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THE ROLE OF SELECTED PLANT PRODUCTS IN THE PREVENTION OF CADMIUM-INDUCED TOXICITY®

Rola wybranych produktów roślinnych w zapobieganiu toksyczności wywołanej przez kadm®

Research financed by Polish Ministry of Science and Higher Education within founds of Institute of Human Nutrition Sciences, Warsaw University of Life Sciences (WULS), for scientific research

Praca finansowana przez Ministerstwo Nauki i Szkolnictwa Wyższego w ramach utrzymania potencjału badawczego Instytutu Nauk o Żywieniu Człowieka, SGGW w Warszawie

Long-term exposure to cadmium (Cd) leads to the development of numerous diseases in human as well as in animals associated with renal and hepatic injury, respiratory, skeletal and cardiovascular disturbances, as well as visual changes, blindness and hearing loss. Cd was classified as human carcinogen (Group 1) by the International Agency for Research on Cancer. The study discusses the effect of selected plant products on reduced toxicity caused by Cd. Animal studies confirmed that the effect of such products as: turmeric, ginger, garlic, onion, black seeds oil, olive oil, blueberry, green tea, hibiscus was efficient in reducing the Cd level in organs and tissues and had positive effect on the urinary tract and cardiovascular, reproductive, nervous, immune and respiratory system. Consumption of these products in occupational and environmental exposure to Cd appears promising.

Key words: cadmium exposure, oxidative stress, turmeric, ginger, garlic, onion, black seeds oil, olive oil, blueberry, green tea, hibiscus.

Długotrwała ekspozycja na kadm (Cd) ludzi jak i zwierząt, prowadzi do rozwoju wielu zmian patologicznych związanych z uszkodzeniem nerek, wątroby, zaburzeniami w układzie oddechowym, kostnym, sercowo-naczyniowym, a ponadto z zaburzeniami widzenia, ślepotą i niedosłuchem. Cd został zaklasyfikowany przez Międzynarodową Agencję Badań nad Rakiem jako substancja rakotwórcza dla człowieka (Grupa 1). W artykule omówiono potencjalnie korzystny wpływ wybranych produktów roślinnych na zmniejszanie toksyczności wywołanej przez Cd. Badania na zwierzętach wykazały, że spożywanie produktów takich jak: kurkuma, imbir, czosnek, cebula, olej z czarnuszki, oliwa z oliwek, borówka amerykańska, zielona herbata, hibiskus było skuteczne w obniżaniu zawartości Cd w narządach i tkankach oraz korzystnie wpływało na układ krwionośny, moczowy, rozrodczy, nerwowy, odpornościowy i oddechowy. Obiecujące wydaje się zalecanie spożywania tych produktów przy zawodowym i środowiskowym narażeniu na Cd.

Słowa kluczowe: ekspozycja na kadm, stres oksydacyjny, kurkuma, imbir, czosnek, cebula, olej z czarnuszki, oliwa z oliwek, borówka amerykańska, zielona herbata, hibiskus.

INTRODUCTION

Cadmium toxicity in humans

Cadmium (Cd) is a heavy metal which occurs naturally in the Earth's crust. Its content in the environment increases as a result of volcano eruption and thermal rock weathering. Environmental pollution with Cd further derives from human activity associated with metal mining, refining, production of phosphorus fertilizers. Cd is used in numerous technological processes, i.a. for the production of batteries, pigments, coatings and platings, stabilizers for plastics. A group of people facing particular exposure to Cd includes workers dealing with welding and electroplating [4]. Apart from professional exposure, tobacco smoking constitutes another source of exposure to Cd [23, 45]. In the non-smoking population, approximately 90% Cd originates from food [18]. According to EFSA main sources of Cd in the diet are the products typically consumed in greater amounts, such as grains and grain products, vegetables and vegetables products and starchy roots and tubers.

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Long-term exposure to Cd leads to the development of numerous diseases associated with renal and hepatic injury, respiratory, skeletal and cardiovascular disturbances, as well as visual changes, blindness, and hearing loss [16, 26, 47, 50]. It has been also demonstrated that Cd has teratogenic and carcinogenic effect [34, 35, 43, 49].

The toxic effect of Cd results from numerous mechanisms, but the most important are the weakening of the antioxidant system and thus related increase of reactive oxygen species concentration. This leads to induced oxidative stress and intensification of lipid peroxidation, protein oxygenation, DNA damage, and as a result of this cell membrane injury and cell death. Another mechanism of toxic effect of Cd stems from homeostasis disturbance (by forming bonds with sulfur, oxygen and hydrogen ions from sulfhydryl, disulfate, carboxyl, imidazole or amine groups of the compounds present in the cells) [15].

Exposure to Cd and its health consequences have provoked the search of efficient means of protection against the unfavorable effects of the element. Antioxidative compounds can play a significant role here. Their potential protective characteristics can be associated with antioxidative enzyme activation and reactive oxygen species scavenging, as well as affecting Cd metabolism (formation of complexes excreted with urine and feces) and interaction at the cellular level [32]. These compounds are contained in numerous plant products, including turmeric, ginger, garlic, onion, black seeds oil, olive oil, blueberry, green tea, hibiscus.

PROTECTIVE EFFECT OF PLANT PRODUCTS ON ANIMALS EXPOSED TO CD

Turmeric

The majority of studies (18) concerned curcumin - the main component of turmeric. In the conducted studies, different doses of curcumin and different time of exposure to Cd were used. Research revealed changes in the level of Cd in the blood, vessels (aorta), organs (heart, kidneys, liver), improved biochemical parameters of the blood, positive impact on liver, muscles, cardiovascular, genitourinary, nervous and immune system. Summary of research results is presented in Table 1.

Two studies in animal models exposed to the effect of Cd demonstrated favorable effect of curcumin on the cardiovascular system, including reduced blood pressure, improvement of vascular responsiveness, reversal of the alterations of the aortas. Protective effect of curcumin was observed, including protein concentration regulation (endothelial nitric oxide synthase and anti-inducible nitric oxide synthase) in aortas, increase of urinary nitrate/nitrite level, restoration of glutathione redox ratio. It was furthermore shown that curcumin reduces lipid and protein peroxidation and in heart, liver and kidneys, which was linked to reduced blood pressure [28, 44].

Alghasham et al. [10] demonstrated that curcumin reduced intensification of inflammation in rats exposed to Cd. Reduced concentration of tumor necrosis factor alpha and interleukin-6 and favorable changes of the activity of superoxide dismutase, glutathione concentration and total antioxidant capacity in plasma were observed.

With exposure to Cd and curcumin supplementation, kidneys were found to inhibit adenosine deaminase activity (ADA), increase of nitric oxide level and functional SH groups as well as reduced Cd accumulation [8]. The favorable effect of curcumin in nephrotoxicity caused by Cd was also revealed in the study conducted by Kim [27]. Curcumin prevented proximal tubular cell damage and it contributed to reducing inflammatory processes and histopathological changes of the kidneys.

The protective effect of curcumin also concerned the animal reproductive system of animals exposed to Cd. A study in mice revealed influence of curcumin on the protection against testicle damage by the activation of Nrf2/ARE signaling pathway. Curcumin also contributed to the improvement of semen quality in mice [51]. Similar effects were obtained in studies on rats. Curcumin improved histological and biochemical testicular fluctuations [1]. On the other hand, the study conducted by Aktas et al. [9] demonstrated that the usage of curcumin resulted in restricted apoptosis in rat testicle tissues and improved histological appearance, as well as increased testosterone level. The study of Oguzturk et al. [37] showed that curcumin limited histopathological changes, reproductive and spermatological damage in rats exposed to Cd. The effect of curcumin on lipid and protein peroxidation and the positive effect on morphometrical parameters in the testis was also observed in studies on mice [31].

Furthermore, curcumin can constitute an agent supporting the treatment of memory dysfunction caused by Cd. Two studies in animal models revealed that curcumin produced AChE (acetylcholinesterase) and ADA activity in cerebral cortex, and restricted lipid peroxidation, which had a positive effect on cognitive functions of the animals [5, 7]. Similar effects were observed in a study conducted on rats. Curcumin reduced lipid peroxidation and modulated the activity of cerebral ectonucleotidases and AChE activities, thus limiting memory disturbances in animals [17]. In a different study, curcumin alleviated the negative effect of Cd by affecting the level of antioxidative enzyme activity as well as hippocampal protein concentration. What is more, restored behavioral changes in Cd-exposed mice was observed [33]. The study of Akinyemi et al. [6] showed neuroprotective effect of curcumin exhibited by inhibiting AChE activity and modulating mRNA expression level in whole cerebral cortex. Other research on animals exposed to Cd provided evidence for the multidirectional effect of curcumin, i.a. changes in social behavior, normalization in blood composition, reproductive hormones and brain AChE [3].

Studies on animal models further revealed the effect of curcumin on the activity of antioxidative enzymes and the concentration of non-enzymatic antioxidants in liver, reduced lipid peroxidation and liver damage [42].

On the other hand, study conducted on carp exposed to Cd showed increased effect of curcumin associated with reduced Cd accumulation in muscle. However, lipid peroxidation reduced by curcumin could not be confirmed [46].

Ginger

Ginger has been another plant product tested for the aspect of reducing Cd induced toxicity. Table 2 presents results of studies in animal models exposed to Cd, which analyzed

the effect of ginger on the urinary, reproductive and nervous system, as well as liver and kidneys.

Protective effect of ginger on urinary system was demonstrated, including reduced DNA damage, histopathological changes and disturbances of renal function of rats exposed to Cd [21]. Another study on rats enabled observation of a positive impact of ginger on urogenital tract. Reduced activity of enzymes (acid phosphatase, prostatic acid phosphatase, alkaline phosphatase) in the serum, improvement of hematological indices and organ weight were observed [39].

In a study on rats subject to the effect of Cd, Akinyemi and Adeniyi [5] observed protective effect of ginger oil on the nervous system. Compounds contained in the essential oil regulated inflammatory cytokine concentration (interleukin-6, interleukin-10) in brain and reduced activity of enzymes (AChE and ADA) in prefrontal-cortex and hippocampus significant in the prevention/treatment of neurodegenerative disorders.

What is more, positive effect of ginger was observed on genetic changes (influence on gene expression) and histopathological changes in liver and kidneys of rabbits exposed to Cd [13].

Garlic

Table 3 presents results of studies with animals exposed to Cd concerning the effect of garlic on liver and urinary and reproductive system.

Research conducted on rats exposed to Cd demonstrated protective effect of garlic in prostate glands. Researchers observed increased activity of enzymes (glutathione S-transferase, catalase, superoxide dismutase, acid phosphatase) in the prostate gland, reduced concentration of glutathione and increased activity of acid phosphatase in the serum [38].

The cytoprotective effect of diallyl tetrasulfide contained in garlic was presented in the study of Ponnusamy and Pari [41]. Administering animals with diallyl tetrasulfide reduced Cd accumulation in testicles and reduced lipid peroxidation and testicle damage.

Study of Nwokocha et al. [36] conducted on rats subject to the effect of Cd showed protective effect of garlic extracts on liver. Garlic extract had positive effect on the body weight and reduced Cd accumulation in liver. On the other hand, the study conducted by Ugwaja et al. [48] revealed positive effect of a spice mix containing garlic, ginger and nutmeg on the parameters of liver function when rats were exposed to Cd. Addition of these spices significantly reduced alanine aminotransferase, aspartate aminotransferase, bilirubin and albumin concentration in blood serum. The spice mix further contributed to improved renal function and cholesterol concentration in blood.

Onion

Table 4 presents results of studies in animal models exposed to Cd, which analyzed the protective effect of onion on the cardiovascular and urinary system and liver.

The study of Alpsoy et al. [11] demonstrated favorable effect of onion extract on the cardiovascular system. It was shown that onion extract reduced histological changes and apoptosis in cardiomyocytes.

What is more, positive effect of onion extract on atherosclerotic changes including improved lipid metabolism parameters in rats exposed to Cd. What is more, onion extract resulted in reduced lipid peroxidation and increased super oxide dismutase activity in liver and kidneys, and improved renal function parameters [24].

Ola-Mudathir and Suru [38] who conducted studies on animals exposed to Cd presented protective effect of onion in prostate glands. They were able to observe increased activity of enzymes (glutathione S-transferase, catalase, superoxide dismutase, acid phosphatase) in the prostate gland, reduced glutathione concentration and increased acid phosphatase concentration in the serum.

Other plant products

Studies on animal models also analyzed the effect of black seeds oil, olive oil, blueberry, hibiscus and green tea in the aspect of reducing Cd induced toxicity. It was observed that these products reduced the unfavorable changes in urinary, respiratory, reproductive and nervous system and in liver (Table 5).

Protective effect of thymoquinone (the main constituent of the essential oil from black seeds) was observed in reducing Cd-induced nephrotoxicity. Thymoquinone, thanks to its antioxidative and anti-apoptotic properties reduced the number of apoptotic cells and expression of nuclear factor-κB in renal tissue. What is more, reduced histopathological changes in renal tissue was demonstrated [20].

Study conducted on rats [19] revealed that the use of black seeds oil in animals exposed to Cd reduced the majority of negative structural changes of the lung parenchyma. The cytoprotective effect was related to the antioxidative and anti-inflammatory properties of the oil.

In the group of animals administered with olive oil and exposed to Cd were found to have restored activity of antioxidative enzymes, alanine transferase and aspartate aminotransferase to normal levels, palliation of changes in the lipid profile and activity of AST, ALT, as well as reduced oxidative stress [12].

Protective effect of DPE (2-(3,4 dihydroxyphenyl)ethanol - phenolic compound present in olive oil) on spleen and testes exposed to Cd was observed in the study conducted by Merra et al. [30]. DPE protected cytosol of spleen in rats exposed to Cd.

Two studies on animals exposed to Cd revealed positive effect of blueberries on ovaries and liver. Izaguirry et al. [25] showed positive effect of blueberries on the level of reactive species in ovaries and δ-aminolevulinate dehydratase activity in mice. Increased number of normal follicles was observed. However, no changes in the activity of 17 β-hydroxysteroid dehydrogenase, glutathione peroxidase and glutathione-S-transferase and in Cd concentration in mice ovaries were observed.

The study on mice exposed to Cd conducted by Gong et al. [22] showed hepatoprotective effect of blueberries via activation of antioxidative enzymes and reduced lipid and protein peroxidation in liver, as well as increased alanine

aminotransferase and aspartate aminotransferase activity in plasma. Furthermore, blueberries limited the DNA damage in hepatic tissue.

Study conducted on mice model [14] revealed that L-theanine (one of the major components in green tea) (at a dose of 50 and 100 mg/day) contributed to reduced Cd concentration in brain and blood of the animals and reduced tau hyperphosphorylation. L-theanine showed protective effect against apoptosis of brain cells, and at higher dose (100 mg/day) it affected brain antioxidative enzyme activation.

In mice exposed to Cd, green tea extract produced increased sperm cell concentration and reduced sperm cell morphological changes. What is more, increased antioxidative enzyme activity and reduced glutathione in animal testes were shown [2]. Positive effect of green tea on reproductive system was also observed in the study conducted by Mahmoudi et al. [29]. Thanks to the antioxidative properties of green tea, reduced lipid peroxidation in testicular tissue was shown. Green tea also had positive impact on the semen quality of animals and reduced spermatogenesis disorders.

The protective effect of hibiscus on reproductive system in rats exposed to the effect of Cd was shown in the study by Oyewopo et al. [40]. Reduced histopathological changes in ovaries and increased level of reproductive hormones was observed.

CONCLUSIONS

Results of studies in animal models revealed positive effect of such plant products as turmeric, ginger, garlic, onion, black seeds oil, blueberry, green tea, hibiscus in the aspect of reducing negative health effects caused by exposure to Cd. The scope of action of these products was very broad and it included improved function of cardiovascular (primarily decreasing blood pressure, reduction changes in blood vessels), nervous (mainly through decreasing activity of acetylcholinesterase in brain and memory improvement), urinary (among others by improvement of creatinine clearance, reduction of creatinine and urea, reduction of degree of kidney damage), reproductive (improvement of sperm quality, increasing testosterone level in serum, reduction of histopathological changes in testes and ovaries) and respiratory system (reduction of changes in lungs), as well as liver (improvement of liver function parameters and reduction Cd accumulation) and muscles (reduction Cd accumulation). What is more, improved biochemical parameters of blood (increase hemoglobin and improvement blood counts) and lipid metabolism of the organism (decrease in total cholesterol, LDL fraction and triglicerydes, increase in HDL cholesterol in the blood) were shown.

Considering the promising results of studies in animal models, it appears valid to promote the consumption of natural plant products cited above in populations exposed to Cd in occupational and environmental terms and conducting research in the aspect of their efficacy rate for the human organism.

WNIOSKI

Wyniki badań na modelach zwierzęcych wykazały korzystny wpływ produktów roślinnych takich jak kurkuma, imbir, czosnek, cebula, olej z czarnuszki, borówka amerykańska, zielona herbata, hibiskus w aspekcie zmniejszania negatywnych skutków zdrowotnych spowodowanych ekspozycją na Cd. Spektrum działania tych produktów było bardzo szerokie i obejmowało poprawę funkcji układu krwionośnego (przede wszystkim obniżenie ciśnienia tętniczego krwi, zmniejszenie zmian w naczyniach krwionośnych), nerwowego (głównie poprzez obniżenie aktywności acetylocholinoestrerazy w mózgu i poprawę pamięci), moczowego (m.in. poprawa klirensu kreatyniny, obniżenie stężenia kreatyniny i mocznika, zmniejszenie stopnia uszkodzenia nerek), rozrodczego (poprzez poprawę jakości spermy, wzrost stężenia testosteronu w surowicy, zmniejszenie zmian histopatologicznych w jądrach i jajnikach), oddechowego (zmniejszenie zmian w płucach), a także wątroby (poprawa parametrów czynności wątroby, obniżenie stężenia Cd) i mięśni (obniżenie stężenia Cd). Wykazano również poprawę parametrów biochemicznych krwi (wzrost stężenia hemoglobiny i elementów morfotycznych krwi) oraz parametrów gospodarki lipidowej organizmu (obniżenie stężenia cholesterolu całkowitego, frakcji LDL i trójglicerydów, wzrost stężenia cholesterolu HDL we krwi).Ze względu na obiecujące wyniki badań prowadzonych na modelach zwierzęcych zasadnym wydaje się promocja spożycia wyżej wymienionych naturalnych produktów roślinnych w populacji osób narażonych zawodowo i środowiskowo na Cd oraz prowadzenie badań w aspekcie skuteczności ich działania na organizm człowieka.

Table 1. Results of studies in animal models with Cd exposure and curcumin treatment

Tabela 1. Wyniki badań przeprowadzonych na zwierzętach indukowanych kadmem i suplementowanych kurkuminą

References	Research model	Study description	Main results
28	Mice, ICR, adult male	CG (n= 8-10) – $CdCl_2$ (100 mg/l orally for 8 weeks) G1 (n= 8-10) – $CdCl_2$ (100 mg/l orally for 8 weeks) and curcumin (100mg/kg bw intragastically for 8 weeks)	G1 vs CG vascular: responsiveness↑, eNOS protein↑, pressure↓ aortic superoxide anion↓ urine: nitrate/nitrite↓ plasma: MDA↓, protein carbonyls↓ blood: GSH↓, Cd↓ aorta, liver, kidneys: Cd↓
44	Mice, ICR, male	CG (n=8-10) – $CdCl_2$ (100mg/l via drinking water, for 8 weeks) G1 (n=8-10) – $CdCl_2$ (100mg/l via drinking water, for 8 weeks) and THU (100 mg/kg bw/day concurently with Cd treatment)	G1 vs CG blood pessure↓, heart rate↔ aortas: eNOS↑, iNOS↓, superoxide anion↓, Cd↓ blood: GSH↑, Cd↓ urine: nitrate/nitrite↓ plasma: MDA↓, protein carbonyl↓ heart: MDA↓, protein carbonyl↓, Cd↓ liver: MDA↓, protein carbonyl↓ kidney: MDA↓, protein carbonyl↓, Cd↓
10	Rats, albino, male	CG (n=10) – $CdCl_2$ (40 mg/L drinking water, for 6 weeks) G1 (n=10) – $CdCl_2$ (40 mg/L drinking water, for 6 weeks) and curcumin (50 mg/kg bw, by gastric tube daily, for 6 weeks)	G1 vs CG body weight↑ plasma: CAT↔, SOD↑, GSH↑, TCA↑, MDA↓, TNF-α↓, IL-6↓
8	Rats, albino, adult male	CG (n=6) – Cd (2.5 mg/kg orally for 7 days) G1 (n=6) – Cd (2.5 mg/kg orally) and curcumin (12,5 mg/kg orally by gavage for 7 days) G2 (n=6) Cd (2.5 mg/kg orally) and curcumin (25 mg/kg orally by gavage for 7 days)	G1 and G2 vs CG kidney: ADA↓, arginase↓, NO↑, urea↓, creatinine↓, Na^+↓, K^+↓, Cl^-↓, MDA↓, TSH↑, NPSH↑, Cd↓
27	Rats, Spraque-Dawley, male	CG (n=6) – $CdCl_2$ (25 mg/kg bw, oral gavage for 7 days) G1(n=6) – $CdCl_2$ (25 mg/kg bw oral for 7 days) and curcumin (50 mg/kg bw pre-treatment, orally)	G1 vs CG weight: body↔, kidney↑, serum: BUN↓, creatinine↓, AST↓, glucose↓, ALT↔, uric acid↔ urine: volume↓, pH↔, creatinine↑, creatinine clearance↑, total protein↑, glucose↓ excretion of Kim-1↓, OPN↓, TIMP-1↓, NGAL↓, netrin-1↓
51	Mice, Kunming, male	CG (n=12) – $CdCl_2$ (2 mg/kg bw, intraperitoneally for 10 day) G1 (n=12) – CdCl2 (2 mg/kg bw, intraperitoneally for 10 day) and curcumin (50 mg/kg bw, co-treatment, for 10 days)	G1 vs CG sperm: motility↑, concentration↑, abnormal↓ serum: testosterone↑ testis: Nrf2 protein↑, GPx↑, GSH↑, T-SOD↑, MDA↓
1	Rats, Sprague Dawley, adult male	CG (n=10) – CdC_2 (1 mg/kg bw, intraperitoneal injection for 3 days) G1 (n=10) – CdC2 (1 mg/kg bw, intraperitoneal injection for 3 days) and after that curcumin (5 mg/kg bw/day, orally for 4 weeks)	G1 vs CG serum: testosterone↑ testis: MDA↓, SOD↑, GSH↑, NO↓, mean number of spermatogenic cells↑
9	Rats, Wistar albino, male	CG (n=10) – Cd (1mg/kg bw, injected for 4 weeks) G1 (n=10) – Cd (1mg/kg bw, injected) and curcumin (100 mg/kg bw by using intra-gastric intubation)	G1 vs CG serum: testosterone↑ testis: MSTD↑, MTBS↑, damage↓, apoptotic index↓, apoptotic index of Leydig cell↓

References	Research model	Study description	Main results
37	Rats, Spraque-Dawley, adult male	CG (n=7) – $CdCl_2$ (1mg/kg intraperitoneally for 3 days) G1 (n=7) – $CdCl_2$ (1mg/kg) and curcumin (100 mg/kg bw orally for 3 days)	G1 vs CG testis: TBARS↓, SOD↑, CAT↔, GPx↑, GSH↑, histopathological changes↓ weight: testis↔, epididymis↔, seminal vesicle↔, prostate↔ sperm: concentration↔, motility↔, abnormal↔
31	Mice, NMRI adult male	CG (n=6) – $CdCl_2$ (5 mg/kg bw, subcutaneously) G1 (n=6) – CdCl2 (5 mg/kg bw, subcutaneously) and curcumin (100 mg/kg bw, single intraperitoneal dose)	G1 vs CG testis: seminiferous tubules diameter↑, seminiferous tubule's lumen diameter↑, serum: MDA↓, TSH↑, CAT↑, SOD↑, GPx↑, GSH↑, H_2O_2↓
7	Rats, albino, adult male	CG (n=6) – Cd (2,5mg/kg by gavage, for 7 days) G1 (n=6) – Cd (2,5 mg/kg by gavage) and curcumin (12,5 mg/kg by gavage, for 7 days) G2 (n=6) – Cd (2,5 mg/kg by gavage) and curcumin (25 mg/kg by gavage for 7 days)	G1 and G2 vs CG cerebral cortex: NOR↑, AChE↓, ADA↓, NO↑, MDA↓, TSH↑, NPSH↑ memory index↑
5	Rats, albino, adult male,	CG (n=10) – Cd (2.5 mg/kg bw orally for 14 days) G1 (n=10) – Cd (2.5 mg/kg bw orally for 14 days) and essential oil from turmenic (50 mg/kg bw orally for 14 day)	G1 vs CG brain: IL-10↑, IL-6↓, TNF-alpha↓, prefronal cortex: AChE↓, ADA↓ hippocampus: AChE↓, ADA↓
17	Rats, Wistar, adult male,	CG (n=15) – Cd (1mg/kg by oral gavage 5 days a week for 3 months) G1(n=15) – Cd (1mg/kg by oral gavage 5 days a week for 3 months) and curcumin (90 mg/kg by oral gavage 5 days a week for 3 months, after Cd)	G1 vs CG cerebral cortex synaptosomes: AChE↓, ATP↔, ADP↔, AMP↑ motor abilities↔, shock sensivity↔ striatum: AChE↓, TBARS↓ cerebral cortex: AChE↓, TBARS↓ hippocampus: AChE↔, TBARS↔ hypothalamus: AChE↔, TBARS↔ cerebellum: AChE↔, TBARS↓ whole blood: AChE↓ lymphocytes: AChE↓
33	Mice, Swiss Albino, young	CG (n=7) – Cd (2.5 mg/kg bw oral for 60 days, pre-treatment) G1 (n=7) – Cd (2.5 mg/kg bw oral for 60 days) and curcumin (160 mg/kg bw oral for 30 days from day 30^{th} to day 60^{th})	G1 vs CG locomotor activity↑, retention memory↑, recognition memory↑ hippocamp: MDA↓, SOD↑, catalase↑, GSH↑, proteins (BDNF, SynII, DCX, CREB)↑
6	Rats, albino, male	CG (n=10) – Cd (2.5 mg/kg bw by oral gavage for 7 days) G1 (n=10) – Cd (2.5 mg/kg bw by oral gavage for 7 days) and curcumin (25 mg/kg bw by oral gavage for 7 days) G2 (n=10) – Cd (2.5 mg/kg bw by oral gavage for 7 days) and curcumin (50 mg/kg bw by oral gavage for 7 days)	G1 and G2 vs CG cerebral cortex: AChE↓, AChE mRNA↓
3	Mice, Swiss-Webster, male and female	CG (n=10) – Cd (100 mg/kg bw oral administration for 2 weeks) G1 (n=10) – Cd (100 mg/kg bw oral administration for 2 weeks) and curcumin (150 mg/kg bw oral administration for 2 weeks) G2 (n=10) – Cd (100 mg/kg bw oral administration for 2 weeks) and curcumin (300 mg/kg bw oral administration for 2 weeks)	G1 and G2 vs CG blood: hemoglobin↑, packed cell volume↑, WBC↑, platelets↑, testosterone (male)↑, progesterone (female)↑ brain: AChE↓ G1 vs CG blood: RBC↑

References	Research model	Study description	Main results
42	Rats, Wistar, albino, male	CG (n=6) – $CdCl_2$ (5 mg/kg bw/day orally administered for 4 weeks) G1 (n=6) – $CdCl_2$ (5 mg/kg bw/day orall administered for 4 weeks) and THC (80 mg/kg bw/day orally administered for 4 weeks)	G1 vs CG body weight↔, food, water intake↑, hepatic index (liver weight-body weight ratio)↓ serum: AST↓, ALT↓, ALP↓, LDH↓, GGT↓, bilirubin↓, blood: Cd↓ liver: Cd↓, TBARS↓, LHP↓, protein carbonyl↓, SOD↑, CAT↑, GPx↑, GST↑, GR↑,G6PD↑, GSH↑, TSH↑
46	Carps, *Cyprinus carpio*, young	CG (n=4) – Cd (5 mg/kg bw, single intraperitoneal dose) G1 (n=4) – Cd (5 mg/kg bw, single intraperitoneal dose) and curcumin (50mg/kg bw, injected after Cd)	G1 vs CG muscle: Cd↓, SOD↑, CAT↔, GPx↑, GR↔, tGSH↔, GSSG↔, GSH↔, TRABS↔, protein↔

AChE – acetylcholinesterase, ADA – adenoine deaminase, ALP – alkaline phosphatase, ALT – alanine aminotransferase, AST – aspartate aminotransferase, BDNF – brain-derived neurotrophic factor, BUN – blood urea nitrogen, CAT – catalase, Cd – cadmium, CG - Control Group, CREB – cAMP response element binding protein, curcumin- the main component of turmenic, DCX – doublecortin, eNOS - endothelial nitric oxide synthase, G1 - Group 1, G2 – Group 2, G6PD – glucose-6-phosphate dehydrogenase, GGT – gamma glutamyl transferase, GPx – glutathione peroxidase, GR – glutathione disulfide reductase, GSH – reduced glutathione, GSSG – oxidized glutathione, IL-10 – interleukin-10, IL-6- interleukin-6, iNOS – anti-inducible nitric oxide synthase, Kim-1 – kidney injury molecule-1, LDH- lactate dehydrogenase, LHP – lipid hydroperoxides, MDA – malondialdehyde, MSTD – mean seminiferous tubule diameter, MTBS – mean testicular biopsy score, NGAL – neutrophil gelatinase-associated lipocalin, NO – nitric oxide, NOR – novel object recognition, NPSH – non-protein thiol, OPN – osteopontin, RBC – red blood cell, SOD – superoxide dismutase, Syn II – synapsin II, TBARS – thiobarbituric acid reactive substances, TCA – total antioxidant capacity, tGSH – total glutathione, THC – tetrahydrocurcumin, TIMP-1 – tissue inhibitor of metalloproteinases 1, TNF-alpha – tumor necrosis factor alpha, TSH – total thiol, T-SOD – total superoxide dismutase, WBC – white blood cell.

AChE – acetylocholinoesteraza, ADA – deaminaza adenozynowa, ALP – fosfataza alkaliczna, ALT – aminotransferaza alaninowa, AST – aminotransferaza asparaginianowa, BDNF – neurotroficzny czynnik pochodzenia mózgowego, BUN – azot mocznika, CAT – katalaza, Cd – kadm, CG – Grupa Kontrolna, CREB - białko wiążące element odpowiedzi cAMP, kurkumina – główny składnik kurkumy, DCX – doublekortyna, eNOS – śródbłonkowa syntaza tlenku azotu, G1 – Grupa 1, G2 – Grupa 2, G6PD – dehydrogenaza glukozo-6-fosforanowa, GGT – gamma-glutamylotransferaza, GPx – peroksydaza glutationowa, GR – reduktaza glutationu, GSH – glutation zredukowany, GSSG – glutation utleniony, IL-10 – interleukina-10, IL-6 – interleukina-6, iNOS – indukowalna syntaza tlenku azotu, Kim-1 – cząsteczka uszkodzenia nerek-1, LDH – dehydrogenaza mleczanowa, LHP – wodoronadtlenki lipidowe, MDA – dialdehyd malonowy, MSTD – średnia średnica kanalików nasiennych, MTBS – średni wynik z biopsji jąder, NGAL – lipokalina związana z żelatynazą neutrofili, NO – tlenek azotu, NOR – test rozpoznawania nowego obiektu, NPSH – niebiałowy tiol, OPN – osteopontyna, RBC – krwinka czerwona, SOD – dysmutaza ponadtlenkowa, SynII – synapsyna II, TBARS – substancje reagujące z kwasem tiobarbiturowym, TCA – całkowita zdolność antyoksydacyjna, tGSH – glutation całkowity, THC – tetrahydrokurkumina, TIMP-1 – tkankowy inhibitor metaloproteinaz-1, TNF-alpha – czynnik martwicy nowotworów alfa, TSH – całowita zawartość tioli, T-SOD – całowita dysmutaza ponadtlenkowa, WBC – krwinki białe.

Source: Own study

Źródło: Opracowanie własne

Table 2. Results of studies in animal models with Cd exposure and ginger treatment

Tabela 2. Wyniki badań przeprowadzonych na zwierzętach indukowanych kadmem i suplementowanych imbirem

References	Research model	Study description	Main results
21	Rats, Wistar, male, adult	CG (n=10) – $CdCl_2$ (5 mg/kg bw for 30 days) G1 (n=10) – $CdCl_2$ (5 mg/kg bw for 30 days) and ginger extract (100 mg/kg for 30 days) G2 (n=10) – CdCl2 (5 mg/kg bw for 30 days) and ginger extract (200 mg/kg for 30 days)	G1 and G2 vs CG body weight↑ kidney: weight↓, creatinine↓ urea↓, urine albumin↓, creatinine clearance↑, MDA↓, TAC↑, DNA↑
39	Rats, Wistar, albino, adult	CG (n=10) – $CdCl_2$ (3 mg/kg bw orally for 28 days) G1 (n=10) – $CdCl_2$ (3 mg/kg bw orally for 28 days) and ginger supplement (0,5 g/kg bw orally for 28 days)	G1 vs CG testes: MDA↓, weight↑ kidney: MDA↓, weight↑ serum: ACP↓, PAP↓, ALP↓ blood: Hb↑, PCV↑, RBCs↑, WBCs↓
5	Rats, albino, male, adult	CG (n=10) – Cd (2.5 mg/kg bw orally for 14 days) G1 (n=10) – Cd (2.5 mg/kg bw orally for 14 days) and essential oil from ginger (50mg/kg bw orally for 14 day)	G1 vs CG brain: IL-10↑, IL-6↓, TNF-alpha↓ prefronal cortex: AChE↓, ADA↓ hippocampus: AChE↓, ADA↓

References	Research model	Study description	Main results
13	Rabbits, male	CG (n=10) – $CdCl_2$ (200 mg/kg bw orally for 12 weeks) G1 (n=10) – $CdCl_2$ (200 mg/kg bw orally for 12 weeks) and ginger supplement (400 mg/kg bw orally for 12 weeks)	G1 vs CG kidney: caspase 3 expression↓, Bcl2 expression↑, GST expression↑, C-fos↓, MKI67↓ liver: caspase 3 expression↓, Bcl2 expression↑, GST expression↓, C-fos↓, MKI67↓

AChE – acetylcholinesterase, ADA – adenoine deaminase, ALP – alkaline phosphatase, Cd – cadmium, CG – Control Group, DNA – deoxyribonucleic acid, G1 – Group 1, Hb – hemoglobin, IL-10 – interleukin-10, IL-6 – interleukin-6, MDA – malondialdehyde, PAP – prostatic acid phosphatase, PCV – packed cell volume, RBC – red blood cell, TAC – total antioxidant status, TNF-alpha – tumor necrosis factor alpha, WBC – white blood cell.

AChE – acetylcholinoesteraza, ADA – deaminaza adenozynowa, ALP – fosfataza alkaliczna, Cd – kadm, CG – Grupa Kontrolna, DNA – kwas deoksyrybonukleinowy, G1 – Grupa 1, Hb – hemoglobina, IL-10 – interleukina-10, IL-6 – interleukina-6, MDA – dialdehyd malonowy, PAP – kwaśna fosfataza sterczowa, PCV – objętość upakowanych komórek, RBC – krwinka czerwona, TAC – całkowity status antyoksydacyjny, TNF-alpha – czynnik martwicy nowotworów alfa, WBC – krwinka biała.

Source: Own study

Źródło: Opracowanie własne

Table 3. Results of studies in animal models with Cd exposure and garlic treatment

Tabela 3. Wyniki badań przeprowadzonych na zwierzętach indukowanych kadmem i suplementowanych czosnkiem

References	Research model	Study description	Main results
38	Rats, Wistar, male, adult	CG (n=6) – Cd (1.5 mg/100 g bw/ day by gavage for 3 weeks) G1(n=6) – Cd (1.5 mg/100 g bw/ day) and garlic extract (1 ml/100 g bw/day by gavage for 3 weeks)	G1 vs CG prostate glands: MDA↓, GSH↑, CAT↑, SOD↑ GST↓, PAP↑ plasma: ACP↓
41	Rats, Wistar, male,	CG (n=6) – $CdCl_2$ (3 mg/kg bw/day, subcutaneously for 3 weeks) G1 (n=6) – $CdCl_2$ (3 mg/kg bw/day for 3 weeks) and DTS (40 mg/kg bw/day subcutaneously for 3 weeks)	G1 vs CG testis: weight↑, Cd↓, TBARS↓, hydroperoxides↓, protein carbonyls↓, GSH↑, TSH↑, SOD↑, CAT↑, GPx↑, GST↑, GR↑, G6PD↑
36	Rats, Wistar, male	CG (n=15) – $CdCl_2$ (200 ppm in drinking water for 6 weeks) G1(n=15) – $CdCl_2$ (200 ppm in drinking water for 6 weeks) and garlic (7%w/w for 6 weeks)	G1 vs CG weight changes↔ liver: Cd↓
48	Rats, albino, male	CG (n=6) – Cd (25 mg/kg bw/day orally for 4 weeks) G1(n=6) – SM (300 mg/kg bw/day orally for 2 weeks) and after Cd (25 mg/kg bw/day orally for 4 weeks) G2 (n=6) – Cd (25 mg/kg bw/day orally for 4 weeks) and SM (300 mg/kg bw/day orally for 4 weeks) G3 (n=6) – Cd (25 mg/kg bw/day orally for 4 weeks) and after SM (300 mg/kg bw/day orally for 2 weeks)	G1, G2, G3 vs CG urine: creatinine↓ serum: AST↓, ALT↓ G1 vs CG urine: urea↔, uric acid↓, total cholesterol↓ G2 vs CG urine: urea↑, uric acid↑, total cholesterol↑ G3 vs CG urine: urea↓, uric acid↓, total cholesterol↑

CAT – catalase, Cd – cadmium, CG – Control Group, DTS – diallyl tetrasulfide from garlic, G1 – Group 1, G2 – Group 2, G3 – Group 3, G6PD – glucose-6-phosphate dehydrogenase, GPx – glutathione peroxidase, GR – glutathione disulfide reductase, GSH – reduced glutathione, GST – glutathione-S-transferase, MDA – malondialdehyde, PAP – prostatic acid phosphatase, SM – spice mixture (ginger rhizomes, garlic bulbs and nutmeg), SOD – superoxide dismutase, TBARS – thiobarbituric acid reactive substances, TSH – total sulphydryl groups.

CAT – katalaza, Cd – kadm, CG – Grupa kontrolna, DTS – tetrasiarczek diallilu z czosnku, G1 – Grupa 1, G2 – Grupa 2, G3 – Grupa 3, G6PD – dehydrogenaza glukozo-6-fosforanowa, GPx – peroksydaza glutationowa, GR – reduktaza glutationu, GSH – glutation zredukowany, GST – transferaza glutationowa, MDA – dialdehyd malonowy, PAP – kwaśna fosfataza sterczowa, SM – mieszanka przypraw (kłącze imbiru, czosnek i gałka muszkatołowa), SOD – dysmutaza ponadtlenkowa, TBARS – substancje reagujące z kwasem tiobarbiturowym, TSH – wszystkie grupy sulfhudrylowe.

Source: Own study

Źródło: Opracowanie własne

Table 4. Results of studies in animal models with Cd exposure and onion treatment

Tabela 4. Wyniki badań przeprowadzonych na zwierzętach indukowanych kadmem i suplementowanych cebulą

References	Research model	Study description	Main results
11	Rats, Sprague-Dawley, male	CG (n=8) – $CdCl_2$ (2 ml/kg bw injected subcutaneously for 30 days) G1(n=8) – $CdCl_2$ (2 ml/kg bw injected subcutaneously for 30 days) and AcE (1 ml via intragastric intubation for 30 days)	G1 vs CG cardiac tissue: MDA↓, SOD↑, CAT↑, GPx↑, degenerative changes↓
24	Rats, Wistar, male	CG (n=10) – $CdSO_4$ (1.5 ml/kg bw via drinking for 4 weeks) G1 (n=10) – $CdSO_4$ (1.5 ml/kg bw via drinking for 4 weeks) and AcE (1 ml/100 g bw orally for 8 weeks)	G1 vs CG weight: ↑ liver: weight↑, MDA↓, SOD↑ kidney: weight↔, 24-hour urinary volume↑, creatinine clearance↑, urea clearance↑, MDA↓, SOD↑ plasma: cholesterol↓, TG↓, HDL↑, LDL↓, total protein↑, albumin↓, MDA↓, SOD↑
38	Rats, Wistar, male, adult	CG (n=6) – Cd (1.5 mg/100 g bw/ day by gavage for 3 weeks) G1(n=6) – Cd (1.5 mg/100 g bw/ day) and onion extract (1 ml/100 g bw/day by gavage for 3 weeks)	G1 vs CG prostate glands: MDA↓, GSH↑, CAT↑, SOD↑ GST↓, ACP↑ plasma: PAP↓

AcE – onion extract, CAT – catalase, Cd – cadmium, CG – Control Group, G1 – Group 1, GPx – glutathione peroxidase, GSH – reduced glutathione, GST – glutathione- S-transfera, HDL – high density lipoprotein, LDL – low density lipoprotein, MDA – malondialdehyde, PAP – prostatic acid phosphatase, SOD – superoxide dismutase, TG – triglyceride.

AcE – ekstrakt z cebuli, CAT – katalaza, Cd – kadm, CG – Grupa Kontrolna, G1 – Grupa 1, GPx – peroksydaza glutationowa, GSH – glutation zredukowany, GST – transferaza glutationowa, HDL – lipoproteina o dużej gęstości, LDL – lipoproteina o niskiej gęstości, MDA – dialdehyd malonowy, PAP – kwaśna fosfataza sterczowa, SOD – dysmutaza ponadtlenkowa, TG – triglicerydy.

Source: Own study

Źródło: Opracowanie własne

Table 5. Results of studies in animal models with Cd exposure and black seeds oil, olive oil, blueberry, green tea and hibiscus treatment

Tabela 5. Wyniki badań przeprowadzonych na zwierzętach indukowanych kadmem i suplementowanych olejem z czarnuszki, oliwą z oliwek, borówką amerykańską, zieloną herbatą i hibiskusem

References	Research model	Study description	Main results
Black seeds oil			
20	Rats, Wistar albino	CG (n=8) – $CdCl_2$ (1 mg/kg bw injrcted subcutaneously for 30 days) G1(n=8) – $CdCl_2$ (1 mg/kg bw injrcted subcutaneously for 30 days) and TQ (50 mg/kg bw/day orally)	G1 vs CG renal tissue: injury↓, MDA↓, SOD↑, CAT↑, GPx↑ immunoreactivity NK-κB p65↓, apoptotic cells↓
19	Rats, Wistar, male, adult	CG (n=10) – $CdCl_2$ (2 mg/kg intraperitoneally for 28 days) G1 (n=10) – $CdCl_2$ (2 mg/kg intraperitoneally for 28 days) and NSO (1 ml/kg bw by gastric gavage for 28 days)	G1 vs CG lungs: changes↓
Olive oil			
12	Rats, Wistar, male	CG (n = 5) – olive oil (contain olive oil of diet 4% for 8 weeks) G1 (n = 5) – Cd (50 mg/l orally - drinking water for 8 weeks) and olive oil (contain olive oil of diet 4% for 8 weeks)	G1 vs CG plasma: total cholesterol↔, HDL↓, LDL↓, VLDL↑, TG↑, CAT↓, SOD↓, MDA↑ protein carbonyls↔ liver: total cholesterol↔, total lipids↔, TG↓, CAT↓, SOD↓, MDA↔, protein carbonyls↔, GSH↓ serum: AST↑, ALT↑
30	Rats, Wistar, male	CG (n=5) – $CdCl_2$ (2.5 mg/kg bw injected i.p.) G1 (n=5) – $CdCl_2$ (2.5 mg/kg bw injected) and DPE (9 mg/kg bw injected i.p.)	G1 vs CG spleen: TBARS↓, CytCAT↑, MitCAT↔, testes: TBARS↔, CytCAT↔, MitCAT↔

References	Research model	Study description	Main results
Blueberry			
25	Mice, Swiss, adult, female	CG (n = 6) – $CdCl_2$ (10 µmol/kg bw, injected subcutaneously for 3 weeks) G1 (n=6) – $CdCl_2$ (10 µmol/kg bw, injected subcutaneously for 3 weeks) and blueberry extract (2,5 mg/kg, orally via gavage for 3 weeks)	G1 vs CG ovaries: reactive species↓, follicle viability↑, Cd↔, δ-ALA-D↑, GPx↔, GST↔, 17-β HSD↔
22	Mice, Kunmig, male	CG (n=7) – $CdCl_2$ (2 mg/kg/day intraperitoneal injections for 14 days) G1 (n=7) – $CdCl_2$ (2 mg/kg/day intraperitoneal injections for 14 days) and Ay (0,3 mg/kg/day, intragastric for 14 days) G2 (n=7) – $CdCl_2$ (2 mg/kg/day intraperitoneal injections for 14 days) and Ay (3 mg/kg/day, intragastric for 14 days) G3 (n=7) – $CdCl_2$ (2 mg/kg/day intraperitoneal injections, for 14 days) and Ay (30 mg/kg/day, intragastric for 14 days)	G1, G2, G3 vs CG plasma: AST↑, ALT↑ liver: MDA↓, protein carbonyl↓, SOD↑, CAT↑, GHS↓, NO↓, DNA fragmentation↓
Green tea			
14	Mice, ICR, male	CG (n=10) – $CdCl_2$ (3.75-6 mg/kg bw orally administered, 30 mg/l in water for 8 weeks) G1 (n=10) – $CdCl_2$ (3.75-6 mg/kg bw orally administered, 30 mg/l in water for 8 weeks) and L-theanine (100 mg/kg bw intraperitoneally injected for 8 weeks) G2 (n=10) – $CdCl_2$ (3.75-6 mg/kg bw orally administered, 30 mg/l in water for 8 weeks) and L-theanine (200 mg/kg bw intraperitoneally injected for 8 weeks)	G1 and G2 vs CG brain: Cd↓, ROS↓, tau hyperphosphorylation↓, apoptotic neuronal cell death↓ plasma: Cd↓ G1 vs CG brain: MDA↔, GSH↔, CAT↔, GPx↔ G2 vs CG brain: MDA↓, GSH↑, CAT↑, GPx↑
2	Rats, Wistar, male	CG (n=9) – $CdCl_2$ (3 mg/kd bw, gavage for 63 days) G1 (n=9) – $CdCl_2$ (3 mg/kd bw, gavage for 63 days) and GTE (70 mg/kg gavage for 63 days)	G1 vs CG weight: body↑, sexual organs↑ sperm: cell concentration↑, viability↔, abnormalities↓ serum: total cholesterol↓ testis: CAT↑, SOD↑, GSH↑
29	Rats, Wistar, male	CG (n=12) – $CdCl_2$ (1,5 mg/kg bw intraperitoneally, single dose) G1 (n=12) – $CdCl_2$ (1,5 mg/kg bw intraperitoneally, single dose) and GT solution (1,5% w/v until the end of the treatment – 49th day)	G1 vs CG sperm motility: fast↑, slow↔, non-motile↔ sperm: morphology↔, count↑ serum: testosterone↑ testis: MDA↓
Hibiscus			
40	Rats, Wistar, female	CG (n=5) – Cd (cadmium sufphate 5 mg/kg bw for 5 days i.p.) G1 (n=5) – Cd (cadmium sufphate 5 mg/kg bw for 5 days i.p.) and hibiscus (100 mg/kg bw orally for 28 days)	G1 vs CG body weight↑ serum: FSH↑, LH↑ ovaries: histopathologiacal changes↓

17-β HSD – 17 β-hydroxysteroid dehydrogenase, ALT – alanine aminotransferase, AST – aspartate aminotransferase, Ay – anthocyanin, CAT – catalase, Cd – cadmium,, CG – Control Group, CytCAT – catalase in cytosol, DNA – deoxyribonucleic acid, DPE – 2-(3,4 dihydroxyphenyl) ethanol (phenolic compound present in olive oil), FSH – follicle stimulating hormone, G1 – Group 1, GPx – glutathione peroxidase, GSH – reduced glutathione, GST – glutathione-S-transferase, GTE – green tea extract, HDL – high density lipoprotein, LDL – low density lipoprotein, LH – luteinizing hormone, LYM – lymphocyte, MDA – malondialdehyde, MitCAT – catalase in mitochondria, NO – nitric oxide, NSO – black seeds oil, SOD – superoxide dismutase, TBARS – thiobarbituric acid reactive substances, TG – triglyceride, TQ – thymoquinone (the main constituent of the essential oil from black seeds), VLDL – very low density lipoprotein, δ-ALA-D - δ-aminolevulinate dehydratase.

17-β HSD – dehydrogenaza 17-ß-hydroksy-steroidowa, ALT – aminotransferaza alaninowa, AST – aminotransferaza asparaginianowa, Ay – antocyjany, CAT – katalaza, Cd – kadm CG – Grupa Kontrolna, CytCAT – katalaza cytozolowa, DNA – kwas deoksyrybonukleinowy, DPE 2-(3,4-hydroksyfenylo)etanol (związek fenolowy obecny w oliwie z oliwek), FSH – hormon folikulotropowy, G1 – Grupa 1, GPx – peroksydaza glutationowa, GSH – glutation zredukowany, GST – transferaza glutationowa, GTE – ekstrakt z zielonej herbaty, HDL – lipoproteina o dużej gęstości, LDL – lipoproteina o niskiej gęstości, LH – hormone luteinizujący, LYM – limfocyt, MDA – dialdehyd malonowy, MitCAT – katalaza

mitochondrialna, NO – tlenek azotu, NSO – olej z czarnuszki, SOD – dysmutaza ponadtlenkowa, TBARS – substancje reagujące z kwasem tiobarbiturowym, TG – triglicerydy, TQ – tymochinon (główny składnik olejku eterycznego *z czarnuszki*), VLDL – lipoproteina o bardzo małej gęstości, δ-ALA-D – dehydrataza kwasu delta-aminolewulinowego.

Source: Own study

Źródło: Opracowanie własne

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