and thromboxanes with reduced pro-aggregatory and vasoconstrictive properties (Schmitz and Ecker, 2008). The key enzymes, cyclooxygenase (COX), lipoxygenase (LOX) and P450 epoxygenases normally use arachidonic acid released from phospholipids and convert it to eicosanoids (Madsen *et al*. 2005).

It is worth mentioning that some abnormal conditions such as inflammation, hypertension, asthma, and some types of cancer result from dysregulation of the eicosanoid pathway (Harris *et al*. 2002). The n-3 PUFAs with a similar structure to arachidonic acid may replace it in phospholipids.

Some n-3 PUFAs and specially DHA, are able to inhibit cyclooxygenases (and possibly lipoxygenases). Thus, a diet rich in n-3 PUFAs (specifically EPA and DHA) can in this manner influence eicosanoid biosynthesis (Serhan *et al*. 2002).

Effects of dietary fat on gene expression

Dietary FAs are able to regulate gene expression in a hormonal-independent approach. Nutrient mediated regulation of gene expression has important effects on metabolism, cellular differentiation, growth, development and health (Jump and Clarke, 1999). It has been suggested that dietary n-3 and n-6 PUFA's change liver FA synthesis and other lipogenic enzymes by regulating mRNA synthesis (Clarke *et al*. 1990). The FAs decrease lipogenesis by suppressing gene expression in liver, including that of FA synthase, spot14 and stearoyl-CoA desaturase (Jump *et al*. 1994). The n-3 and n-6 PUFA's also regulate the gene expression in adipose tissue, so that some studies have shown adipocyte-specific gene regulation by linolenic acid (18:3 n-3). A high dietary n-6 PUFA reduces the stearoyl-CoA desaturase 1 mRNA activity in rodent adipose tissue (Jones *et al*. 1996). Dietary PUFAs which inhibit lipogenesis are transported through plasma membrane and then bind to the cytosolic fatty acid binding protein (FABP). The FABP transport the 18-carbon FAs to the Δ6-desaturase and then carries FA products to the nucleus, where cytosolic FABP is transferred to a specific nuclear FABP (Benjamin and Friedrich, 2009). The inhibitory effect of dietary fats on lipogenesis, depends on the quantity and quality of the FA constituents. To inhibit the expression of lipogenesis related genes, a dietary FA should have a minimum of 18 carbons and at least 2 conjugated double bonds placed at the 9 and 12 positions (Clarke and Clarke, 1982; Clarke and Jump, 1993). Therefore, PUFA of the n-9 family are not able to suppress FA synthesis (Clarke and Jump, 1993). PUFAs may regulate gene expression through three manners: (a) releasing of the PUFA-regulated signal, (b) by PUFA-mediated regulation of the transacting factors, and (c) through interaction between trans-acting factor(s) and the target genes (Clarke and Jump, 1994).

Effects of PUFAs on regulation of transcription factors

Transcription factors belong to nuclear receptor (NR) family. NRs are defined as ligand-activated transcription factors which directly and indirectly regulate a number of genes of lipid metabolism and inflammatory signaling. The members of NRs family have comparable structural organization spite the wide variation in ligand specificity (Schmitz and Ecker, 2008). NRs act as sensors for FAs and cholesterol-derived metabolites and in this manner mediate the effect of nutrients on gene expression (Harini and Ntambi, 2006). NRs capability to regulate transcription is mediated via binding their conserved DNA-binding domains to DNA-response elements which contain conserved hexameric sequences with the ability to arrange in various bipartite configurations, including inverted and direct repeats (Khorasanizadeh and Rastinejad, 2001).

Changing in membrane composition or synthesis of secondary signaling intermediates are not the exclusively regulatory mechanisms of PUFAs on gene expression. Gottlicher *et al.* (1992) discovered a nuclear receptor with the ability of binding to FAs which attributed to a direct involvement of PUFAs on gene regulation. This mechanism may explain the rapid effects of PUFAs on gene transcription (Jump *et al.* 1994). PUFAs also have an effect on nuclear mechanisms that alter the expression of various genes encoding enzymes such as FAS (Blake and Clarke, 1990; Clarke *et al.* 1990), malic enzyme (Katsurada *et al.* 1987; Schwartz and Abraham, 1983), delta 9 desaturase (Ntambi, 1991; Tebbey and Buttke, 1992), acetyl-CoA carboxylase (Katsurada *et al.* 1990), and the S14 protein (Blake and Clarke, 1990; Clarke *et al.* 1990) that are involved in lipid metabolism. Inclusion of safflower oil or corn oil in diet reduced the synthesis of hepatic FAS by 80%, while hydrogenated coconut or cottonseed oil had no inhibitory effect (Schwartz and Abraham, 1982). A six day period of feeding young growing rats with 10% dietary safflower oil suppressed mRNAs encoding of FAS and S14 up to 30 and 50%, respectively compared with the values found with a fat-free, high-glucose diet (Clarke and Jump, 1994).

Two mechanisms may be involved in this suppression of enzyme synthesis: (a) interference with the expression rate of mRNA and/or (b) a decrease in the quantity of mRNA encoding the enzyme (Clarke and Jump, 1994). Dietary PUFAs control the synthesis of hepatic FAS and S14 mainly by decreasing the amount of mRNA encoding these proteins. PUFAs could control the phosphorylation state of a specific nuclear protein that operates the expression of genes encoding the lipogenic proteins (Blake and Clarke, 1990).

A fundamental hypothesis about the FA effects on gene expression has been that FAs enter cells and regulate the activity or amount of transcription factors (Jump *et al.* 2005). FABP and acyl CoA binding protein (ACBP) are involved in transportation of Non-esterified fatty acids (NEFAs) and fatty acyl CoA (FACoA) to intracellular compartments for metabolism or to the nucleus to cooperate with transcription factors (Hertzel and Bernlohr, 2000). The acyl CoA can be oxidized or after esterification can enter complex lipids such as TGs, phospholipids, or diacylglycerols. These complex lipids can also supply the cellular FA needs as necessary. On the other hand, FACoAs can produce prostaglandins, leukotrienes and thromboxanes. These secondary metabolites as well as complex lipids such as diacylglycerol, can raise cellular levels of secondary messengers such as cyclic AMP (cAMP), inositol triphosphate (IP3), and calcium. Both the secondary messengers and their lipid precursors can affect gene expression. On the other hand, free FAs and fatty acyl CoAs are able to act

directly at the nuclear level. In the nucleus, signaling mediated by FAs or their metabolites can cause alteration in nuclear receptor activation (Harini and Ntambi. 2006).

NEFAs are transported into the cell through transporters (fatty acid transport protein (FATP) or fatty acid transporter CD36 (FAT) or diffusion and are rapidly converted to FACoA by FATP or FACoA synthetases (Coleman *et al.* 2002; Jump *et al.* 2005). PUFAs of both n-3 and n-6 families control transcription of lipogenic genes (such as FA synthase, L-pyruvate kinase and stearoyl-CoA desaturase-1) via reducing of sterol regulatory element binding protein-1c (SREBP-1c) nuclear activity, through proteolysis, or changing SREBP-1c mRNA transcription (Schmitz and Ecker, 2008). This observation is important because competitive inhibition of PUFAs decrease oxysterols binding to liver X receptor (LXR), leading to a decreased SREBP-1c expression (Coleman and Lee, 2004).

Effects of PUFAs on PPARs expression

There are many transcription factors recognized as probable targets for fatty acid regulation, including SREBP-1c, LXRα, hepatic nuclear factors (HNF-4α and γ), retinoid X receptor (RXRα) and peroxisome proliferator-activated receptors (PPARα, δ, γ1, and γ2) (Bordoni *et al.* 2006). Most PUFAs (Forman *et al.* 1997; Yu *et al.* 1995; Kliewer *et al.* 1997) except erucic acid (Keller *et al.* 1993; Kliewer *et al.* 1997) are able to activate the members of the PPAR family (Yu *et al.* 1995; Kliewer *et al.* 1997). Study of the configuration of PPAR binding domains has demonstrated that the nuclear receptor suitably binds to different conformations of FAs (Nolte *et al.* 1998). Several reports clearly showed that the 5' flanking regions of genes encoding carnitine palmitoyltransferase, mitochondrial hydroxymethylglutaryl CoA synthase, fatty acyl CoA synthetase, acyl-CoA oxidase and mitochondrial UCPs all have DNA identification sequences for PPAR (Mascaro *et al.* 1998; Rodriguez *et al.* 1994; Varanasi *et al.* 1996; Aubert *et al.* 1997).

The PPARs have a binding affinity order as PUFA > MUFA > SFA (Kliewer *et al.* 1997). The n-3 PUFAs can stimulate PPARs by direct binding or via binding their cyclooxygenase and lipoxygenase metabolites. The pharmacological properties of the synthetic PPAR ligands confirm the idea that some effects of MUFAs and PUFAs may be mediated through the PPARs (Patsouris, 2006). The EPA and DHA have been identified as potent agonists and result in high PPARα expression (Desvergne and Wahli, 1999). There are several reports that dietary fat level and composition are able to affect PPARγ activity (Sato *et al.* 2004). In a report in humans, infusion of a TG mixture for 5 hours noticeably increasesd PPARγ mRNA expression in subcutaneous adipose tissue (Nisoli, *et al.* 2000). In animals, Vidal-Puig *et al.* (1996) reported that PPARγ expres-

sion increased in adipose tissue of mice fed high fat diets. In pigs, dietary safflower oil caused a 4-fold increase in PPARγ gene expression in adipose tissue (Spurlock *et al.* 2000).

It is reported that chicken PPARγ gene is moderately altered by dietary FAs. Sato *et al.* (2004) showed that PPARγ gene in chickens fed linoleic acid was up-regulated more than those fed oleic acid and that diet containing cholesterol did not affect PPARγ expression. These findings indicate nutritional modulation of chicken PPARγ mRNA expression might be manifested in a similar manner to mammals. Moreover, it seems that there is a feedback mechanism whereby changes in the levels of FAs in serum induce catabolism or storage of FAs through up-regulation of PPARα or PPARγ, respectively (Kliewer *et al.* 1997). In Chambrier *et al.* (2002) report in human adipocytes, EPA highly up-regulated PPARγ1 mRNA expression without any effect on levels, PPARγ2. Linolenic acid, DHA and n-6 PUFAs did not affect PPARγ1 or PPARγ2 expression.

CONCLUSION

This article briefly reviewed the most important aspects of PUFAs in poultry nutrition and metabolism. The higher digestibility of unsaturated fats, as well as their well known health promoting effects on human, has been increased the importance of these valuable ingredients in poulty diets.

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