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Effect of fasting on lipid metabolism and oxidative stability in fattening chicken fed a diet supplemented with organic selenium

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Abstract

The aim of the study was to assess the effect of organic selenium dietary supplementation on serum and tissue lipid composition after fattening period and after 48 h fasting in fattening chicken. The study was performed in the Ross hybrid line chicken divided into two groups: control group fed standard diet and Sel Plex group fed standard diet supplemented with 0.3 ppm organic selenium. Blood, liver, intestine and adipose tissue samples were taken upon fattening completion and after 48 h fasting. Total cholesterol, high-density lipoprotein (HDL) cholesterol, low-density lipoprotein (LDL) cholesterol, triacylglycerol and lipoprotein concentrations were determined in serum, whereas total and free cholesterol, triacylglycerol, lipid peroxide and selenium concentrations and glutathione peroxidase activity (GSH-Px) were determined in tissue samples. During fattening period, the correlation of organic selenium with lipid metabolism manifested as decreased concentrations of serum triacylglycerols and very-low-density lipoprotein (VLDL) ($P<0.05$), liver triacylglycerols ($P<0.05$), adipose tissue cholesterol ($P<0.05$) and small intestine cholesterol ($P<0.05$), and reduced lipid peroxidation ($P<0.05$). Upon chicken exposure to 48 h fasting, organic selenium supplementation resulted in increased free cholesterol concentration in adipose tissue ($P<0.05$), GSH-Px activity in the liver ($P<0.05$), and selenium ($P<0.05$) accumulation in the liver.

Keywords: organic selenium, glutathione peroxidase, lipid peroxides, cholesterols, triacylglycerols

Introduction

Oxidative stress refers to impairment in the oxidative and antioxidative compound balance, which is due to either a reduced intake or synthesis of antioxidative molecules, or to an increased synthesis of oxidative compounds. Various models have been used to investigate the causes and consequences of oxidative stress, e.g., environmental hypothermia (Gradinski-Vrbanac *et al.* 1999) or hyperthermia (Mahmoud & Edens 2003), animal exposure to physical stress (Tsopanakis & Tesserommatis 1990), and restrictive feeding and fasting (Lamošová *et al.* 2004, Milinkovič *et al.* 2007). Fasting is a form of oxidative stress characterised by mobilisation and consumption of lipid stores. Depending on the length of fasting, lipid concentrations

vary according to metabolic properties of a particular tissue and organ (Lamošová *et al.* 2004, Yasuhara *et al.* 1991). Study results have revealed great differences in the effect of fasting on lipid metabolism in humans (Sävendahl & Underwood 1999) and animals (Yasuhara *et al.* 1991).

The intensity of oxidative stress can be monitored by measuring oxidation products or by determination of enzymatic and nonenzymatic antioxidative compounds. Antioxidants protect biological molecules from damages occurring either in physiologic conditions or upon exposure to the action of oxidative compounds. Dietary antioxidants such as vitamins A, C and E, and selenium are an important component of antioxidative protection (Temple 2000, Castillo *et al.* 2001, Kolodziej & Jacyno 2005, Balicka-Ramisz *et al.* 2006). Selenium is taken to the body with food in its organic (selenomethionine, selenocysteine) or inorganic (selenates, selenites) form. Organic selenium has shown significantly better antioxidative properties in both mammals (Vinson *et al.* 1998) and birds (Mahmoud & Edens 2003). The antioxidative action of organic selenium is elicited through selenoamino acid incorporation into selenoproteins. Selenoproteins, i.e. glutathione peroxidase (GSH-Px) and 5'-diodinase enzymes, influence lipid metabolism in both physiologic and stress conditions. Glutathione peroxidase eliminates hydrogen peroxide and oxidation-altered lipids from tissues, while protecting low-density lipoproteins (LDL) in serum from oxidative changes (Holben & Smith 1999, Köhrle *et al.* 2000). The enzyme 5'-deiodinase catalyses synthesis of thyroid hormones, which regulate expression of LDL receptors and apolipoprotein B, thus playing a role in maintaining cholesterol level in the circulation (Dhingra & Bansai 2005).

Studies in hamsters have demonstrated that dietary supplementation with organic selenium reduces the rate of lipoprotein oxidation and cholesterol concentration in serum (Vinson *et al.* 1998). Iizuka *et al.* (2001) found selenium supplementation to decrease serum triacylglycerol and cholesterol concentrations, as well as hepatic triacylglycerol and cholesterol levels in rats fed cholesterol-rich diet, whereas Dhingra & Bansal (2005) report on an increased GSH-Px activity and restitution of selenium stores in rat liver.

Some specific features of lipid metabolism in birds, such as lipid synthesis mostly in the liver, intestinal resorption in the form of portomicrons, and specificities of apolipoprotein structure make birds an interesting research model. As fasting is accompanied by changes in lipid metabolism and selenium supplementation influences lipid metabolism in physiological and stress conditions, the aim of the present study was to assess the effect of dietary supplementation with organic selenium on lipid metabolism in chicken serum and some tissues upon completion of fattening period and after 48 h fasting.

Material and methods

Animals

The study was performed on the Ross heavy line hybrid chicken of both sexes (n=100). Animals were bred on the floor in the experimental rooms of Department of Physiology and Radiobiology Faculty of Veterinary Medicine University of Zagreb, Croatia. During the first seven days animals were fed a commercial starter diet (crude protein 22 %, metabolic energy (ME) 12.85 MJ/kg, Na selenite 0.15 ppm), then were randomised into two groups:

control group of animals that continued receiving this diet, whereas experimental (Sel Plex: Alltech, Lexington, KY, USA) group animals received the same diet supplemented with 0.3 ppm organic selenium in the form of selenomethionine from the Sel Plex product. In the subsequent fattening period, animals were fed a standard grower diet (crude protein 20 %, ME 12.85 MJ/kg, Na selenate 0.15 ppm) and finisher diet (crude protein 16 %, ME 12 MJ/kg, Na selenate 0.15 ppm), whereby the same amount of selenium was added to both diets given to control group (Table 1). Both groups were offered feed and water *ad libitum* until 42 days of age.

Table 1
Diet composition according to manufacturer's specification (Sljeme Animal Feed Industry, Sesvete, Croatia)

	Starter diet	Grower diet	Finisher diet
Nutrient content			
Protein min., %	22.00	20.00	16.00
Fat, %	5.00	5.00	5.00
Humidity, %	13.0	13.5	13.5
Raw fiber, %	5.00	5.00	5.00
Calcium, %	1.00	1.00	1.0
Phosphorus min., %	0.60	0.60	0.60
Sodium max., %	0.16	0.16	0.15-0.2
ME, MJ/kg	12.85	12.85	12
Added per kg feed:			
Manganese min., mg	100	100	50
Na-selenate min., mg	0.15	0.15	0.15
Zinc min., mg	80	80	50
Vitamin A min., IU	15 000	14 000	12 000
Vitamin D3 min., IU	5 000	5 000	2 000
Vitamin E min., mg	50	50	25
Vitamin B min., mg	8	8	4
Coccidiostatic, mg	-	60	-

ME: metabolic energy

At the age of 42 days, blood was sampled by wing vein venepuncture without anticoagulant in both experimental and control animals, then the animals were sacrificed. Immediately upon sacrifice and bleeding, liver, duodenum and adipose tissue were extracted, washed in cold 0.9 % NaCl, wiped with tissue paper, and placed on ice. Upon weighing, organs were stored at −80 °C until analysis. The rest of animals were exposed to 48 h fasting, with water access *ad libitum*. After 48 h fasting, sampling and sample processing for analysis were repeated.

Processing of blood and tissue samples

Blood was centrifuged at 1 500 g for 15 min at 20 °C. The concentrations of total cholesterol, HDL-cholesterol, LDL-cholesterol and triacylglycerols were determined in serum, whereas lipoprotein fractions were separated by electrophoresis.

Liver and intestine samples were homogenised in 0.14 mol/L KCl on ice with teflon-glass Schütt homogen^{plus} homogeniser (Schütt Biotec, Göttingen, Germany) at 2 800 rpm. The tissue mass to buffer ratio was 1:5 (w/v). The homogenates were centrifuged at 10 500 rpm

for 15 min at 4 °C, then GSH-Px activity was determined in the supernatant, lipid substances were extracted from the supernatant (Folch *et al.* 1957), and concentrations of triacylglycerols, total and free cholesterol, and lipid peroxides were determined. Selenium concentration was determined in the liver before and after fasting, and in small intestine before fasting.

Adipose tissue samples were homogenised in a chloroform and methanol mixture (2:1) (Folch *et al.* 1957); the tissue mass to mixture ratio was 1:5 (w/v). Tissues were homogenised for 60 s at room temperature. The homogenates were centrifuged at 3 000 rpm for 10 min at 25 °C, then the concentrations of triacylglycerols, total and free cholesterol, thiobarbituric and reactive substances (TBARS) were determined.

Biochemistry

Total cholesterol, HDL-cholesterol, LDL-cholesterol and triacylglycerol concentrations and GSH-Px activity were determined by use of Randox reagents (Randox Laboratories, Crumlin, UK) on a SABA 18 biochemistry analyser (AMS, Rome, Italy). The concentration of free cholesterol was determined upon extraction with isopropanol and precipitation with 0.5 % alcohol solution of digitonin (Sobel & Mayer 1945).

The concentration of lipid peroxides was determined by the method of Trotta *et al.* (1982) on a Thermospectronic Helios delta spectrophotometer (Unicam, Cambridge, UK). Serum lipoproteins were separated by electrophoresis on celogel strips, while their relations and absolute concentrations of lipoprotein fractions were determined on a Glob-al Scan densitometer (Malta Chemetron, Milan, Italy).

Selenium concentration in homogenates was measured by electrothermal method of atomic absorption spectrophotometry (Matek & Blanuša 1998).

The concentrations or activity of the study parameters were expressed *per* gram of tissue.

Statistical data processing

Results were statistically processed by use of the Statistica 7.1 software (StatSoft Inc., Tulsa, OK, USA) and expressed as $\chi \pm SEM$. The significance of differences was assessed by Student's t-test in case of normal distribution (Shapiro-Wilk's test), whereas Mann-Whitney U test was employed if null hypothesis was rejected. The correlation between study parameters was tested by use of the same statistical software.

Results

Adipose tissue

Results obtained by determination of lipid concentrations in chicken adipose tissue upon fattening completion and after 48 h fasting are presented in Table 2. Upon completion of the fattening period, total cholesterol concentration in adipose tissue of Sel Plex group was statistically significantly lower as compared with the values recorded in control group ($P<0.05$). After 48 h fasting, a decrease in total and free cholesterol concentrations, and reduced lipid oxidation were recorded in control group ($P<0.05$ all). In this phase, dietary supplementation with organic selenium led to an increase in free cholesterol concentration ($P<0.05$).

Table 2
Lipid concentrations in adipose tissue of control and Sel Plex group chicken upon fattening completion and after 48 h fasting (mean ± SEM)

	After fattening period		After 48 h fasting	
	control	Sel Plex	control	Sel Plex
Total cholesterol, mmol/g tissue	244.32±24.03	118.59±13.58•	98.73±5.99*	112.17±10.18
Free cholesterol, mmol/g tissue	2.12±0.22	1.84±0.23	1.26±0.12*	2.63±0.28**•
Triacylglycerols, mol/g tissue	0.91±0.08	1.01±0.06	1.00±0.07	0.86±0.08
Lipid peroxides, µmol/g tissue	0.57±0.13	0.78±0.33	0.24±0.08*	0.25±0.07

*significant differences between control groups (P<0.05), **significant differences between Sel Plex groups (P<0.05), •significant differences between control and Sel Plex groups (P<0.05)

Liver

Liver concentration of triacylglycerols upon fattening completion was lower in Sel Plex group as compared to control group (P<0.05) (Table 3). After 48 h fasting, an increase in free cholesterol concentration and GSH-Px activity in the liver was observed in both Sel Plex and control groups of animals, along with a decrease in triacylglycerol and selenium concentrations in control group (P<0.05). In addition, fasting resulted in higher GSH-Px activity and higher liver concentrations of selenium and lipid peroxides in control group as compared with Sel Plex group (P<0.05) (Table 3).

Table 3
Lipid concentrations and GSH-Px activity in the liver of control and Sel Plex group chicken upon fattening completion and after 48 h fasting (mean ± SEM)

	After fattening period		After 48 h fasting	
	control	Sel Plex	control	Sel Plex
Total cholesterol, mmol/g tissue	18.67±1.14	19.62±0.059	20.55±0.91	18.29±0.67
Free cholesterol, mmol/g tissue	8.41±0.59	8.44±0.21	12.63±0.96*	10.65±0.26**
Triacylglycerols, mmol/g tissue	0.13±0.01	0.10±0.01•	0.10±0.01*	0.11±0.01
GSH-Px, U/g tissue	12 426±512	11 774±535	16 360±654*	18 315±501•**
Lipid peroxides, µmol/g tissue	4.22±0.37	3.55±0.28	4.70±0.16	4.89±0.15**
Selenium, µg/g tissue	302.64±28.36	283.54±12.81	159.34±10.68*	302.19±24.10•

*significant differences between control groups (P<0.05); **significant differences between Sel Plex groups (P<0.05); •significant differences between control and Sel Plex groups (P<0.05)

Small intestine

Lipid, lipid peroxide and selenium concentrations, and GSH-Px activity in the chicken small intestine are shown in Table 4. Upon fattening, small intestine concentrations of total cholesterol and lipid peroxides were lower in Sel Plex group as compared to control group (P<0.05). Forty-eight hour fasting resulted in increased small intestine concentration of total cholesterol in control group, and increased free cholesterol and triacylglycerol concentrations and decreased GSH-Px activity in both control and Sel Plex groups (P<0.05). After 48 h fasting, the activity of GSH-Px was higher in Sel Plex group as compared to control group (P<0.05).

Table 4
Lipid concentrations and GSH-Px activitiy in small intestine of control and Sel Plex group chicken upon fattening completion and after 48 h fasting (mean ± SEM)

	After fattening period		After 48 h fasting	
	control	Sel Plex	control	Sel Plex
Total cholesterol, mmol/g tissue	25.13±0.67	23.33±0.57•	26.99±1.39	28.73±1.37**
Free cholesterol, mmol/g tissue	8.98±0.26	8.66±0.22	13.67±1.38*	15.99±1.21**
Triacylglycerols, mmol/g tissue	0.06±0.01	0.07±0.01	0.11±0.01*	0.12±0.01**
GSH-Px, U/g tissue	5 000±300	4 746±201	3 469±136*	3 871±116•**
Lipid peroxides, µmol/g tissue	8.78±0.41	6.77±0.36•	6.85±0.39	7.36±0.41
Selenium, µg/g tissue	120.56±9.37	109.59±14.32	–	–

*significant differences between control groups ($P<0.05$), **significant differences between Sel Plex groups ($P<0.05$), •significant differences between control and Sel Plex groups ($P<0.05$), –: concentration not determined

Serum

Serum lipid and lipoprotein concentrations are shown in Table 5. Dietary supplementation with organic selenium resulted in lower triacylglycerol and VLDL concentrations upon fattening completion ($P<0.05$). Forty-eight hour fasting led to a decrease in serum concentrations of triacylglycerols and VLDL in both Sel Plex and control groups, decreased serum HDL concentration in control group, and increased serum LDL concentration in Sel Plex group ($P<0.05$ all). After 48 h fasting, comparable concentrations of two high-density lipoprotein fractions, HDL and HDL2, were found in both Sel Plex and control groups (Table 5).

Discussion

Results of this study pointed to a correlation of organic selenium and lipid metabolism in chicken serum and some tissues upon completion of fattening period and their stability against stress induced by 48 h fasting.

Lipid metabolism and oxidative stability upon fattening completion

Liver is the main site of lipid synthesis in birds (Hermier 1997). Synthesised triacylglycerols and cholesterol incorporated in lipoprotein particles are transported from the liver to the circulation and peripheral tissues (Tarugi *et al.* 1989), while triacylglycerols are in part stored in hepatocytes (Hermier 1997). In the present study, organic selenium dietary supplementation led to lower triacylglycerol concentrations in chicken liver upon fattening completion, which is consistent with the findings reported by Asha & Indira (2004) in guinea pigs. Decreased hepatic triacylglycerol concentrations recorded in the present study were probably due to a reduced hepatic synthesis of triacylglycerols and VLDL, as also demonstrated by their lower serum concentrations (Table 5).

The majority of triacylglycerols (Gibbons *et al.* 2000) and cholesterol (Angel & Farkas 1974) are deposited in adipose tissue in both mammals and birds (Hermier 1997). Adipose tissue is known to contain lowest levels of selenium (Ullrey 1987), thus the effect of selenium on triacylglycerol and cholesterol metabolism in adipose tissue is likely to result from its influence on lipid synthesis and transport outside adipose tissue. In the present study, dietary supplementation with organic selenium resulted in decreased cholesterol deposition in the chicken adipose tissue.

Table 5
Lipid concentrations in serum of control and Sel Plex group chicken upon fattening completion and after 48 h fasting (mean ± SEM)

	After fattening period		After 48-h fasting	
	control	Sel Plex	control	Sel Plex
Serum lipids				
Total cholesterol, mmol/L	4.81±0.21	5.04±0.29	5.27±0.20	5.03±0.15
HDL-cholesterol, mmol/L	0.89±0.04	0.98±0.05	0.97±0.03	0.91±0.03
LDL-cholesterol, mmol/L	4.42±0.20	4.65±0.20	4.86±0.19	4.74±0.12
Triacylglycerols, mmol/L	0.72±0.04	0.59±0.03•	0.28±0.02*	0.30±0.03**
Serum lipoproteins				
VLDL, g/L	1.30±0.15	0.87±0.07•	0.54±0.17*	0.35±0.07**
LDL, g/L	1.40±0.12	1.20±0.13	1.77±0.13	1.62±0.07**
HDL, g/L	3.74±0.36	4.44±0.08	3.27±0.22	3.58±0.12**
HDL 1, g/L	-	-	2.71±019	3.03±0.11
HDL 2, g/L	-	-	0.57±0.08	0.55±0.07

*significant differences between control groups ($P<0.05$), **significant differences between Sel Plex groups ($P<0.05$), •significant differences between control and Sel Plex groups ($P<0.05$)

Besides resorption, the intestine is also involved in lipid and lipoprotein metabolism (Green & Glickman 1981). Decreased cholesterol concentration in the small intestine of chicken fed a standard diet supplemented with organic selenium may be due to either reduced cholesterol resorption or to faster elimination of resorbed cholesterol in the form of lipoproteins from enterocytes (Green & Glickman 1981).

Glutathione peroxidase activity depends on selenium concentration in the body (Daun & Akesson 2004). In our study, dietary supplementation with organic selenium during fattening period had no effect on this enzyme activity or on hepatic and intestinal selenium concentration upon fattening completion, which is in contrast with the findings reported by Cheng *et al.* (1997) in mouse liver and intestine, and by Arai *et al.* (1994) and Mahmoud & Edens (2003) in poultry liver. Upon fattening completion, low intensity lipid peroxidation in the intestine was observed in the group of animals having received organic selenium, probably due to antioxidative properties of selenomethionine (Briviba *et al.* 1996).

Lipid metabolism and oxidative stability after 48 h fasting

Animal exposure to fasting leads to species-specific mobilisation and consumption of energy stores (Yasuhara *et al.* 1991, Sävendahl & Underwood 1999, Peebles *et al.* 2004). In the present study 48 h fasting led to a decrease in triacylglycerol concentration in both groups of chicken, which is consistent with the findings reported by Lamošová *et al.* (2004) and Didier *et al.* (1983) in serum of Japanese quail. As VLDL are the main transport form of triacylglycerols in serum, in our study a reduced serum concentration of triacylglycerols was accompanied by lower VLDL concentration after fasting, which is consistent with the reports by Hermier *et al.* (1984) in chicken and Lien *et al.* (2005) in duck. Furthermore, a reduced serum VLDL concentration was associated with an increase in serum LDL concentration in experimental group of chicken, which could be explained by enhanced VLDL breakdown and triacylglycerol consumption as a source of energy.

Triacylglycerols stored in the liver can be mobilised from the liver in case of enhanced energy demands such as fasting (Lamošová *et al.* 2004) and laying (Hermier 1997). In the present study, it was observed in the liver of chicken on standard diet after fasting. Similar results are reported by Andriamampandy *et al.* (1996), who consider the decrease in hepatic triacylglycerols and serum beta-hydroxybutyrate to be a sign of transition from the second to the third fasting stage, i.e. body protein catabolism. In our study, protein breakdown for energy supply could be presumed to have been delayed in order to maintain the hepatic level of triacylglycerols in Sel Plex group stable after fasting.

After 48 h fasting, small intestine triacylglycerol concentration was increased in both groups of chicken, which could have been related to the presence of triacylglycerol pool in enterocytes as a triacylglycerol store during fasting period (Mansbach & Nevin 1998).

Organic selenium in the form of selenomethionine and selenocysteine is incorporated into antioxidative compounds and selenoproteins, which serve as a store and source of selenium in stress conditions (Tapiero *et al.* 2003), as also demonstrated in the present study. Selenium concentration was significantly higher in the liver of chicken fed a selenium-supplemented diet as compared to control group without selenium supplementation. In addition, there was negative correlation between selenium and total cholesterol in the liver of Sel Plex group after 48 h fasting ($r=-0.8174$; $P<0.05$) probably due to a reduced cholesterol synthesis in the liver.

Fasting leads to increased serum cholesterol concentration (Lamošová *et al.* 2004, Klauda & Zilversmit 1975, Sävendahl & Underwood 1999) due to cholesterol mobilisation from adipose tissue (Swaner & Connor 1975). In the present study, the level of cholesterol in adipose tissue of the Sel Plex group did not change with fasting, thus preventing serum cholesterol concentration to increase due to selenium supplementation.

As significant changes were recorded in adipose tissue concentrations of total and free cholesterol in both groups of chicken after 48 h fasting without any changes in triacylglycerols, it could be postulated that various lipid components in adipocytes vary independently with fasting (Angel & Farkas 1974).

The increase in small intestine cholesterol concentration recorded in both groups of chicken after 48 h fasting was probably consequential to cholesterol resorption from the bile, which is rich in free cholesterol that is easily resorbed (Wilson & Rudel 1994) because esterified dietary cholesterol accounts for 65 % and biliary cholesterol for 35 % of the cholesterol found in intestinal lumen.

The intensity of lipid peroxidation in chicken blood is reduced with fasting (Piršljin *et al.* 2006), whereas in the present study it was increased in the liver of chicken fed a selenium-supplemented diet. The increase in lipid peroxidation concentration was accompanied by an increase in GSH-Px activity in both groups because liver is the main site for elimination of oxidatively damaged lipid molecules transported to the liver by lipoproteins. After fasting, intestinal GSH-Px activity was reduced in both groups, however, with significantly higher values recorded in Sel Plex group. The reasons for this reduction could probably be found in the lack of feed breakdown and of enzyme induction by dietary peroxides, and because the intestine is a source of extracellular GSH-Px, which is necessary for defense against oxidative stress provoked by fasting (Tham *et al.* 1998).

In conlusion, organic selenium dietary supplementation influenced the composition and oxidative stability of lipids in serum and tissues of chicken upon fattening completion and led to the following:

- decrease in serum and liver triacylglycerol concentrations upon completion of the fattening period, probably due to a reduced hepatic triacylglycerol and VLDL synthesis;
- decrease in adipose tissue and intestinal cholesterol concentrations due to reduced cholesterol stores and resorption;
- and reduced intensity of lipid peroxidation because of its antioxidative properties.

The effect of organic selenium was also recorded after fasting period and resulted in the following:

- increased free cholesterol concentration in adipose tissue, possibly suggesting selenium involvement in intracellular changes in the total to free cholesterol ratio in stress conditions;
- increased selenium concentration in the liver because of its improved resorption and bioavailability;
- and increased GSH-Px activity in the liver and intestine in response to fasting-induced oxidative stress.

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