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Efficiency of fatty acid accumulation into breast muscles of chickens fed diets with lycopene, fish oil and different chemical selenium forms

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The purpose of the investigation was to determine the effect of the addition of 12 ppm lycopene (Lyc), 2% fish oil (FO) or 0.25 ppm Se as selenate (SeVI) or selenized yeast (SeY) to an isoenergetic and isonitrogenous basal diet containing sunflower oil (SO) as the source of energy on the concentrations of fatty acids (FA), especially saturated- (SFA), mono- (MUFA) and polyunsaturated (PUFA) acids, in breast muscles of female and male chickens for six weeks. The influence of these additives on the capacity of Δ9-, Δ4- and Δ5-desaturations, the elongation of FA, and the yield of PUFA peroxidation (an oxidative stress) in breast muscles of female and male chickens were also studied. Dietary SeY most efficiently decreased the concentrations of SFA, MUFA and PUFA as well as malondialdehyde (the marker of the oxidative stress) in muscles of female and male chickens. The addition of FO most efficiently increased the concentration of n-3 long-chain PUFA (n-3LPUFA) and most effectively increased the concentration ratio of n-3LPUFA to SFA (n-3LPUFA/SFA), while most effectively decreased the concentration ratio of n-6PUFA to n-3PUFA (n-6PUFA/n-3PUFA) in muscles of chickens that are beneficial to human health. We conclude that further studies are necessary to determine if diets containing other chemical form of selenium compounds and other vegetable oils induce changes in the profiles of fatty acids in muscles of chickens that are beneficial to human health.

Key words: Chicken, lycopene, selenium, fish oil, sunflower oil, breast muscles, fatty acids, malondialdehyde.

INTRODUCTION

There is interest in meat containing higher levels of polyunsaturated fatty acids (PUFA), especially long-chain PUFA (LPUFA), because of their beneficial effects on human health, mainly in the prevention of cardiovascular

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disease (Harris et al., 2007, 2009). For this reason, there are numerous investigations concerning the enrichment of chicken meat with PUFA by the addition of fish or plant oils to diets (Betti et al., 2009a; b; Zuidhof et al., 2009; Rozbicka-Wieczorek et al., 2012).

However, chicken meat enriched with PUFA contains especially LPUFA with a high number of double bonds, which increases the susceptibility of meat to oxidation (Betti et al., 2009b; Cortinas et al., 2005; Rozbicka-Wieczorek et al., 2012). PUFA oxidation causes loss of sensory and nutritional values as well as the formation of potentially toxic species that compromise meat and adipose tissue quality and reduce its shelf life. One such product is malondialdehyde (MDA), which has long been considered as the index of oxidative rancidity. Indeed, MDA as well as other carbonyl compounds are naturally occurring byproducts of PUFA peroxidation and prostaglandin biosynthesis (Cortinas et al., 2005; Czauderna et al., 2011; Urso and Clarkson, 2003). Therefore, the oxidative stability of stored edible carcass parts of farm animals (e.g. pigs, ruminants or poultry) decreases when animals are fed a ration enriched in plant or/and fish oils (Perez et al., 2010; Rahimi et al., 2011; Rozbicka-Wieczorek et al., 2012; Urso and Clarkson, 2003).

Fortunately, the oxidative stability of edible carcass parts of animal can be manipulated by supplements added to a diet. The balance of unsaturated fatty acids (UFA), especially LPUFA, and antioxidants, like lycopenes, seleno-compounds or tocopherols in diets is a fundamental factor affecting the quality of edible carcass parts of farm animals (Betti et al., 2009b; Czauderna et al., 2009, Navarro-Alarcon and Cabrera-Vique, 2008; Rozbicka-Wieczorek et al., 2012; Tapiero et al., 2003; Yu et al., 2008). Indeed, numerous studies documented that the concentration of PUFA, especially phospholipids and cholesterol esters, in living organisms, was positively correlated with the selenium (Se) level in diets (Pappas et al., 2008; Schweizer et al., 2005; Yu et al., 2008). Really, Se is an important part of at least 25 Se-proteins possessing antioxidant, anti-inflammatory and chemoprotective properties; the most important are glutathione peroxidases, iodothyronine deiodinases, thioredoxin reductases, selenoprotein P, selenophosphate synthetase and selenoprotein (Navarro-Alarcon and Cabrera-Vique, 2008; Rayman, 2004; Tapiero et al., 2003).

Another approach to obtain animal products with low levels of lipid oxidation could be the addition of lycopene (Lyc) to diets (Boileau et al., 2013; Heber and Lu, 2002; Rao and Agarwal, 1998). Indeed, Lyc, an open-chain hydrocarbon carotenoid (C40), is the most potent antioxidant among common carotenoids (Boileau et al., 2013); Lyc can trap singlet oxygen and reduce mutagenesis and the risk of chronic diseases such as cardiovascular diseases (Rao and Agarwal, 1998). Moreover, Lyc has been found to inhibit the proliferation of several types of cancer cells, including those of the breast, lung and endometrium (Heber and Lu, 2002). Recent investigations have found that the influence of dietary Se (as high-selenized yeast (SeY) or and selenate (SeVI) affected the concentration of MDA, Lyc and/or fatty acids, especially PUFA, in tissues of experimental animals (Cortinas et al., 2005; Czauderna et al., 2009; Perez et al., 2010; Rao and Agarwal, 1998; Rozbicka-Wieczorek et al., 2012). Therefore, we hypothesized that dietary Lyc or Se as SeY and SeVI stimulate the accumulation of UFA, especially LPUFA, while decrease the concentration of MDA in breast muscles of chickens. Moreover, we intended to compare the impact of dietary anti-oxidants (that is, Lyc, SeY and SeVI) on the accumulation of UFA in breast muscles with dietary FO.

Therefore, the aim of the present study was to explore the effect of dietary Lyc, SeY, SeVI and FO on the concentration of UFA and the size of the oxidative stress in breast muscles of female and male chickens. The use of chickens significantly lowered the cost of our preliminary investigations.

MATERIALS AND METHODS

Birds, housing, nutrition and experimental design

One hundred and eighty two (182) one-day-old non-sexed hybrid Ross-308 chicks were obtained from a commercial hatchery and raised in five pens (groups) with 35 to 38 chicks per pen at the Poultry Research Station of the National Research Institute of Animal Production in Kraków-Balice (Poland). The experiment was carried out in accordance with established standards for use of birds. The chick room temperature, air exchange and humidity were maintained according to the recommendations for zoo-hygiene for young chickens. The protocol was approved by the local ethics and scientific authorities. Chicks were kept in metal cages (18 birds/m^2), on litter from deciduous trees. After 21 days of the experiment, the number of birds in pens was controlled, so, as to be in line with the Council Directive 2007/43/EC. Thus, at the end of the experiment, there were approximately 33 kg of live weight of chickens per m^2. Throughout the study, feed and water were provided for consumption *ad libitum*. Starter (1 to 21 days) and grower (22 to 42 days) rations were formulated based on maize-wheat-soyabean meal as was presented in our previous publication (Rozbicka-Wieczorek et al., 2012).

Basal and experimental diets were formulated to be isoenergetic and isonitrogenous. All rations contain sunflower oil (SO) as the source of energy (Table 1). The mean fatty acid composition (%) of dietary SO was as follows: C12:0 ~0.05, C14:0 ~1.9, C16:0 5.4, C16:1 0.3, C17:0 0.2, C17:1 ~0.03, C18:0 2.7, cis9C18:1 (*c9*C18:1) 39.1, *c9c12*C18:2 48.3, *c9c12c15*C18:3 0.3, C20:0 0.25, C20:1 0.14, C22:0 0.9, C22:1 0.15, C22:2 ~0.05, C24:0 0.25, C24:1 ~0.27. Basal and experimental diets were administered as a powder. During the experiment, feed consumption was monitored and feed intake was calculated per kg body weight gain of chickens; mortality was monitored. On the fifth day of breeding, the chicks were vaccinated against Goomboro disease and on the twelfth day, against the Newcastle disease (NCD). The vaccines against Goomboro and NCD were supplied by CEVAC® IBD L and Intervet

Table 1. The experimental design and the composition of the control and experimental diets.

Group	Additives added to the basal diet
I Control (negative)	4% SO[1]
II	4% SO and 12 ppm Lyc[2]
III	4% SO and 0.25 ppm Se as SeVI[3]
IV	4% SO and 0.25 ppm Se as SeY[4]
V	2% SO and 2% FO[5]

[1]SO, Sunflower oil; the iodine value of SO: 119 to 135; the acid value of SO: 1.3 mg KOH/g SO; gross energy of SO: 23.1 MJ Kg^{-1}; [2]Lyc, lycopene; [3]SeVI, sodium selenate; [4]SeY selenized yeast; [5]FO, fish oil; the iodine value of FO: 50 to 65 g/100 g FO; the acid value of FO: 20 mg KOH/g FO; the fatty acid profile of FO: C14:0 3.3%; C14:1 0.3%; C15:0 0.2%; C16:0 10.5%; C16:1 3,9%; C16:2 0.3%; C16:3 0.2%; C17:0 0.2%; C17:1 0.1%; C18:0 2.5%; *trans*C18:1 0.5%; *cis9*C18:1 31.8%; *cis11*C18:1 3.0%; other *cis*C18:1 0.4%; C18:2n-6 10.4%; C18:3n-3 3.9%; C20:0 0.3%; C18:4n-3 1.2%; *cis11*C20:1 5.9%; *cis11cis14*C20:2 0.9%; *cis8cis11cis14*C20:3 0.2%; C20:4n-6 0.3%; C20:5n-3 4.0%; C22:0 0.1%; *cis13*C22:1 6.6%; C22:4n-6 0.3%; C22:5n-3 1.6%; C22:6n-3 5.6%; C24:0 0.1%; C24:1 0.5% (the total PUFA abundance in FO: 42%); poultry digestible energy: 31.46 MJ Kg^{-1}; poultry gross energy: 34.2 MJ Kg^{-1}.

Sp. z o.o. (Poland), respectively. For a total of eight times in intervals of a few days during the entire experimental period, the chickens were fed a vitamin mixture, "Vitasol" (as a lyophilizate in drinking water). 'Vitasol' (BIOWET DRWALEW S.A., Poland) stimulated survival and the weight gain of birds. The body weight of chickens was determined after 21 and 42 days of the experiments; before each weighing, chickens were fasted for 12 h. Prior to feeding chickens, all experimental diets (that is, starter and grower) were supplemented with 12 ppm Lyc, 0.25 ppm Se (as SeVI or SeY) or 2% FO; the experimental design is shown in Table 1. The ingredients in the rations were determined by chemical analysis (AOAC, 2005).

On day 43 of the experimental period, 12 birds from each group, six male chickens (♂) and six female chickens (♀), were randomly selected. These birds were slaughtered by decapitation after stunning. The right breast muscles were quickly removed, weighed, homogenized and frozen at -32°C until chemical analyses. Each right breast muscle was analysed individually.

Reagents

All organic solvents were of high performance liquid chromatography (HPLC) grade and chemicals were of analytical grade. KOH, NaOH, dichloromethane (DCM), Na_2SO_4 and conc. HCl were purchased from POCH (Gliwice, Poland). Methanol, acetonitrile, n-hexane and n-heptane (99%, GC) were supplied by Lab-Scan (Ireland), whereas the CLA isomer mixture (2.1% *tt*CLA, 7.1% *c11t13*CLA, 40.8% *c9t11*CLA, 41.3% *t10c12*CLA, 6.7% *c8t10*CLA and 2.0% *cc*CLA) by the Industrial Chemistry Research Institute (Warsaw, Poland). Fatty acid methyl ester standards, sodium selenate (SeVI), and 25% BF_3 in methanol were purchased from Supelco and Sigma-Aldrich Co. (St. Louis, MO, USA). SeY (Se-*Saccharomyces cerevisiae*) was donated by Sel-Plex (non-commercial yeast sample; Alltech Inc., USA). About 83% of the total selenium content of SeY represents Se in the form of seleno-methionine (Se-Met) and 5% in the form of seleno-cysteine (Se-Cys) incorporated into the proteins of *S. cerevisiae* (Rayman, 2004); the chemical composition of the selenized yeast was presented in our previous publication (Czauderna et al., 2009). Trichloroacetic acid, 2,6-di-tert-butyl-p-cresol, 25% aqueous 1,5-pentanedi-aldehyde (PDA) solution, 2,4-dinitrophenylhydrazine (DNPH), containing about 30% water and 1,1,3,3-tetra-methoxy-propane (99%) were supplied by Sigma-Aldrich (St. Louis, MO, USA). Lycopene (Lyc), 10% in sunflower oil (LycoVit Dispersion 10%; Product-No. 30264388) was donated by BASF, the Chemical Company (Germany). SO was donated by Company AGROSOL (Pacanów, Poland), while FO, by Meals Manufacturing Company ZPM (Bokiny-Łapy, Poland). Water used for the preparation of mobile phases and chemical reagents was prepared using an Elix™ water purification system (Millipore). The mobile phases were filtered through a 0.45 µm membrane filter (Millipore).

Saponification and gentle base- and acid-catalyzed methylation of fatty acids

Homogenized chicken breast muscle samples (45 to 55 mg) were placed in vials and treated with a mixture of 2 ml of 2 M KOH in water and 2 ml of 1 M KOH in methanol. Next, 50 µl of the internal standard (IS) solution (17 mg ml^{-1} nonadecanoic acid in chloroform) were added to the obtained mixture. The resulting mixture was flushed with argon (Ar) for ~4 min. The vial was then sealed and the mixture vortexed were heated under Ar at 95°C for 10 min, cooled for 10 min at room temperature, and sonicated for 10 min. The resulting mixture was protected from the light and stored in the sealed vial under Ar at ~22°C overnight. Next, 3 ml of water were added to the hydrolysate and the solution was again vortexed. The obtained solution was acidified with 4 M HCl to ~pH 2 and free fatty acids were extracted four times with 3 ml of DCM. Extraction was repeated four times using 3 ml of n-hexane. The upper n-hexane layer was combined with the DCM layer, and next the resulting organic phase was dried with ~0.1 g of Na_2SO_4. The organic solvents were removed under a stream of Ar at room temperature. The obtained residue (I) was stored at -20°C until gentle base- and acid-catalyzed methylation of free fatty acids. 2 ml of 2 M NaOH in methanol were added to the residue (I) while mixing, then flushed with Ar, and reacted for 1 h at 40°C. After cooling the reaction mixture to ~4°C, 2 ml of 25% BF_3 in methanol were added, flushed with Ar, and heated for 1 h at 40°C. To the cooled reaction mixture 5 ml of water were added and then methylated fatty acids (FAME) were extracted with 5 ml of n-hexane. The supernatant was transferred to a GC vial.

Gas chromatographic equipments and analyses of FAME

The analyses of all FAME were performed on a SHIMADZU GC-MS-QP2010 Plus EI equipped with a BPX70 fused silica capillary column (120 m × 0.25 mm i.d. × 0.25 µm film thickness; SHIM-POL), quadrupole mass selective (MS) detector (Model 5973N) and injection port. Helium as the carrier gas operated at a constant pressure (223.4 kPa) and flow rate of 1 ml/min. Injector and MS detector temperatures were maintained at 200 and 240°C, respectively. The FAME profile in a 1 µl sample at a split ratio of 10:1 was determined using the column temperature gradient programme. The oven temperature was programmed as follows: initially 70°C for 4 min, then increasing by 12°C/min to 150°C, held for 6 min, programmed at 8°C/min to 168°C, held for 27 min, programmed at 0.75°C/min to 190°C, held for 10 min, programmed at 1.8°C/min to 210°C, held for 15 min, programmed at 6°C/min to 234°C, held for 4 min, programmed at 6°C/min to 236°C, held for 20 min. FAME identification was validated based on electron impact ionization spectra of FAME and compared with authentic FAME

standards and NIST 2007 reference mass spectra library.

Derivatisation of MDA in breast muscles, liquid chromatographic equipments and analyses

The MDA concentrations in muscle were determined after saponification followed by derivatization with DNPH to MDA-DNPH according to Czauderna et al. (2011). The chromatographic separation of MDA-DNPH from muscles was conducted using an ultra-fast liquid chromatography system (SHIMADZU, Kyoto, Japan), incorporating two LC-20AD$_{XR}$ liquid chromatographic pumps (UFLC$_{XR}$), a SIL-20AC$_{XR}$ autosampler (LFLC$_{XR}$), a CBM-20A communications bus module (UFLC), a CTO-20A column oven, a DGU-20A5 degasser, and a SPD diode array detector. The column was a Phenomenex C18-column (Synergi 2.5 µm, Hydro-RP, 100 Å, 100 mm in length) with an inner diameter of 2 mm. MDA in samples was analysed using a linear gradient of acetonitrile in water and the photodiode detector was set to 307 nm for UV detection (Czauderna et al., 2011). The concentration of MDA was calculated based on the fresh weight of breast muscle samples.

Statistical analyses

Results are presented as means of individually analysed samples of breast muscles. Mean values in columns having the same superscripts are significantly different at $^{a,b}P<0.05$ and $^{A,B}P<0.01$, while differences at $^{\alpha,\beta}P<0.1$ are indicated as tendencies. These one-factorial statistical analyses of the effects of additives (Lyc, SeVI, SeY or OF) in the ration were conducted using the non-parametric Mann-Whitney U test for comparing independent experimental groups. Statistical analyses were performed separately between the female chicken groups and between male chicken groups. Statistical analyses were performed using the Statistica v. 10 software package (Statistica, 2010).

RESULTS AND DISCUSSION

In the current study, neither macroscopic lesions nor pathological changes were found in the internal organs and muscles and adipose tissues of chickens fed the experimental diets enriched with Lyc, SeVI, SeY or FO (Table 1). Indeed, only long-term consumption of Se compounds, particularly selenite or selenide, at rates of more than 5 ppm Se can be teratogenic and hepatotoxic in animals and humans (Navarro-Alarcon and Cabrera-Vique, 2008; Tapiero et al., 2003). On the other hand, SeVI is less reactive and toxic in living organisms. Moreover, seleno-methionine (Se-Me), the predominant chemical form of Se in dietary SeY, is least reactive, as $tRNA_{Met}$ does not discriminate between Se-Met and methionine. Therefore, dietary Se-Met (derived from SeY) is incorporated into body protein in place of methionine (Rayman, 2004; Tapiero et al., 2003). Se-Met as Se-Met residue in proteins is a stable and safe-storage mode for Se in the body of animals and humans. Moreover, our recent studies with chickens documented that feeding chickens the experimental diets containing extra Se (like SeY) increased the average live body weight and breast muscle masses of chickens compared with the control chickens (Rozbicka-Wieczorek et al., 2012). In line with the above, we found also that SeY added to a diet improved feed conversion efficiency, stimulated the body mass gain and protein synthesis (the repartition) in body of experimental animals (Czauderna et al., 2009; Rozbicka-Wieczorek et al., 2012). These observations are consistent with the effect of the diet enriched in SeY (group IV) on the decrease in the concentration sum of all assayed fatty acids (ΣFA) in breast muscles of chickens (Table 2). Similarly, our newest studies documented that the diet with SeY resulted in the lower concentration of ΣFA in thigh muscles of female and male chickens compared with the control birds (Rozbicka-Wieczorek et al., 2014).

Effect of the experimental diets on the saturated fatty acid profile of breast muscles

The effects of the experimental diets on the concentration of saturated fatty acids (SFA) in chicken breast muscles are summarized in Table 2. The addition of SeY to the diet (Groups IV) resulted in the decrease in the concentrations of C14:0, C16:0 and C18:0 as well as the concentration sum of SFA and atherogenic (A-SFA) and thrombogenic (T-SFA) saturated fatty acids in muscles of female and male chickens in comparison with the control birds and usually other experimental female and male chickens (Groups II, III and V). Similarly, the diet with SeY most efficiently decreased the concentration sum of assayed FA (ΣFA) in muscles of female and male chickens. In line with the above, our recent studies revealed also that dietary extra SeY decreased the concentrations of SFA, A-SFA and T-SFA in thigh muscles of female and male chickens compared with the control birds (Rozbicka-Wieczorek et al., 2014). The values of the A-SFA and T-SFA indexes ($_{index}A^{SFA}$ and $_{index}T^{SFA}$) (Ulbricht and Southgate, 1991) in muscles of chickens fed the diets with SeIV, SeY or FO were greater, however, than those in the control birds and the chickens fed with the diet Lyc (Group II). These findings are due to the concentrations of PUFA, including n-6PUFA, n-3PUFA, being usually more significantly decreased in muscles of SeVI-, SeY- or FO-fed chickens (Groups III, IV and V) compared with those in the control birds or Lyc-fed chickens (Group II). As can be seen from the data summarized in Table 2, the addition of SeY or FO to the diets (Groups IV and V) also increased the concentration ratios of A-SFA or T-SFA to ΣFA (that is, A-SFA/ΣFA and T-SFA/ΣFA) in muscles of chickens in comparison with the control group and other experimental groups. These results are due to the concentration of ΣFA being usually more significantly decreased in muscles compared with the muscle concentrations of A-SFA and T-SFA in SeY- or FO-fed chicken.

Our results show more profound effects of dietary SeY

Table 2. The concentration of C14:0, C16:0, C18:0, the sum of saturated fatty acids (SFA), atherogenic (A-SFA) and thrombogenic (T-SFA) saturated fatty acids, indexes of A-SFA ($_{index}A^{SFA}$)[1] and T-SFA ($_{index}T^{SFA}$)[2], and ratios of A-SFA to ΣFA[3] and T-SFA to ΣFA in breast muscles[4].

Group		C14:0 µg/g	C16:0 mg/g	C18:0 µg/g	SFA[5] mg/g	A-SFA[6] mg/g	A-SFA / ΣFA[2]	$_{index}A^{SFA}$	T-SFA[7] mg/g	T-SFA / ΣFA	$_{index}T^{SFA}$	ΣFA[3] mg/g
I Negative Control	♂	28^{β}	$1.34^{B\alpha d}$	598^{α}	2.06	1.39	0.202^{cd}	0.291^{IJKL}	1.97	0.287	0.632^{GHIc}	6.86^{df}
	♀	47^{B}	1.67^{A}	689^{Bgh}	2.51^{Bde}	$1.72^{Ad\alpha}$	$0.197^{EF\beta\alpha}$	$0.288^{\beta OP}$	2.41^{Ade}	$0.276^{\sigma cd}$	$0.607^{KL\alpha}$	8.73^{Be}
	Σ[8]	38^{A}	1.51^{a}	644^{Aab}	$2.29^{A\alpha}$	1.55^{a}	0.199^{ABa}	0.289^{ABCa}	2.20^{a}	0.281^{AB}	0.618^{ABCD}	7.79^{Aa}
II	♂	37^{σ}	$1.71^{\sigma B}$	675^{ef}	2.53^{c}	1.78^{α}	0.205^{ef}	0.295^{IMN}	2.42^{α}	$0.279^{\beta\Delta b}$	0.648^{cdeJ}	8.68^{f}
	♀	34^{Δ}	0.97^{AC}	466^{B}	1.54^{BC}	1.01^{AB}	$0.209^{GH\beta}$	$0.316^{\Delta R}$	1.47^{AB}	$0.305^{\sigma\gamma}$	$0.680^{\alpha\beta f}$	$4.83^{B\alpha}$
	Σ	$35^{a\alpha}$	1.34^{b}	570^{c}	2.04^{a}	1.39^{b}	0.206^{CD}	0.303^{DEFGa}	1.95^{b}	0.288^{CD}	0.660^{AEab}	6.76^{b}
III	♂	$21^{\sigma c}$	1.24	523	1.87	1.27	0.213	$0.310^{J\alpha}$	1.79	0.301^{β}	0.737^{Gd}	5.95
	♀	42	$1.41^{C\mu}$	$563^{\beta\delta}$	$2.10^{CD\beta}$	$1.45^{B\sigma}$	0.211^{α}	$0.318^{\beta c}$	$2.01^{B\beta}$	0.294^{e}	0.687	$6.85^{\alpha\beta}$
	Σ	31^{α}	1.33^{c}	543^{ad}	1.98^{b}	1.36^{c}	0.212^{ab}	0.315^{AHb}	1.90^{c}	0.297^{a}	0.710^{Ba}	6.40^{c}
IV	♂	20^{d}	$1.00^{\sigma\alpha d}$	$399^{\alpha e}$	1.48^{c}	1.02^{α}	0.225^{ce}	0.338^{KM}	1.42^{α}	0.312^{Δ}	0.757^{HJ}	4.55^{d}
	♀	$17^{B\Delta}$	0.99	$408^{g\beta}$	1.46^{dD}	1.00^{α}	0.229^{EG}	$0.341^{O\Delta}$	1.41^{d}	0.322^{c}	0.763^{Kfg}	4.39^{e}
	Σ	19^{Aab}	0.99^{abc}	403^{Acd}	1.47^{Aab}	1.01^{abc}	0.227^{ACb}	0.339^{BDFb}	1.42^{abc}	$0.317^{ACa\alpha}$	0.760^{CEF}	4.47^{Abc}
V	♂	$42^{\beta cd}$	1.41	536^{f}	2.04	1.46	0.228^{df}	$0.356^{LN\alpha}$	1.99	0.311^{b}	0.707^{Ie}	6.40
	♀	28	0.99^{μ}	$449^{h\delta}$	$1.51^{e\beta}$	$1.02^{d\sigma}$	0.224^{FH}	0.353^{PRc}	$1.46^{e\beta}$	$0.323^{d\gamma e}$	$0.711^{L\beta g}$	$4.54^{d\beta}$
	Σ	35^{b}	1.20	492^{b}	1.72^{α}	1.12	0.226^{BD}	0.360^{CEGH}	1.73	$0.317^{BD\alpha}$	0.710^{DFb}	5.47^{a}

[1]The atherogenic index = (C12:0 + 4*C14:0 + C16:0)/(MUFA +PUFAn-6 + PUFAn-3); [2]the thrombogenic index = (C14:0 +C16:0+ C18:0)/0.5*MUFA + 0.5*PUFAn-6 + 3*PUFAn-3 + PUFAn-3/PUFAn-6); [3]the sum of all assayed fatty acids; [4]mean values in columns having the same superscripts are significantly different at $^{a,b}P<0.05$ and $^{A,B}P<0.01$; [5]SFA, the concentration sum of C8:0, C10:0, C12:0, C14:0, C16:0, C18:0, C20:0, C22:0 and C24:0; [6]A-SFA: C12:0+C14:0+C16:0; [7]T-SFA: C14:0+C16:0+C18:0; [8]the concentration of FA in muscles of broilers of both sexes (Σ).

On the concentration of ΣFA in muscles (Table 2). Indeed, it has been shown that dietary SO, rich in *c9c12*C18:2, act synergistically with SeY towards the activation of the carnitine palmitoyltransferase 1 (CPT1) gene through the peroxisomes proliferator-activated receptor a (PPARa) (Stahle et al., 2009). Thus, we argue that the substantial decrease in the concentrations of SFA, MUFA and PUFA in thigh muscle of SeY supplemented chickens may be the upregulation of CPT1, with increased activity of PPARa (Goto et al., 2011). In addition, a study on laboratory animal lymphocytes showed that dietary Se addition was effective for improving β-oxidation (Kuryl et al., 2008). In line with the above, we found that dietary extra SeY also significantly reduced the concentrations of SFA, MUFA and PUFA in breast muscles of female and male chickens compared with the control birds (Rozbicka-Wieczorek et al., 2012). Moreover, our results show more profound dietary effects of SeY than SeVI on the abundance of SFA, MUFA and PUFA in breast muscles (Tables 2 to 5). This may reflect the lover yield of accumulation of Se in the body of

Table 3. The concentration of *c9*C18:1, the sum of monounsaturated fatty acids (MUFA), *c9t11*CLA (*c9t11*), linoleic acid (LA), α-linolenic acid (αLNA), *c5c8c11c14*C20:4 (AA), *c7c10c13c16c19*C22:5 (DPA), *c4c7c10c13c16c19*C22:6 (DHA), n-6PUFA (n-6) and n-3PUFA (n-3) in breast muscles.

Group		*c9*C18:1 mg/g	MUFA mg/g	Desaturase index $_{C18:0}\Delta9$ [3]	Desaturase index $_{C16:0}\Delta9$ [4]	*c9t11* µg/g	LA mg/g	AA µg/g	αLNA µg/g	DPA µg/g	DHA µg/g	n-6 mg/g	n-3 µg/g
I	♂	2.11^{g}	2.63^{bcd}	0.779	0.021^{a}	-	1.63^{α}	259^{Ja}	97^{abc}	33^{GHc}	22^{Fc}	2.18^{π}	238^{bc}
Negative	♀	2.90^{Bef}	3.63^{Eef}	0.808^{bc}	0.039^{HIJ}	-	2.02^{Bfg}	237^{Oc}	140^{FGef}	$36^{Ke\Delta}$	$30^{K\Delta\pi c}$	$2.54^{gh\Delta}$	$294^{KLd\alpha}$
Control	Σ[2]	$2.51^{A\alpha ab}$	3.13^{ABCD}	$0.796^{A\alpha\Delta a}$	$0.030^{ABa\alpha}$	-	1.82^{Aab}	248^{AB}	119^{ABCa}	35^{ABC}	$26^{ABa\sigma}$	2.36^{ab}	266^{ABa}
II	♂	2.42^{β}	2.68^{α}	0.782	0.034^{FGab}	-	2.50^{ef}	296^{KLM}	$82^{\Delta Ed}$	$24^{\alpha d}$	18^{G}	$3.14^{\pi fg}$	214^{GH}
	♀	1.17^{Δ}	1.58^{E}	0.715^{b}	0.039^{KL}	-	1.23^{B}	250^{RS}	38^{e}	28^{β}	$13^{\Delta L}$	$1.78^{g\delta\beta}$	164^{de}
	Σ	$1.79^{\alpha c}$	2.13^{A}	0.759^{α}	$0.036^{CDE\alpha}$	-	1.86^{c}	273^{CD}	$60^{Da\alpha}$	26^{Dab}	$15^{C\sigma}$	2.46^{cd}	189^{CDa}
III	♂	1.50	$1\ 87^{b}$	0.742	0.016^{F}	1.75^{b}	1.82	221^{KNb}	$40^{a\Delta}$	$10^{GI\alpha}$	10^{Hc}	2.30	115^{GIb}
	♀	1.83^{Δ}	2.32^{Δ}	0.764	$0.016^{HK\beta}$	1.83	$1.94^{\Delta\beta}$	227^{T}	$59^{fg\delta}$	14^{eL}	24^{π}	$2.44^{\delta\mu}$	183^{α}
	Σ	1.67^{ad}	2.09^{Ba}	0.754^{Δ}	0.016^{AC}	1.79^{a}	1.88^{ad}	224^{CFH}	49^{Ab}	12^{AEa}	17^{ADb}	$2.37^{e\sigma}$	149^{ACE}
IV	♂	1.21^{β}	$1.55^{c\alpha}$	0.753	0.019^{b}	2.68	$1.20^{\alpha e}$	160^{Lab}	25^{Eb}	13^{HJ}	14^{I}	1.55^{f}	111^{HJc}
	♀	1.16^{e}	$1.47^{e\Delta}$	0.741^{c}	0.014^{IL}	3.11	$1.11^{f\Delta}$	182^{RZc}	$23^{F\delta}$	$11^{KN\beta}$	$18^{Nc\chi}$	1.50^{Δ}	120^{KNe}
	Σ	1.19^{Acd}	1.51^{Ca}	0.747^{A}	0.017^{Da}	2.89	1.15^{Acd}	171^{ADFI}	$24^{BDb\beta}$	12^{BFb}	16^{Eab}	1.52^{ace}	115^{BDF}
V	♂	1.44^{g}	1.90^{d}	0.777	0.017^{G}	5.84^{b}	1.55^{f}	124^{JMN}	46^{cd}	52^{IJcd}	85^{FGI}	1.85^{g}	236^{IJ}
	♀	1.24^{f}	1.56^{f}	0.735^{d}	$0.014^{J\beta}$	3.89	$1.10^{g\beta}$	109^{OSTZ}	29^{Gg}	$43^{\Delta LN}$	$72^{KLN\chi}$	$1.36^{h\beta\mu}$	200^{LN}
	Σ	1.34^{b}	1.73^{D}	0.748^{a}	0.015^{BE}	4.85^{a}	1.32^{b}	113^{BEHJ}	$37^{C\alpha\beta}$	47^{CDEF}	78^{BCDE}	$1.60^{bd\alpha}$	217^{EF}

[1]Mean values in columns having the same superscripts are significantly different at $^{a,b}P<0.05$ and $^{A,B}P<0.01$; [2]the concentration of FA in muscles of broilers of both sexes; [3] Δ9-desaturase index ($_{C18:0}\Delta9$ index) = *c9*C18:1/(*c9*C18:1+C18:0); [4]Δ9-desaturase index ($_{C16:0}\Delta9$ index) = *c9*C16:1/(*c9*C16:1+C16:0); [5]below the quantification limit (L_Q).

chickens fed with the diet enriched in SeVI than SeY (Rayman, 2004).

Influence of the experimental diets on the monounsaturated fatty acid profile of breast muscles

As can be seen from the results summarized in Table 3, the addition of SeVI, SeY or RO to the diet resulted in the decrease in the concentration of MUFA, including *c9*C18:1, compared with the control birds and Lyc-fed chickens. Interestingly, SeY added to the diet most efficiently decreased the concentration of MUFA, including *c9*C18:1, in breast muscles. The presented experiments document the significant negative influence of dietary SeVI and SeY on the capacity of Δ 9-desaturation of C18:0 and C16:0 compared with the control birds and Lyc-fed chickens. Thus, the current studies support our earlier investigations in which dietary SeVI and SeY also decreased the capacity of Δ9-desaturase (that is, stearoyl-CoA desaturase-1) in thigh muscles of broiler chickens and rats (Czauderna et al., 2009; Rozbicka-Wieczorek et al., 2014). Our recent studies (Rozbicka-Wieczorek et al., 2012) and the current investigation also reinforce the finding that the yield of Δ9-desaturation depends on the length of the carbon atom chain of desaturated fatty acids (Table 3). Indeed, the values of the Δ9-desaturase index for C18:0 (substrate of Δ9-desaturase) are higher than the values of this

Table 4. The concentration of long-chain PUFA (LPUFA), n-6LPUFA (n-6_{LPUFA}), n-3LPUFA (n-3_{LPUFA}) and values of the ratio of n-6PUFA/n-3PUFA (n-6/n-3), the elongase index, Δ4- and Δ5-desaturase indexes ($\Delta4_{index}$, $\Delta5_{index}$), the ratios of PUFA, LPUFA, n-6_{LPUFA} and n-3_{LPUFA} to SFA in breast muscles[1].

Group		LPUFA µg/g	LPUFA, µg/g		n-6 n-3	elongase index[3]	$\Delta4_{index}$[4]	$\Delta5_{index}$[5]	PUFA SFA	LPUFA SFA	n-6_{LPUFA} SFA	n-3_{LPUFA} SFA
			n-6_{LPUFA}	n-3_{LPUFA}								
I	♂	$438^{d\alpha}$	297^{cd}	$141^{I\beta a}$	9.2^{Fe}	0.588^{Ec}	$0.393^{B\alpha}$	0.882^{de}	1.047^{b}	0.212	0.144^{FGHI}	$0.068^{\Delta Eb}$
Negative	♀	434^{Hf}	280^{NO}	$154^{\Delta R}$	$8.6^{\beta f}$	0.480^{c}	0.457	0.846^{h}	1.035^{β}	$0.173^{\delta\Delta}$	0.122^{J}	0.061^{c}
Control	Σ[2]	436^{ABa}	288^{ABa}	$147^{ABC\alpha}$	8.9^{ABab}	0.539^{Aa}	0.428^{Aa}	0.865^{a}	1.040^{Aa}	0.191^{α}	$0.126^{ABC\alpha}$	0.064^{Aa}
	♂	$475^{FG\alpha e}$	343^{Ie}	132^{JKb}	14.7^{Ge}	0.646^{F}	0.422^{β}	$0.863^{\alpha f}$	1.221^{bcF}	0.187	0.136^{Fc}	0.052^{Δ}
II	♀	424^{Jg}	298^{RS}	$126^{\Delta\delta e}$	$10.8^{\beta\Delta g}$	0.603^{I}	0.317	0.838^{δ}	1.117^{de}	$0.275^{A\delta}$	$0.193^{JLK\Delta}$	$0.082^{\mu\gamma}$
	Σ	449^{CDE}	321^{CDE}	$129^{DEF\alpha}$	13.0^{Acc}	$0.624^{B\alpha}$	0.371^{b}	0.851^{b}	1.181^{ABC}	$0.221^{\alpha a}$	0.157^{ADa}	$0.063^{B\alpha}$
	♂	328^{Fid}	253^{Je}	76^{IJL}	20.0^{FH}	$0.750^{c\Delta G}$	0.489^{δ}	0.875^{g}	1.185^{α}	0.176	0.135^{Gd}	0.040^{EF}
III	♀	393^{i}	269^{fU}	124^{λ}	$13.3^{Ff\Delta}$	0.711^{J}	0.644	0.846^{β}	1.156^{fg}	0.187^{A}	0.128^{L}	$0.059^{d\mu}$
	Σ	361^{Cab}	261^{CGHa}	100^{ADG}	15.9^{BDcd}	$0.729^{C\ a\alpha b}$	0.592^{c}	0.860^{c}	1.169^{DE}	$0.182^{a\beta}$	0.131^{Bab}	$0.050^{Ca\alpha\beta}$
	♂	$276^{G\beta}$	190^{Ic}	$86^{N\beta b}$	14.0^{α}	$0.621^{\Delta H}$	$0.509^{\alpha d}$	$0.841^{Bdg\alpha}$	1.032^{c}	0.187	0.129^{He}	0.058
IV	♀	304^{HJi}	207^{NRfh}	$97^{RO\delta}$	12.5^{μ}	0.646^{K}	0.634	$0.882^{h\delta\beta\lambda}$	0.999^{df}	0.209^{Δ}	$0.142^{\Delta\lambda}$	0.066^{σ}
	Σ	290^{ADbc}	199^{ADGb}	91^{BEH}	13.2^{Ead}	0.633^{Db}	0.574^{Aa}	0.862^{A}	1.016^{BD}	0.198^{β}	0.135^{CE}	0.062^{BD}
	♂	$363^{e\beta}$	173^{Jd}	190^{KLNa}	$7.8^{GH\alpha}$	0.241^{EFH}	$0.619^{B\beta\delta d}$	0.816^{ef}	$0.972^{F\alpha}$	0.178	0.085^{Icde}	0.092^{Fb}
V	♀	324^{fg}	153^{OSUh}	$171^{Oe\lambda}$	$6.8^{g\mu}$	0.259^{IJKc}	0.625	0.834^{λ}	$0.979^{\beta eg}$	0.215	$0.102^{K\lambda}$	$0.113^{cd\gamma\sigma}$
	Σ	339^{BEc}	160^{BEHb}	180^{CFGH}	7.3^{CDEFb}	0.253^{ABCD}	0.621^{bc}	0.820^{ABabc}	0.975^{CEa}	0.209	$0.098^{DEb\alpha}$	0.103^{ABCD}

[1]Mean values in columns having the same superscripts are significantly different at $^{a,b}P<0.05$ and $^{A,B}P<0.01$; [2]the concentration of FA in muscles of broilers of both sexes; [3]the elongase index = the concentration ratio: C24:5n-3/(C24:5n-3+C22:5n-3); [4]Δ4-desaturase index ($\Delta4_{index}$) = C22:6n-3/(C22:6n-3+C22:5n-3); [5]Δ5-desaturase index ($\Delta5_{index}$) = C20:4n-6/(C20:4n-6+C20:3n-6).

index for C16:0 and C14:0 (that is, $_{C18:0}$ Δ9 > $_{C16:0}$ Δ9 > $_{C14:0}$ Δ9; Table 3). Consequently, the values of the Δ9-desaturase index for C14:0 are close to zero (that is, 0.0011 to 0.0007; data not shown). Our results indicate that the diet enriched in FO, reach in LPUFA, also decreased the Δ9-desaturase index, especially for C16:0 ($_{C16:0}$ Δ9) compared with the control female and male chickens.

Considering the above results, we suggest that dietary FO-long-chain fatty acids (e.g.: *c11*C20:1, *c11c14*C20:2, C20:4n-6, C20:5n-3, *c13*C22:1, C22:4n-6, C22:5n-3, C22:6n-3; Table 1) decreased the capacity of Δ9-desaturase, elongation and Δ5-desaturase in muscles of female and male chickens compared with the control birds (Tables 3 and 4). On the other hand, it was found that the addition of Lyc to the diet decreased the Δ9-desaturase index for C18:0 in muscles of female chickens, whereas it increased the Δ9-desaturase indexes for C18:0 and C16:0 in muscles of male chickens (Group II) compared with the control chickens. As consequence, dietary Lyc resulted in the decrease in the concentration of MUFA, including *c9*C18:1 in muscles of female chickens, while increased in muscles of male chickens compared with the control birds.

Influence of the experimental diets on the polyunsaturated fatty acid profile of breast muscles

As can be seen from the data summarized in Tables 3 to 5, the addition of SeY to the diet decreased the concentrations of n-6PUFA, n-3PUFA, including n-6LPUFA and n-3LPUFA, as well as the concentration sum of PUFA and LPUFA in muscles in comparison with the control birds. Fortunately, the diet enriched in SeY revealed negligible influence on the concentration ratios of PUFA/SFA, LPUFA/SFA, n-6LPUFA/SFA and n-3PUFA/SFA in muscles of chickens

Table 5. The concentration of malondialdehyde (MDA), and the concentration sum of PUFA, and the MDA indexes in breast muscles[1].

Group		MDA ng/g	MDA/(FA-PUFA) (ng/g)/(mg/g) ($^{simple}MDA_{index}$)[2]	Peroxidation index (MDA_{index})[3]	PUFA mg/g
I	♂	$1.34^{E\alpha ef}$	0.285^{EFG}	1.620^{FGef}	2.16^{μ}
Negative	♀	1.92^{IKL}	$0.313^{de\beta}$	1.741	$2.59^{ef\sigma}$
control	Σ	1.63^{Aab}	$0.300^{Aab\alpha}$	1.685^{ABa}	$2.38^{a\alpha}$
	♂	$1.50^{\alpha gF}$	0.268^{Hic}	1.485^{HIJeg}	$3.09^{\mu cd}$
II	♀	1.85^{NMO}	0.504^{LNdf}	1.899	$1.72^{e\lambda}$
	Σ	1.68^{Bcd}	0.386^{Ba}	1.697^{Cbc}	$2.41^{A\beta}$
	♂	1.71^{Eg}	0.428^{EHJ}	1.763^{HKfhi}	2.21
III	♀	1.14^{INR}	$0.258^{Lgh\beta\gamma}$	1.469	$2.43^{\lambda\delta}$
	Σ	1.41^{Cac}	$0.343^{C\alpha}$	1.608^{Dabd}	$2.32^{b\Delta}$
	♂	0.52^{EFGH}	0.172^{FJKc}	1.340^{FIKLgi}	1.53^{c}
IV	♀	0.40^{KMRS}	$0.137^{NReg\gamma}$	1.274	1.46^{σ}
	Σ	0.46^{ABCD}	0.154^{ABCD}	1.309^{ACEd}	1.49^{Aab}
	♂	1.69^{fgH}	0.406^{GIK}	1.900^{GJLh}	1.99^{d}
V	♀	1.19^{LOS}	0.365^{Rfh}	1.762	$1.47^{f\delta}$
	Σ	1.44^{Dbd}	0.387^{Db}	1.823^{BDEc}	$1.75^{\alpha\beta\Delta}$

[1]Mean values in columns having the same superscripts are significantly different at $^{a,b}P<0.05$ and $^{A,B}P<0.01$, while differences at $^{\alpha,\beta}P<0.1$ are indicated as tendencies; [2]the concentration ratio (r) of MDA (ng/g) to the difference between the sum of all assayed fatty acids (mg/g) and polyunsaturated fatty acids (mg/g): r = MDA/(FA-PUFA);[3]peroxidation index: MDA_{index} = [MDA (ng/g) + PUFA (mg/g)] / PUFA (mg/g) (Rozbicka-Wieczorek et al., 2012)

compared with the control animals. Interestingly, dietary Lyc or SeVI most efficiently increased the ratio of PUFA/SFA in breast muscles of chickens compared with the control group and SeY- or FO-fed chickens. Data from the current investigation confirm our recent study in which the concentration ratio of PUFA/SFA was highest in thigh muscles of female and male chickens fed with the diets containing Lyc or SeVI (Rozbicka-Wieczorek et al., 2014). Considering the above, we argue that Lyc- or SeVI-dietary manipulations are linked with improved nutritional properties and quality of breast and thigh muscles obtained from chickens. Considering the current results and our other studies (Czauderna et al., 2009; Rozbicka-Wieczorek et al., 2012; 2014) we suggest that the PUFA/SFA ratio was significantly higher in muscles of Lyc- or SeVI-fed chickens, which possibly reflects the effect of oxidation protection of Lyc and SeVI (the antioxidants) on the unsaturated fatty acids in living organisms (Navarro-Alarcon and Cabrera-Vique, 2008; Rayman, 2004; Rao and Agarwal, 1998).

Interestingly, the concentration ratio of n-6PUFA/n-3PUFA (n-6/n-3; Table 4) was significantly greater in muscles of female chickens and especially male chickens fed with the diet enriched in Lyc, SeVI or SeY. The distinct difference in n-6PUFA versus n-3PUFA concentrations may reflect up-regulated control of enzyme transcription by circulating n-3PUFA over n-6PUFA. In addition, dietary Lyc most efficiently stimulated the accumulation of n-6LPUFA (e.g. AA) in breast muscles and thigh muscles (Rozbicka-Wieczorek et al., 2014), while decreased the concentration of n-3LPUFA (e.g. DPA or DHA) in these muscles compared with the control female and male chickens. Thus, our current studies carried out on chickens also reinforce the finding that *c9c12*C18:2 (LA) originating from dietary SO could be metabolized *in vivo* into n-6LPUFA (via Δ6-desaturation, elongation and Δ5-desaturation of LA) using the same pathway as *c9c12c15*C18:3 (that is, n-3PUFA).

As can be seen from the results summarized in Table 4, SeVI or SeY added the diet decreased the concentration of LPUFA, including n-6LPUFA and n-3LPUFA, in muscles compared with the control birds and Lyc-fed chickens. So, our results (Table 4) support our earlier observations that dietary SeY or SeVI activate the

carnitine palmitoyltransferase 1 (CPT1) gene and increased the yield of β-oxidation (Kuryl et al., 2008). On the other hand, dietary FO increased the accumulation of n-3LPUFA, especially DHA, in muscles compared with the control birds and other experimental groups. In line with the above, we found that the diet with FO most effectively increased the concentration ratio of n-3LPUFA to SFA and Δ4-desaturase indexes ($\Delta 4_{index}$), whereas most efficiently decreased the elongase index (Table 4). Indeed, dietary FO was reach in n-3LPUFA (like C20:5n-3, C22:5n-3 or C22:6n-3; Table 1), consequently, the diet enriched in FO stimulated the accumulation of n-3LPUFA, especially C22:6n-3 in breast muscles (Table 4) as well as in thigh muscles of female and male chickens (Rozbicka-Wieczorek et al., 2014).

Influence of the experimental rations on the yield of PUFA peroxidation in breast muscles

The influence of the experimental diets on the concentration of MDA and the MDA indexes of breast muscles are summarized in Table 5. The addition of SeY to the diet most effectively decreased the concentration of MDA as well as values of the peroxidation index (MDA_{index}) and our proposed original MDA index ($^{simple}MDA_{index}$) in breast muscles of male chickens and especially female chickens. Above, we found that dietary extra SeY also most efficiently decreased the concentrations of PUFA (Table 5) and MUFA (Table 3).

Considering the above, we argued that dietary SeY significantly decreased the oxidative stress in breast muscles as dosed SeY stimulated the β-oxidation of fatty acids (including PUFA and MUFA) as well as stimulated the biosynthesis of Se-proteins with antioxidant, chemopro-tective and anti-inflammatory properties (Rayman, 2004; Tapiero et al., 2003; Yu et al., 2008). Moreover, the results summarized in Table 5 documented also that dietary SeVI or SeY more effectively reduced the oxidative stress in breast muscles of female chickens than in muscles of male chickens. We suggest that this effect of dietary SeVI or SeY, may be due to the higher concentration of anti-inflammatory n-3PUFA, including n-3LPUFA, in breast muscles of female chickens compared with muscles of male chickens (Tables 3 and 4).

On the other hand, FO added to the diet increased values of $^{simple}MDA_{index}$ and MDA_{index} indexes in breast muscles in comparison with the control chickens; indeed, dietary FO revealed a negligible effect on the concentration of MDA, while decreased the concentration of PUFA in muscles compared with the control birds. In contrast, SeVI added to the diet significantly decreased the concentration of MDA and values of $^{simple}MDA_{index}$ and MDA_{index} indexes in breast muscles of female chickens, whereas increased those in muscles of male chickens compared with the control birds. Interestingly, the effect of dietary Lyc on the concentration of MDA and indexes also depend upon the gender of chickens. Indeed, the diet containing Lyc more effectively reduced the oxidative stress in breast muscles of male chickens than in muscles of female chickens. Hence, we suggest that this effect of dietary Lyc may be due to the higher concentration of anti-inflammatory n-3PUFA, including n-3LPUFA, in breast muscles of male chickens compared with muscles of female chickens (Tables 3 and 4). Moreover, the diet enriched in Lyc increased values of $^{simple}MDA_{index}$ and MDA_{index} indexes in breast muscles of female chickens, whereas decreased in muscles of male chickens in comparison with the control birds.

According to the above and all of the results summarized in Table 5, we documented that our proposed original $^{simple}MDA_{index}$ (calculated as: $^{simple}MDA_{index}$ = MDA/(FA-PUFA); Table 5) is the best indicator of the yield of PUFA peroxidation in tissues of living organisms; $^{simple}MDA_{index}$ takes into consideration the MDA concentrations as well as the concentration of PUFA and the presence of antioxidants in the examined samples. Indeed, the value of this index more significantly depend on the concentrations of MDA and PUFA than the values of MDA_{index} (calculated as: MDA_{index} = (MDA + PUFA)/PUFA).

Conclusion

Diets enriched in Se (as SeVI or SeY) or Lyc can be regularly used to increase the concentration of Lyc or Se (as the antioxidants) in the body of chickens without adversely influencing the performance of male and female chickens. Moreover, dietary SeY most effectively increased the oxidative stability of breast muscles, whereas decreased the concentration of FA in muscles of birds (that is, dietary SeY increases the leanness of breast muscles). Thus, we concluded that dietary SeY stimulated the repartition in breast muscles (that is, increased the level of proteins, while decreased the content of fat in breast muscles of chickens). The addition of FO to the diet most efficiently increased the concentration of n-3LPUFA and the concentration ratio of n-3LPUFA to SFA (n-3LPUFA/SFA), whereas most effectively decreased the concentration ratio of n-6PUFA to n-3PUFA (n-6PUFA/n-3PUFA) in muscles. Considering the above, we argue that dietary FO changes in the profiles of fatty acids in muscles of chickens that are beneficial to human health. Further investigations are necessary to determine if diets containing other chemical form of selenium compounds (like selenite or Se-cysteine), other vegetable oils or the higher concentrations of Lyc induce changes in the profiles of fatty acids in breast and thigh muscles and adipose tissues of chickens that are beneficial to human health.

Conflict of Interests

The author(s) have not declared any conflict of interests.

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