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EFFECT OF RESVERATROL ON SELECTED BIOCHEMICAL PARAMETERS IN RATS FED HIGH FRUCTOSE DIET

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ABSTRACT

Background. High consumption of food products rich in rapidly digested starch, saccharose, glucose and fructose, animal fat and low physical activity are the major risks of overweight, obesity and cardiovascular diseases. The aim of this study was to evaluate the effect of resveratrol on the activity of selected enzymes, heme oxygenase-1 (Hmox1) and glutathione reductase (Gsr) gene expression, as well as fatty acids composition in the visceral adipose tissue in rats fed a high fructose diet.
Material and methods. Laboratory male Wistar rats (n = 30) were fed for four months with the high fructose diet (HF diet) (63% w/w) based on American Institute of Nutrition diet (AIN-93G diet). Group I was fed with the AIN-93G diet (NFC – negative control group), group II – high fructose diet (HF), group III – HF with 0.02% of resveratrol, group IV – high fructose with 0.04% of resveratrol and group V – high fructose with 0.06% of resveratrol. At the end of the experiment fasted rats were anaesthetized, blood and livers were collected and stored until analyses.
Results. Concentration of glucose was significantly increased in the blood of rats fed with the HF containing 0.02% and 0.06% of RSV in diets compared to animals fed with the HF diet. Activity of heme oxygenase-1 (HO-1) was significantly lower in the serum of rats fed with the HF diet containing 0.06% RSV compared to the serum of rodents fed with the negative control diet and the HF diets. Gsr and Hmox1 gene expression was significantly increased in livers of rodents fed with HF containing 0.04% RSV compared to the HF group.
Conclusions. Resveratrol may protect from damage caused by high fructose intake in rats.

Key words: fructose, HO-1, gene expression, rats, resveratrol

INTRODUCTION

Nutrients and bioactive food components – functional substances found in food, have a direct or indirect effect on scavenging of free radicals and the structure and/or expression of genes. Therefore, an unbalanced diet (malnutrition or excess nutrients intake) is a risk factor for many diseases [Kaput 2004].

In current nutritional guidelines intake of no more than 10% energy from added sugars is recommended. Albeit in many countries intake of simple carbohydrates with average daily diets is higher than recommended values [WHO/FAO 2003]. It has been reported that high consumption of food products rich in rapidly digested starch, saccharose, glucose and fructose (usually added to food as a syrup), animal fat and low physical activity are the major risks of overweight, obesity and cardiovascular diseases [Rebello et al. 2012, WHO/FAO 2003, Suliburska 2013]. It has been also documented that exceeded addition of fructose to experimental diets of rodents, increases oxidative stress, synthesis of fatty acids, triacyloglycerols

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in liver and it increases the risk of cardiovascular diseases and metabolic disorders [Abdullah et al. 2009, Bagul et al. 2012, Bizeau and Pagliassoti 2005, Rebello et al. 2012].

Reactive oxygen species (ROS) in the body can cause damage of many structures (proteins, lipids, DNA) and oxidative stress. Antioxidants and other chemical bioactive compounds detoxify ROS and prevent from damage of the cellular macromolecules and organelles through various mechanisms [Gramza-Michałowska and Korczak 2013, Lee et al. 2004, Sikora and Bodziarczyk 2013]. Several mechanisms that defend from free radicals exist in the human body (for example antioxidant enzymes). Heme oxygenase-1 (HO-1) is an enzyme which is now accepted as the mediator of cyto- and tissue protection against harmful substances (free radicals, UV, heavy metals, heme, nitric oxide and pro-inflammatory cytokines). The synthesis of this enzyme is induced by many factors (oxidative stress, cytotoxic agents and infection) [Immenschuh and Ramadori 2000, Ryter et al. 2006, Schmidt et al. 2012].

Since resveratrol has been identified in wine, the "French Paradox" is explained by it. The French population has lower incidences of heart attacks, even though they consume more food products rich in saturated fat, especially animal origin, than other populations [Palsamy and Subramanian 2008]. In addition, previously published studies have shown that resveratrol added to the animals' diets in different doses decreased oxidative stress, improved function of the liver, lowered the total cholesterol, HDL cholesterol and decreased gene expression involved in lipid and phospholipid metabolism with exception of the Peroxisome proliferator activated receptor alpha (Ppara) [Miattelo et al. 2005, Ahn et al. 2008, Rivera et al. 2009].

The objective of this study was to investigate the effects of resveratrol on the activity of selected enzymes, some biochemical parameters, Hmox1 and Gsr gene expression, and fatty acids composition in the visceral adipose tissue in Wistar rats fed the diet containing 63% of fructose.

MATERIAL AND METHODS

Animal study

Laboratory male Wistar rats were fed with the high fructose diet (63% fructose). Rats were divided into 5 groups (n = 6) and fed experimental diets containing different levels of RSV. All procedures associated with the animal study were described previously [Kopeć et al. 2013]. Experimental procedures were complied with the Polish Ethical Standards. Rodents were acclimatized for one week. After it animals were randomly divided into five experimental groups (n = 6) and fed with modified diets based on AIN-93G diets [Reeves 1997]. The negative fructose control diet (NFC) was composed of the following ingredients: corn starch (53%) (Cargill, Bielany Wrocławskie, Poland), casein (20%), saccharose (10%) (Kazeina Polska, Legionowo, Poland), soybean oil (7%), cellulose (5%), mineral mix (3.5), vitamin mix (1%), choline chloride and *tert*-butylhydroquinone. Vitamin and miner mixture were prepared accordingly Reeves [1997]. Group I was fed with the AIN-93G diet (NFC – negative control group), group II – high fructose diet (HF), group III – HF with 0.02% of resveratrol (HF + 0.02% RSV), group IV – high fructose with 0.04% of resveratrol (HF + 0.04 RSV) and group V – high fructose with 0.06% of resveratrol (HF + 0.06% RSV).

On the last day of the experiment fasted rats were anaesthetized and blood was collected to obtain the serum. The livers and adipose tissues were collected and stored in –80°C until analyses.

Enzymes activity, glucose, uric acid and creatinine level

The activity of aspartate aminotransferase (AST) and alanine aminotransferase (ALT) in the serum was measured using the Alpha Diagnostic kits (Alpha Diagnostic, Warsaw, Poland; cat no. A6661-050, A6624-050 respectively). The activity of HO-1 in the serum was measured in accordance to the Turcanu et al. [1998] method. Results were shown as the concentration of bilirubin. The activity of glutathione reductase was measured in the serum of rats with a kit (cat no. GR 2368, Randox Laboratories, Crumlin, United Kingdom). The level of glucose was measured in whole blood with glucometer (Accu-chek, Roche Diagnostic, Mannheim, Germany).

The concentration of C-reactive proteins (CRP) was measured using the rat CRP Elisa kit (cat no. RH 951CRP01R Biovendor, Brno, Czech Republic). Uric acid concentration was determined with commercially available kit (cat no. 2-208; PZ Cormay, Lublin, Poland).

Gene expression

mRNA was isolated from livers with a commercially available kit (cat no. 610.12 Invitrogen Life Technologies, Oslo, Norway). The concentration of mRNA was measured by a spectrophotometer at absorbance 260 nm and 280 nm (Nanodrop 2000, Thermo Scientific Electron Corporation, Wilmington, Delaware, USA). For cDNA synthesis mRNA was reverse transcribed with the use of SuperScriptÒ VILO cDNA Synthesis Kit (cat no. 11754-050 Invitrogen Life Technologies, Oslo, Norway). cDNA was subjected to real time PCR in a reaction of a mixture containing TaqMan Gene Expression Master mix and primers with fluorescent marked starters (Invitrogen, Life Technologies, Oslo, Norway). The thermal profile of the PCR reaction included initial denaturation (15 min at 95°C), 40 amplification cycles of denaturation (1 s at 95°C), annealing (20 s at 60°C), and elongation (20 s at 72°C) with the use of the following equipment Applied Biosystems 7900HT Fast Real-Time PCR System (Applied Biosystem, Foster City, California, USA). The expression rates were calculated as the normalized threshold cycle (C_T) difference between a control sample and a sample with the adjustment for the amplification efficiency relative to the expression level of the housekeeping gene *Sp1*.

Fatty acids composition

The fatty acids compositions in the adipose tissue were determined with the use of gas chromatography. Adipose tissue (20 mg) was saponificated in 0.5 mol·l^{-1} KOH for 15 min in 60°C. After this fatty acids were methylated with BF_3 (14% w/w in methanol) for 15 min in 60°C. All chemicals used for the analysis of fatty acids were obtained from Chempur, Piekary Śląskie, Poland). Analysis of the profile of fatty acids was conducted with the use of gas chromatography Shimadzu QP5050A (Shimadzu, Kyoto, Japan) equipped with a column SP-2560 (100 m × 0.25 mm × 0.25 μm, Supelco, Bellefonte, Pennsylvania, USA). Results were interoperated as the percentage of fatty acids in the adipose tissue of all fatty acids.

Statistical analysis

The data were presented as mean ±standard deviation (SD). One-way, non-parametric analysis of variance (Statistica v. 6.1, StatSoft Inc., Tulsa, Oklahoma, USA) was applied for testing the differences between experimental treatments. The Kruskal-Wallis test was used for the identification of statistically significant differences at a level of $p < 0.05$.

RESULTS AND DISCUSSION

The activity of AST and ALT was not affected by different dietary treatments. However it was found that AST activity tended to be lower ($p < 0.07$) in serum of animals fed with diet containing 0.02% and 0.04% of RSV compared to the serum of rodents fed with HF diet. In published papers there is a lack of information concerning the effect of long term treatment of RSV with high fructose diet on ALT, AST activity. It can be suggested that RSV protects hepatocytes from damage. It was reported that activity of these enzymes is affected by RSV in animal studies (with various animal models) treated with different diets. Juan et al. [2002] reported that RSV in high dose (20 mg·kg^{-1} per day) did not affect the activity of ALT and AST in serum rats. Hamadi et al. [2012] found that RSV added to the diet of streptozotocin induced diabetes in rats, decreased activity of AST and ALT in the serum compared to control rats. These authors suggested that RSV decreased oxidative stress and inflammation in livers caused by hyperglycemia. Additionally in some studies RSV significantly decreased ALT and/or AST activity in animal models (mice or rats) with induced by different factors hepatotoxicity or hepatitis (acetaminophen, CCL_4, naphtalene, lipopolysaccharide) [Bujanda et al. 2006, Şener et al. 2006, Farghali et al. 2009, Ebyl et al. 2006, Şehirli et al. 2008].

Four months administration of RSV with the HF diet significantly increased the blood glucose level in rats fed with 0.02% and 0.06% of RSV compared to blood of rodents fed with the HF diet. It should be noted that glucose level was not significantly different in animas fed with the NCF diet compared to the blood of rodents fed with RSV diets. It has been reported that

high intake of fructose increased glucose production from fructose, and additionally the synthesis of glycogen in liver was increased. This effect is probably connected with reduced insulin resistance and modulation of gene expression involved in fructose metabolism [Bizeau and Pagliassotti 2005, Haring and Harris 2011, Nuttall et al. 2000]. Probably these caused lower level of glucose in rodents fed with the HF diet in our study. The addition of RSV to diets might interrupt glycogen synthesis and more glucose was used as the source of energy. Our results concerning glucose concentration are different to data published by Deng et al. [2008] and Miatello et al. [2005]. Deng et al. [2008] did not report any effect of RSV on the glucose level in rats fed for 15 days and 15 weeks with high cholesterol, high fructose diet. It should be also noted that fructose was administrated in drinking water. Our results are not confirmed either by data published by Bagul et al. [2012]. These authors reported that rats fed for 8 weeks with high fructose diet (60%) with addition of RSV (10 mg·kg^{-1} per day administrated orally) had lower level of glucose compared to the animals fed with the control diet.

We did not find any effect of RSV on the CRP level in the serum rats. It can be suggested that long term administration of RSV decreased oxidative stress and the production of CRP in the liver was not increased. These data are not confirmed by the results published by Bruckbauer et al. [2012]. The authors have found that CRP significantly decreased in obese mice fed low (15.5 mg·kg^{-1} diet) or high dose (225 mg·kg^{-1} diet) of RSV.

Gsr and Hmox1 gene expression was significantly increased in the liver of rodents fed HF + 0.04% RSV compared to the HF group. Additionally, the activity of GSR was significantly increased in the same group of rats compared to rodents fed with HF diet. GSR tended to be higher in rats fed diet with 0.06% of RSV (Table 1). It can be suggested that long term of the RSV treatment increased synthesis of GSR and it protected the liver from the oxidative damage. Probably it was the reason why the activity of HO-1 (reported as the bilirubin concentration) was lower in the serum

Table 1. Selected enzymes activity, biochemical parameters concentration in serum and mRNA Hmox1, Gsr expression in experimental rats

Treatment	NCF	HF	HF + 0.02% RSV	HF + 0.04% RSV	HF + 0.06% RSV
Serum					
AST, U·l^{-1}	155.03 ±43.74^{a}	164.66 ±98.91^{a}	106.00 ±13.00^{a}	95.50 ±21.92^{a}	153.00 ±55.53^{a}
ALT, U·l^{-1}	48.17 ±15.63^{a}	62.33 ±18.24^{a}	56.00 ±20.07^{a}	60.66 ±18.55^{a}	106.5 ±68.76^{a}
Glucose, mg·dl^{-1}	106.0 ±10.8^{ab}	99.0 ±6.6^{a}	112.2 ±7.1^{b}	109.0 ±5.4^{ab}	112.2 ±4.4^{b}
CRP, µg·ml^{-1}	255 ±15^{a}	259 ±26^{a}	268 ±25^{a}	259 ±18^{a}	267 ±12^{a}
HO-1, mmol·l^{-1}	2.37 ±0.87^{a}	2.27 ±0.53^{a}	1.69 ±0.38^{ab}	1.44 ±0.54^{ab}	1.16 ±0.28^{b}
GSR, U·l^{-1}	148 ±67^{a}	61 ±17^{b}	71 ±23^{b}	137 ±25^{a}	116 ±43^{ab}
Uric acid, mmol·l^{-1}	143 ±32^{a}	306 ±77^{c}	219 ±64^{b}	201 ±69^{b}	201 ±31^{b}
Relative gene expression in liver, %					
Hmox1	1.77 ±0.09^{ab}	1.77 ±0.01^{a}	1.80 ±0.01^{ab}	1.89 ±0.05^{b}	1.76 ±0.06^{ab}
Gsr	1.84 ±0.03^{ab}	1.80 ±0.01^{a}	1.83 ±0.01^{ab}	1.98 ±0.06^{b}	1.88 ±0.03^{ab}

Values in column with different letters (a, b, c) are significantly different, $p \leq 0.05$.
NFC – negative fructose control. HF – high fructose diet. HO-1 was expressed as the concentration of bilirubin. Glucose was measured in whole blood. HF + RSV – high fructose diet with addition of resveratrol. RSV – resveratrol, AST – aspartate aminotransferase. ALT – alanine aminotransferase. CRP – C-reactive proteins. HO-1 – heme oxygenase 1. Hmox1 – gene of heme oxygenase 1. Gsr – gene of glutathione reductase.

of rats fed with 0.06% RSV and tended to be lower in groups fed with 0.02% or 0.04% of RSV in presence of fructose. This enzyme removes the prooxidant heme and biliverdin [Foresti et al. 2001]. What is more the expression of Hmox1 was significantly increased in the livers of animals fed 0.04% RSV and tended to be higher in animals fed with 0.02% RSV. High intake of fructose with experimental diet probably increased production of prooxidant heme and RSV would scavenge it. It could also protect hepatocytes from damage, and it resulted in lower level of HO-1 activity in the serum of animals. Hamadi et al. [2012] also have found that RSV decreased synthesis of the bilirubin in STZ diabetic rats. It was reported that HO-1 expression was higher in human lens epithelial cells against H_2O_2 induced stress [Zheng et al. 2010].

The uric acid level was significantly lower in the serum of animals fed different doses of RSV compared to rodents fed the HF diet. The uric acid is the antioxidant but also its high concentration in blood may be a risk factor of cardiovascular diseases and obesity. It has been also reported that HF diet increased the synthesis of uric acid. Hyperuricemia decreases endothelium nitric oxide bioavailbility, it may cause the resistance of insulin [Nakagawa et al. 2006, Kanellis and Kang 2006]. It can be suggested that presence the RSV in the HF diet affected the synthesis of uric acid. These results are similar to the data published

Table 2. Fatty acids composition in adipose tissue of experimental rats

Treatment	NFC	HF	HF + 0.02% RSV	HF + 0.04% RSV	HF + 0.06% RSV
			%		
C12:0	0.07 ±0.004^{a}	0.07 ±0.01^{a}	0.07 ±0.01^{a}	0.072 ±0.01^{a}	0.070 ±0.06^{a}
C14:0	1.30 ±0.16^{a}	1.46 ±0.11^{a}	1.37 ±0.14^{a}	1.56 ±0.23^{a}	1.38 ±0.08^{a}
C15:0	0.16 ±0.02^{a}	0.19 ±0.03^{a}	0.18 ±0.04^{a}	0.22 ±0.04^{a}	0.19 ±0.03^{a}
C16:0	19.3 ±0.3^{a}	19.5 ±0.6^{a}	19.2 ±0.8^{a}	18.6 ±0.9^{a}	18.7 ±0.5^{a}
C16:1	6.60 ±0.9^{a}	5.96 ±0.51^{a}	6.34 ±1.03^{a}	6.88 ±0.57^{a}	6.01 ±0.73^{a}
C17:0	0.15 ±0.01^{ab}	0.16 ±0.02^{ab}	0.14 ±0.02^{a}	0.18 ±0.02^{b}	0.16 ±0.02^{ab}
C17:1	0.15 ±0.01^{a}	0.16 ±0.03^{ab}	0.15 ±0.02^{ab}	0.19 ±0.01^{b}	0.16 ±0.032^{ab}
C18:0	3.37 ±0.15^{ab}	3.59 ±0.55^{ab}	3.04 ±0.28^{a}	3.81 ±0.41^{b}	3.29 ±0.34^{ab}
C18:1	35.1 ±0.7^{a}	38.2 ±0.5^{ab}	38.7 ±1.6^{b}	38.3 ±1.1^{ab}	39.3 ±1.3^{b}
C18:2 (n-6)	30.3 ±1.7^{a}	27.3 ±0.8^{ab}	27.5 ±2.3^{ab}	26.4 ±0.9^{b}	27.5 ±1.9^{ab}
C18:3 (n-6)	0.22 ±0.03^{ab}	0.21 ±0.02^{ab}	0.20 ±0.03^{ab}	0.27 ±0.03^{a}	0.18 ±0.02^{b}
C18:3 (n-3)	2.46 ±0.2^{a}	2.27 ±0.24^{a}	2.26 ±0.34^{a}	2.50 ±0.17^{a}	2.24 ±0.25^{a}
C20:0	0.49 ±0.02^{ab}	0.51 ±0.02^{ab}	0.38 ±0.01^{ab}	0.53 ±0.01^{a}	0.42 ±0.01^{b}
C20:1	0.26 ±0.01^{a}	0.33 ±0.07^{ab}	0.31 ±0.02^{ab}	0.36 ±0.06^{b}	0.30 ±0.04^{ab}
C20:1	0.14 ±0.02^{a}	0.13 ±0.04^{a}	0.13 ±0.02^{a}	0.13 ±0.03^{a}	0.12 ±0.02^{a}
C20:2	0.08 ±0.03^{a}	0.07 ±0.02^{a}	0.08 ±0.03^{a}	0.09 ±0.02^{a}	0.07 ±0.01^{a}
C20:4 n-6	0.28 ±0.03^{a}	0.26 ±0.06^{a}	0.25 ±0.04^{a}	0.28 ±0.06^{a}	0.23 ±0.06^{a}

Values in column with different letters (a, b, c) are significantly different, $P \leq 0.05$.
NFC – negative fructose control. HF – high fructose diet. HF + RSV – high fructose diet with addition of resveratrol. RSV – resveratrol.

by Bagul et al. [2012]. These authors also reported that concentration of uric acid was significantly lower in serum of rats fed with high fructose diet (65% w/w) supplemented with 10 $mg \cdot kg^{-1}$ per day of RSV. Also Deng et al. [2008] reported that RSV decreased concentration of uric acid in the plasma of rats fed high cholesterol high fructose diet.

The addition of 0.02% of RSV to the experimental diet significantly decreased C17:0 and C18:0 in visceral fat compared to fat of rats fed the diet with 0.04% of RSV (Table 2). The percentage of C17:1 was significantly increased in adipose tissue of rats fed 0.04% of RSV in comparison to animals fed with NFC diet. The level of C18:1 was significantly higher in fat of rats fed diets the addition of 0.02% and 0.06% as compared to NFC group. The level of C18:2 (n-6) was significantly lower in visceral fat of rodents fed 0.04% of RSV in comparison to the NFC animals. The concentration of C20:0 and C18:3 (n-6) was significantly decreased in fat of rats fed with 0.06% of RSV in comparison to the fat of rodents fed the HF diet with 0.04% of RSV. The level of C20:1 was significantly higher in the fat of rats fed with 0.04% of RSV compared to adipose the tissue of rodents fed with the NFC diet (Table 2). We also found that the content of C18:1 tended to be lower and the level of C18:2 (n-6) significantly decreased in rats fed with 0.04% RSV. Additionally the concentration of C18:3 (n-6) significantly increased in adipose tissue of the rodents from the same group. It can be suggested that resveratrol activated the elongases and desaturases involved in synthesis these fatty acids, which caused these changes. There is a lack of data in available literature about the fatty acids composition in adipose tissue of rodents fed HF diet with RSV. Abdullah et al. [2009] reported that rats fed a HF diet had higher level of C16:0, C16:1, C18:1n-9 in the liver compared to control rodents. Because of high standard deviation, we found only tendency in changes of these fatty acids. Albeit it has been reported that RSV modulate fatty acids synthesis via inhibition of *de novo* lipogenesis and their oxidation in white adipose tissue (WAT). WAT is the major site where synthesis of the fatty acids occurs. They can be re-esterified to triacyloglicerydes or used for production energy in mitochondria [Cho et al. 2012]. Additionally Ahn et al. [2008] reported that RSV added to the atherogenic diet containing 1% of cholesterol decreased the expression of Apoa4 gene which is required for lipoprotein lipase activation and Mttp gene required for encode of subunits of the triglyceride transfer protein in livers of mice. It can be suggested that in our studies, RSV in dose-dependent manner, changes in metabolism of lipids in the liver caused also the changes in fatty acids metabolism in adipose tissue. What is more it maybe suggested that the dose which affects fatty acids composition is 0.04% of RSV and the changes in fatty acid composition were probably affected by synthesis and oxidation of them. These results suggest that more studies are required to explain these changes.

CONCLUSIONS

Long term administration of RSV with high fructose diet significantly decreased the concentration of HO-1 and uric acid. mRNA gene expression of Gsr and Hmox1 was significantly higher in rodents fed with 0.02% RSV. Resveratrol in dose dependent-manner changed the content selected fatty acids which maybe the results of activation of the enzymes involved in desaturation and elongation. Resveratrol may protect from damage caused by high fructose intake in rats.

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WPŁYW DODATKU RESWERATROLU DO DIETY WYSOKOFRUKTOZOWEJ NA WYBRANE PARAMETRY BIOCHEMICZNE W ORGANIZMIE SZCZURÓW DOŚWIADCZALNYCH

STRESZCZENIE

Wstęp. Zbyt duże, przedłużające się w czasie, przespożycie produktów spożywczych obfitujących w łatwostrawną skrobię, sacharozę, fruktozę i tłuszcze pochodzenia zwierzęcego, a równocześnie mała aktywność fizyczna należą do głównych czynników ryzyka nadwagi, otyłości oraz chorób układu krążenia. Celem niniejszej pracy była ocena wpływu dodatku resweratrolu (RSV) do diety wysokofrukozowej na aktywność wybranych enzymów, ekspresji genów hemowej oksygenazy-1 (Hmox1), reduktazy glutationowej (Gsr) oraz profilu kwasów tłuszczowych w tkance tłuszczowej trzewnej.
Materiał i metody. Szczury stada Wistar, płci męskiej (n = 30), przez cztery miesiące były karmione dietą wysokofruktozową, przygotowaną na bazie diety AIN-93G. Grupa I była żywiona dietą AIN-93G (NFC – grupa kontrolna negatywna), grupa II – dietą wysokofruktozową (HF), grupa III, IV i V – dietą HF z dodatkiem resweratrolu odpowiednio w ilości 0,02%, 0,04% oraz 0,06%. Po czterech miesiącach doświadczenia zwierzęta zostały poddane eutanazji. Od szczurów pobrano krew w celu otrzymania surowicy, wątroby oraz tkankę tłuszczową trzewną. Pobrany materiał został zamrożony.
Wyniki. Stężenie glukozy wzrosło istotnie w surowicy krwi szczurów karmionych dietą wysokofruktozową z dodatkiem RSV w ilości 0,02% oraz 0,06% w porównaniu ze zwierzętami żywionymi dietą wysokofruktozową. Aktywność hemowej oksygenazy-1 zmniejszyła się istotnie w surowicy krwi szczurów żywionych dietą wysokofruktozową z dodatkiem RSV w ilości 0,06% w porównaniu z surowicą krwi gryzoni żywionych dietą AIN-93G. Ekspresja genów Gsr i Hmox1 istotnie wzrosła w wątrobach szczurów żywionych dietą wysokofruktozową z dodatkiem RSV w ilości 0,04% w porównaniu ze zwierzętami karmionymi dietą wysokofruktozową.
Wnioski. Resweratrol może zapobiegać niekorzystnym zmianom ocenianych parametrów biochemicznych w organizmach zwierząt żywionych dietą wysokofruktozową.

Słowa kluczowe: fruktoza, HO-1, ekspresja genów, resweratrol

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