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Effect of feeding some selected food processing by-products on blood oxidant and antioxidant status of obese rats

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Abstract: The present study aims to investigate the effect of feeding some selected food processing by-products on blood oxidant and antioxidant status of obese rats. Forty two male rats, (weight 139 ± 5.3 g per each), were divided into two main groups, the first group (Group 1, 6 rats) still fed on basal diet and the other main group (36 rats) was feed with diet-induced obesity (DIO) for 8 weeks which classified into sex sub groups as follow: group (2), fed on DIO as a positive control; groups (3-7), fed on DIO containing 7.5 % tomato pomace powder (TPP), potato peel powder (PPP), cauliflower leaves powder (CLP), eggplant peel powder (EPP and their mixture, respectively. At the end of the experiment (8 weeks), rats of the obese group recorded body weight gain (BWG, 149.02%, as a percent of the baseline. Feeding of TPP, PPP, CLP, EPP and their mixture (Mix) induced significant decreasing on BWG of the obese rats which recorded 120.83, 138.32, 135.51, 127.65 and 119.02% as a percent of the baseline, respectively. Biochemical analysis data indicated that obesity induced a significant increased ($p \leq 0.05$) in plasma oxidants concentration (TBARS, 39.10%; NO_2 , 31.02% and NO_2/NO_3 , 27.10%) and significant decreased ($p \leq 0.05$) in plasma non-enzymes antioxidant (GSH, -35.45% and GSSG, -18.07%), plasma antioxidant vitamins (vitamin A, -27.43%; vitamin C, -18.39% and vitamin E, -23.04%) as well as RBC's antioxidant enzymes (GSH-Px, -39.35%; GSH-Rd, -31.52%; CAT, -29.65% and SOD, -25.03%) as a percent of normal control group. Feeding on 7.5% of TPP, PPP, CLP, EPP and their mixture exhibited a significant improvement ($p \leq 0.05$) in all of these parameters by different rates. The higher amelioration effects were recorded for the mixture treatment followed by TPP, EPP, CLP and PPP, respectively. In conclusion, the present data support the benefits of dietary modification, including bioactive compounds in plant parts supplementation, in alleviating oxidative stress associated obesity.

Keywords: Tomato pomace, potato peel, cauliflower leaves, eggplant peel, TBARS, GSH fractions, antioxidant enzymes, antioxidant vitamins.

Introduction

Obesity is a medical condition in which excess body fat has accumulated to the extent that it may have an adverse effect on health, leading to reduced life expectancy and/or increased health problems (Haslam and James, 2005). According to the Faculty of Public Health (FPH), obesity is “an excess of body fat frequently resulting in a significant impairment of health and longevity (Nammi, *et al.*, 2004). Body fatness is most commonly assessed by body mass index (BMI) which is calculated by dividing an individual's weight measured in kilograms by their height in meters squared. Overweight is generally defined as a BMI greater than 25; individuals with a BMI greater than 30 are classified as obese. According to the World Health Organization, there are more than one billion overweight adults in the world. At least 300 million of



them are clinically obese (WHO, 2000) and of these about 115 million come from developing countries. Furthermore, in the past 20 years, the rates of obesity have tripled in developing countries. Egypt, a developing country, is undergoing rapid urbanization changes. This has a direct impact on its people's dietary habits and physical activity patterns. According to national studies, it is common to skip meals and to replace them with daily snacks, and most of these snacks are high in calories and low in nutrients. So, Egypt appeared in No. 8 ranking among the countries of the world where obesity - adult prevalence rate, 30.3% (http://www.indexmundi.com/egypt/obesity_adult_prevalence_rate.html).

Oxidative stress (OS) was initially defined by Sies (1985) as a serious imbalance between oxidation and antioxidants, "a disturbance in the prooxidant-antioxidant balance in favor of the former, leading to potential damage". So, it reflects an imbalance between the systemic manifestation of reactive oxygen species (ROS) and a biological system's ability to readily detoxify the reactive intermediates or to repair the resulting damage. Disturbances in the normal redox state of cells can cause toxic effects through the production of peroxides and free radicals that damage all components of the cell, including proteins, lipids, and DNA. OS from oxidative metabolism causes base damage, as well as strand breaks in DNA (Toshniwal and Zarling, 1992 and Rahman *et al.*, 2012). Base damage is mostly indirect and caused by reactive oxygen species (ROS) generated, e.g. O_2^- (superoxide radical), OH (hydroxyl radical) and H_2O_2 (hydrogen peroxide) (Toshniwal and Zarling, 1992). Further, some reactive oxidative species act as cellular messengers in redox signaling. Thus, OS can cause disruptions in normal mechanisms of cellular signaling (Evans *et al.*, 2005). In humans, OS is thought to be involved in the development of in several diseases including cancer, atherosclerosis, malaria, chronic fatigue syndrome, rheumatoid arthritis and neurodegenerative diseases such as Parkinson's disease, Alzheimer's disease, and Huntington's disease (Halliwell, 1991 and Chaitanya *et al.*, 2010). Also, it is contributing to tissue injury following irradiation and hyperoxia as well as in diabetes and is likely to be involved in age-related development of cancer. Infection by *Helicobacter pylori* which increases the production of reactive oxygen and nitrogen species in human stomach is also thought to be important in the development of gastric cancer (Vasavidevi *et al.*, 2006 and Rahman *et al.*, 2012). Furthermore, associations between obesity and markers of oxidative stress and the susceptibility of lipid to oxidative modification have been observed in humans (Van Gaal *et al.*, 1998).

Industrialization of agriculture in the Arab world represent a large proportion of waste was estimated at 18.14 million tonnes per year and represent remnants of fruit and vegetables manufacture about 6.14% of this amount (http://elasaala.blogspot.com/2012/01/blog-post_2703.html). Processing of fruits and vegetables are resulting in high amounts of waste materials/by-products such as peels, seeds, stones, meals etc. It is well known that agro-industrial by-products are rich in dietary fibers, some of which contain appreciable amounts of colorants, antioxidant compounds or other substances with positive health effects, while some of them, like the oilseed meals, are rich in proteins. Some major source of food by-products are tomato, potatoes, cauliflower and eggplant some of the most popular vegetables and fruits. Tomato (*Lycopersicon esculentum* L.) juice is the most important vegetable juice with respect to per capita consumption. About 3-7% of the raw material is lost as waste during tomato juice pressing (Otto and Sulc, 2001). Tomato pomace consists of the dried and crushed skins



and seeds of the fruit (Avelino *et al.*, 1997). Potato (*Solanum tuberosum* L.) is the largest vegetable crop worldwide, amounting to approximately 320 million metric tons annually. Processing of potatoes (mainly for the production of chips, French fries, and dehydrated products) has presented a steady increase during the last decades, exceeding considerably the amount of the vegetable consumed as fresh. Solid waste generated during processing consists mostly of potato peels amounts to 15–20% depending on the procedure applied. Cauliflower *Brassica L. var. Botrytis* belongs to cruciferous family *Cruciferae (Brassicaceae)*, which comprises also: cabbage, broccoli, Brussels sprouts, turnip, Swedish turnip. Cauliflower leaves considered as a waste by-product which obtained it during processing (freezing and cooking) of Cauliflower, huge amount of leaves is generated, and its disposal is a major problem and causes environmental pollution. Leaves constitutes about 40-50% of cauliflower fruit. Eggplant, (*Solanum melongena*) one of the most widespread vegetable consumed around the world. Eggplant peel is usually treated as waste, i.e. byproduct, omitting its potentially beneficial characteristics. Due to higher vitamin C and phenolic compounds contents, many investigations suggested on possible use of eggplant peel as natural ingredient for functional products formulation (Cao *et al.*, 1996, Esther *et al.*, 2013 and Sepideh *et al.*, 2016).

Many studies reported that all of the previous by-products are rich sources of bioactive compounds including vitamins (C, E and β -carotene), polyphenols, organo-sulphur compounds, dietary fiber etc (Kadivec *et al.*, 2015 and Elhassaneen *et al.*, 2016-a and Salama *et al.*, 2017). Varied bioactive components at different levels may be responsible for the offered health protection. A number of experiments indicate that such by-products added to laboratory animals diet had positive effects on serum lipid profile, liver and kidney functions and serum glucose (Coskun *et al.*, 2005). In the present study we will try to open new avenue for extending the using of such four food processing by-products (potato peel, cauliflower leaves, onion skin, and mango peel) in therapeutic nutritional applications through mixing them in loaves bread to improve the obesity disease complications in rats. The association between oxidative stress and obesity are discussing by many authors (Bakker *et al.*, 2000; Chaitanya *et al.*, 2010 and Le Lay *et al.*, 2014). Oxidative stress appears as a major contributor in the development of many metabolic complications associated obesity. Therefore, therapeutics designed to lower ROS production may have beneficial effects on health. Practically, many therapeutically strategies used currently to treat obesity-associated metabolic disorders have the potential to decrease OS, which might, at least partially, participate in their beneficial effect. Unfortunately, data related to this issue is still in dearth. Therefore, the present work was carried out to investigate the status of blood oxidant and antioxidant in obese rats. Also, the effect of feeding some selected food processing by-products rich in bioactive compounds (acts as natural antioxidants) on that status will be in the scope of this investigation.

Materials and Methods

Materials

Food processing by-products: Potato (*Solanum tuberosum* L.) peel was obtained from SFCO For Manufacturing & Export Agricultural Products, El Negila, Kom Hamada, Behira Government, Egypt while tomato (*Lycopersicon esculentum* L.) peel from Faragalla Company for Food Industries, Borg El-Arab, Alexandria, Egypt. Cauliflower (*Brassica oleracea* L. cv Copania) leaves and eggplant, (*Solanum*

melongena) fruits peel were obtained by special arrangement with some farmers in El Minia city, El Minia Governorate, Egypt. The collected samples were transported immediately to in cooling state to the laboratory and used immediately for dehydration process and powders preparation.

Chemicals: Vitamins standards (A, C, and E) and thiols compounds (GSH and GSSG) were purchased from Sigma Chemical Co., St. Louis, MO, USA. All other reagents and solvent were of analytical or HPLC grade were purchased from (Fisher, UK). Casein was obtained from Morgan Chemical Co., Cairo, Egypt. The rest of chemicals, reagents and solvents were of analytical grade and purchased from ElGhomhorya Company for Trading Drugs, Chemicals and Medical Instruments Trading Co., Cairo, Egypt).

Equipments: Throughout this study a SP Thermo Separation Products Liquid Chromatograph (Thermo Separation products, San Jose, CA, USA) was used with a Consta Metvic 4100 pump, a Spectra Series AS100, Spectra System UV 1000 UV/Visible Spectrophotometer Detector, Spectra System FL 3000 and a PC 1000 system software. The columns used (Alltech, Deerfield, IL, USA) were a Spherosorb ODC-2 (5 μ m, 150 x 4.6 mm I.d.) for glutathione fractions ; a reversed-phase water Adsorbosil C₁₈ (5 μ m, 100 mm x 4.6 mm I.d.) for vitamin C; and normal Ultrasphere Si (5 μ m, 250 mm x 4.6 mm I.d.) for analysis of vitamins A and E. Also, absorbance and fluorescence for different assays were measured using Labo-med. Inc., spectrophotometer, CA and Schematzu fluorescence apparatus, Japan, respectively.

Methods

Preparation of food by-products powder

Potato peel, eggplant peel and cauliflower leaves were washed while tomato pomace was directly dried in a hot air oven (Horizontal Forced Air Drier, Proctor and Schwartz Inc., Philadelphia, PA) at two stages 50 °C for 6 h followed by 40 °C for 10 hrs. All of the dried peels were milled separately in high mixer speed (Moulinex Egypt, Al-Araby Co., Egypt). The material that passed through an 80 mesh sieve was retained for use.

Biological Experiments

Animals

Animals used in this study, adult male albino rats (140 \pm 10 g per each) were obtained from Research Institute of Ophthalmology, Medical Analysis Department, Giza, Egypt.

Basal Diet

The basic diet prepared according to the following formula as mentioned by (AIN, 1993) as follow: protein (10%), corn oil (10%), vitamin mixture (1%), mineral mixture (4%), choline chloride (0.2%), methionine (0.3%), cellulose (5%), and the remained is corn starch (69.5%). The diet induced obesity (DIO) prepared according to Research Diets, Inc. NJ, as follow: casein, 80 mesh (23.3%), L-cystine (0.35%), corn starch (8.48%), maltodextrin (11.65%), sucrose (20.14%), soybean oil (2.91%), lard fat (20.69%), mineral mixture (1.17%), dicalcium phosphate (1.52%), calcium carbonate (0.64%), potassium citrate.1 H₂O (1.92%), vitamin mixture (1.17%), choline bitartrate (0.23%). The used vitamins and salt mixtures components were formulated according to Campbell, (1963) and Hegsted, (1941), respectively.

Experimental design

Biological experiments performed a complied with the rulings of the Institute of Laboratory Animal Resources, Commission on life Sciences, National Research Council (NRC, 1996). Rats (n= 42 rats), weight 139 ± 5.3 g per each, were housed individually in wire cages in a room maintained at 25 ± 2 °C and kept under normal healthy conditions. All rats were fed on basal diet for one-week before starting the experiment for acclimatization. After one week period, the rats were divided into two main groups, the first group (Group 1, 6 rats) still fed on basal diet and the other main group (36 rats) was feed with diet-induced obesity (DIO) for 8 weeks which classified into sex sub groups as follow: group (2), fed on DIO as a positive control; group (3), fed on DIO containing 7.5 % tomato pomace powder (TPP); group (4), fed on DIO containing 7.5 % potato peel powder (PPP); group (5), fed on DIO containing 7.5 % cauliflower leaves powder (CLP), group (6), fed on DIO containing 7.5% eggplant peel powder (EPP) and group (7): fed on DIO containing 7.5 % mixture, TPP, PPP, CLP and EPP by equal parts. Body weight gain (as percent of initial weight) was assayed every week in rats. The percent of by-products powder (7.5%) was selected according to the studies of and Elhassaneen *et al.*, (2016-a) and salama *et al.*, (2017).

Blood sampling

Blood samples were collected at the end of experiment period, 8 weeks, after 12 hours fasting using the abdominal aorta and rats were scarified under ether anesthetized. Blood samples were received into into glass centrifuge tubes, containing oxalate solution (1.34 %) as anticoagulant. After centrifugation at 3000 rpm for 10 min., plasma was with drown and used for the analysis of blood lipid parameters and vitamins. The erythrocyte residue was washed with three successive portions of sodium chloride solution (0.9 %) and then haemolysed with deionised water for 30 min. Haemolysate was then centrifuged at 30,000 rpm for 30 min. and the supernatant fractions was transferred to a clean test tube and analyzed of antioxidant enzymes (Stroev and Makarova, 1989).

Hematological analysis

Glutathione fractions

GSH and GSSG were determined by HPLC according to the method of McFarris and Reed (1987). In brief, 100 µl of aliquot were placed in 2 ml of 10% perchloric acid containing 1 mM bathophenanthroline disulfonic acid and homogenized. The homogenate was cold centrifuged at 10000 rpm for 5 min and the internal standard (γ -glutamyl glutamate) was added to the supernatant. A 250 µl aliquot of acidic extract was mixed with 100 µl of 100 mM iodoacetic acid in 0.2 mM cresol purple solution. The acid solution was brought to pH 8.9 by the addition of 0.4 ml of KOH (2 M) – KHCO₃ (2.4 M) and allowed to incubate in the dark at room temperature for 1 h to obtain S-carboxymethyl derivatives. The N-nitrophenol derivatization of the samples were taken overnight at 4 °C in the presence of 0.2 ml of 1% 1-fluoro-2,4-dinitrobenzene and injected onto the HPLC system.

Antioxidant enzymes

GSH-Px and CAT activities were measured as described by Splittgerber and Tappel, 1979, and Aebi, 1974, respectively. SOD activity was measured by Ransod kit (Randox laboratories mmited, Germany). GSH-Rd activity was determined according to the method recommended by the International Committee for Standardization in Haematology (ICSH, 1979). Activities of SOD and GSH-Px enzymes were expressed in international unit per milliliter erythrocyte sediment and one unit of SOD was expressed as the enzyme protein amount causing 50% inhibition in 2- (4-iodophenyl)-3 (4-nitrophenol) 5-phenyltetrazolium chloride (INTH₂) reduction rate.



Antioxidant vitamins

All vitamins (A, C, and E) were extracted and analyzed by HPLC techniques according to the methods of Epler *et al.*, (1993), Hung, *et al.* (1980) and Moeslinger *et al.*, (1994), respectively. Quantitative determination of each vitamin was determined from its respective peak area and corresponding response factor. The percent recoveries of vitamins were also studied by adding each vitamin to serum after sample preparation and HPLC determination. Under such chromatographic conditions, the Mean \pm SD values of vitamins A, C and E recoveries were 91.34 ± 2.3 , 81.95 ± 6.02 , and 89.92 ± 4.98 , respectively.

Nitrite determination

Nitrite was determined fluorometric such as described by Misko *et al.*, (1993). In brief: Ten μ l of freshly prepared 2,3-diaminonaphthalene (DAN, 0.05 mg/ml in 0.62 M HCl, protected from light) is added to 100 μ l of sample and mixed immediately. Nitrate standards (> 98% pure, Sigma) are routinely made fresh, dissolved in DI H₂O, and kept on ice prior to use. After 10 min incubation at 20 °C, the reaction was terminated with 5 μ l of 2.8 N NaOH. The intensity of the fluorescent signal produced by the product is maximized by the addition of base. Formation of the 2,3-diaminonaphthtriazole was measured using a Schematzu fluorescence apparatus with excitation at 365 nm and emission read at 450 nm with a gain setting at 100%.

Nitrite/nitrate detection

Plasma is filtered through an ultrafree microcentrifuge filter unit (14000 rpm for 15 min) to remove the hemoglobin resulting from cell lysis. The filtrate should contain mostly nitrate (recovery greater than 90%) due to the reaction of NO with the iron-heme center of the protein. Nitrate is converted to nitrite by the action of nitrate reductase (from *Aspergillus niger*, Sigma Chemical Co., St. Louis, MO, USA) such as follow: the sample is incubated with 40 μ M NADPH (to initiate the reaction) and 14 mU of enzyme in a final volume of 50 μ l of 20 mM Tris buffer (pH, 7.6). The reaction is terminated after 5 min at 20 °C by dilution with 50 μ l of water followed by addition of the DNA reagent for determination of nitrite. Nitrite levels in samples are then calculated by first subtracting the value of the enzyme blank (i.e., nitrate reductase plus NADPH) from the experimental and then calculating the value using a standard curve for nitrite to which NADPH has been added.

Thiobarbituric acid reactive substances (TBARS) content

TBARS were measured as described by Buege and Aust, (1978). Half milliliter of plasma were added to 1.0 ml of thiobarbituric acid reagent, consisting of 15% TCA, 0.375% thiobarbituric acid (TBA) and 0.01% butylated hydroxytoluene in 0.25 N HCl. Twenty-five microliters of 0.1 M FeSO₄.7H₂O was added and the mixture was heated for 20 min in boiling water. The samples were centrifuged at 1000 xg for 10 min and the absorbance was read at 535 nm using Labo-med. Inc., spectrophotometer against a reagent blank. The absorbance of the samples was compared to a standard curve of known concentrations of malonicdialdehyde.

Statistical Analysis

All measurements were done in triplicate and recorded as mean \pm standard deviation (SD). Statistical analysis was performed with the Student *t*-test and MINITAB-12 computer program (Minitab Inc., State College, PA).

Results and Discussion

Effect of feeding the selected food processing by-products on body weight gain (BWG) of obese rats

The effect of feeding the selected food processing by-products on body weight (Percent of baseline) of obese rats was shown in Figure (1). From such data it could be noticed that feeding of rats on diet induced obesity (DIO) leads to increase the BW than the control group. At the end of the experiment (8 weeks), rats of the obese group recorded 149.02% of the control (normal) group for the BWG. Feeding of tomato pomace powder (TPP), potato peel powder (PPP), cauliflower leaves powder (CLP), eggplant peel powder (EPP) and their mixture (Mix) induced significant decreasing on BWG of the obese rats which recorded 120.83, 138.32, 135.51, 127.65 and 119.02% as a percent of the baseline, respectively. The higher effect on weigh decreasing was recorded for the by-products mixture followed by TPP, EPP, CLP and PPP, respectively. The positive effects of the selected by-products plant parts i.e. TPP,

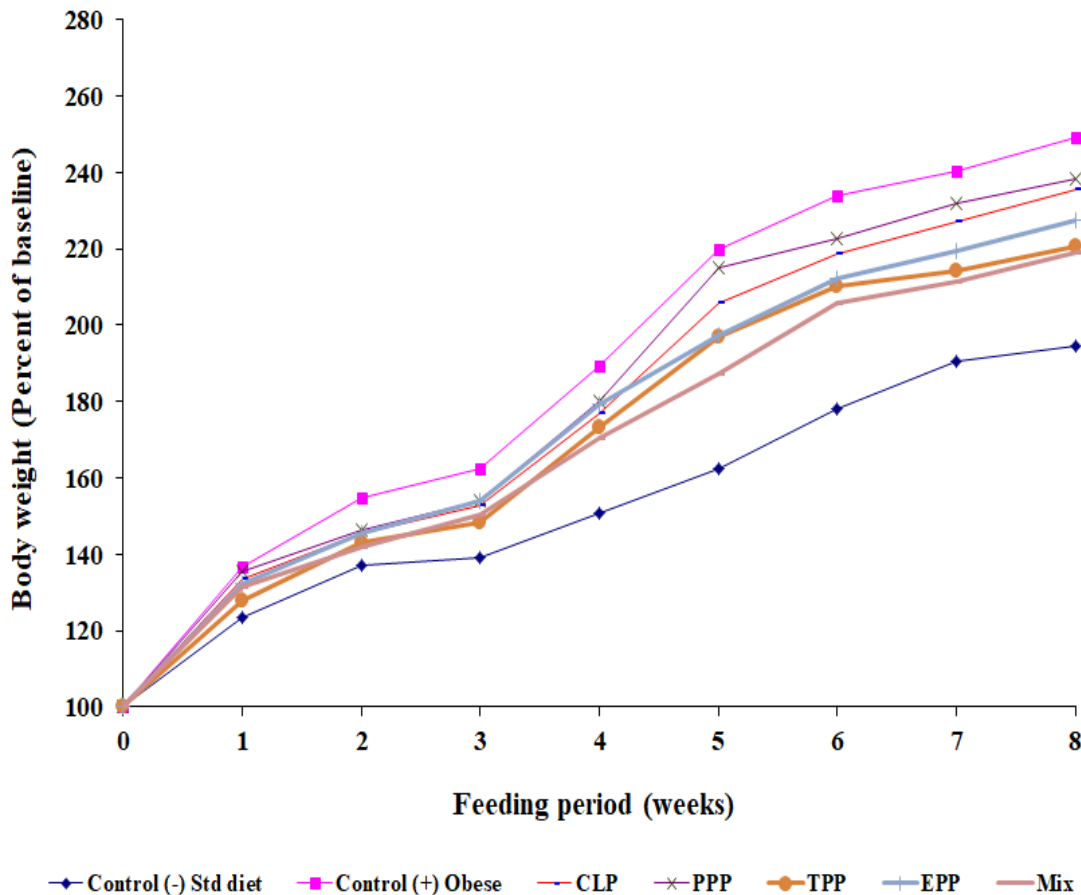


Figure 1. Effect of feeding the selected food processing by-products on body weight (Percent of baseline) of obese rats *

* TPP, Tomato pomace powder; PPP, potato peel powder; CLP, cauliflower leaves powder; EPP, Eggplant peel powder and Mix, mixture of TPP + PPP + CLP+ EPP by equal parts.

PPP, CLP, EPP and their mixture in reducing the BWG could be attributed to their high level content of different classes bioactive compounds called phytochemicals (Le Lay *et al.*, 2014). Previous studies indicated that such selected plant parts were rich in several classes of phytochemicals including flavonols, phenolic acids, anthocyanins, alkaloids, carotenoids, phytosterols and organosulfur compounds (Kadivec *et al.*, 2015 and Elhassaneen *et al.*, 2016-a and Salama *et al.*, 2017). Such bioactive compounds and their conversion products have been shown to induce/participate in several mechanisms which contribute to their action control of adipocyte function, adiposity subsequently obesity (Bonet *et al.*, 2015). Amongst of these mechanisms, they could be interacted with several transcription factors of the nuclear receptor superfamily, interfered with the activity of other transcription factors, modulated signaling pathways which are associated with inflammatory and OS responses; and scavenged of reactive species such reactive oxygen species (ROS) and reactive nitrogen species (RNS) (Le Lay *et al.*, 2014 and Bonet *et al.*, 2015).

Effect of feeding the selected food processing by-products on blood oxidants concentration of obese rats

Effect of feeding the selected food processing by-products on blood oxidants (thiobarbituric acid reactive substances, TBARS and nitric oxides, NO₂ and NO₃) concentration of obese rats was shown in Table (1) and Figure (2). From such data it could be noticed that DIO induced a significant ($p \leq 0.05$) increased in TBARS, NO₂ and NO₂/NO₃ concentrations in plasma by 39.10, 31.02 and 27.01 % compared to normal control group, respectively. Supplementation of the rat diets with 7.5% w/w by TPP, PPP, CLP, EPP and their mixture induced significant ($p \leq 0.05$) decreasing on these parameters concentration in plasma by the ratio of -25.87, -18.41, -16.67, -21.89 and -27.36%; -20.29, -14.96, -13.00, -19.17 and -21.70%; and -16.67, -13.60, -10.54, -13.98 and -20.31%, respectively. The higher effect was recorded for the by-product mixtures treatment followed by TPP, EPP, CLP and PPP, respectively. Such data are partially in accordance with that reported by Sayed Ahmed, (2016). Also, Elhassaneen-a and Salem, (2014) reported that clinical evidences for obesity-associated OS have been provided by measurement of either biomarkers or end-products of free radical-mediated oxidative processes. For instance, lipid peroxidation markers such as malondialdehyde (MDA), one of the most important compounds in TBARS and major products of the oxidation of polyunsaturated fatty acids, lipid hydroperoxides and conjugated dienes are found to be increased in plasma from obese subjects in many clinical studies (Vincent and Taylor, 2006). On the other side, systemic metabolic alterations associated with obesity contribute to the increase in OD have been reported by many authors. Aso, hyperglycemia as a hallmark of type II diabetes, a metabolic complication of obesity, induces OS through activation of the polyol and hexosamine pathways, production of advanced glycation end-products (AGE), and increase of diacylglycerols (DAG) synthesis (DCCTRG, 1993 and Le Lay *et al.*, 2014). Excess of circulating lipids induces ROS formation pathways, which contribute to the increase in lipid oxidation and protein carbonylation (Jensen *et al.*, 1989). Furthermore, leptin and angiotensin II, secreted at high levels by adipocytes, are inducers of ROS generation and might therefore promote inflammation and lipid peroxidation (Bouloumie *et al.*, 1999). Regarding the RNS, Endothelial NO synthase- (eNOS-) and inducible NO synthase- (iNOS-) dependent NO are abundant in adipocytes. iNOS expression has been shown to be increased in white adipose tissue (WAT) derived from diet- induced or genetic models of obesity (Perreault and Marette, 2001). Similarly, both eNOS and iNOS are expressed at higher levels in WAT from obese patients compared to lean controls (Elizalde *et al.*, 2000 and Engeli *et al.*, 2004).

Table 1. Effect of feeding the selected food processing by-products on blood oxidants concentration of obese rats*

Value	Control (-)	Control (+)	By-products (7.5%, W/W)				
			TPP	PPP	CLP	EPP	Mix
Thiobarbituric acid reactive substances (TBARS, nmol/mL)							
Range	1.87-4.11	3.67-5.02	2.11-3.78	2.69-4.11	2.97-5.01	2.63-3.89	2.11-3.89
Mean	2.89 ^b	4.02 ^a	2.98	3.28 ^a	3.35 ^a	3.14 ^a	2.92 ^b
SD	0.55	0.62	0.44	0.62	0.86	0.87	0.49
% of Change	0.00	39.10	-25.87	-18.41	-16.67	-21.89	-27.36
Nitrite (NO₂, nmol/L)							
Range	1.89-3.11	2.87-5.01	1.99-3.12	2.01-3.76	2.78-3.98	2.11-3.76	2.45-3.87
Mean	2.72 ^b	3.56 ^a	2.84 ^b	3.03 ^a	3.10 ^a	2.88 ^b	2.79 ^b
SD	0.55	0.76	0.87	0.56	0.13	0.3	0.24
% of Change	0.00	31.02	-20.29	-14.96	-13.00	-19.17	-21.70
Nitrite/Nitrate (NO₂/NO₃, nmol/L)							
Range	3.89-5.11	4.11-6.67	3.98-5.78	3.94-5.86	4.01-5.52	3.51-4.81	3.55-4.65
Mean	4.11 ^{ab}	5.22 ^a	4.35 ^{ab}	4.51 ^a	4.67 ^a	4.49 ^a	4.16 ^{ab}
SD	0.66	1.04	1.11	0.89	0.73	0.83	0.59
% of Change	0.00	27.01	-16.67	-13.60	-10.54	-13.98	-20.31

*TPP, Tomato pomace powder; PPP, potato peel powder; CLP, cauliflower leaves powder; EPP, Eggplant peel powder and Mix, mixture of TPP + PPP + CLP+ EPP by equal parts. Means in the same row with different superscript letters are significantly different at p ≤ 0.05.

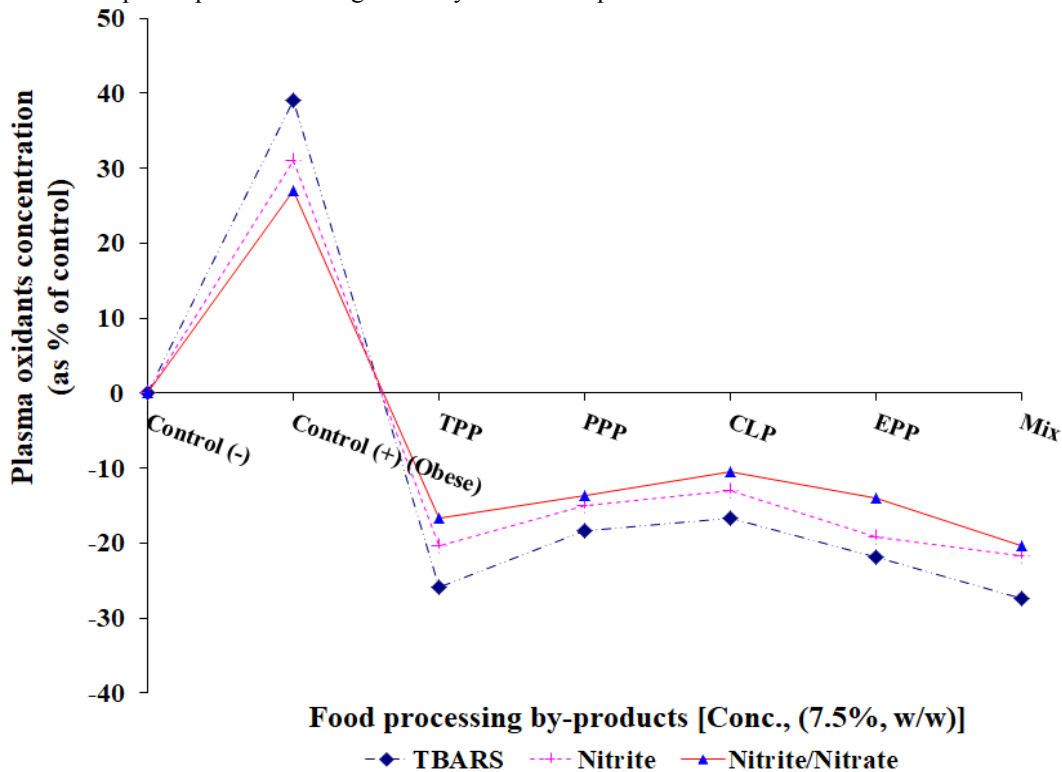


Figure 2. Effect of feeding the selected food processing by-products on blood oxidants concentration of obese rats*

*TPP, Tomato pomace powder; PPP, potato peel powder; CLP, cauliflower leaves powder; EPP, Eggplant peel powder and Mix, mixture of TPP + PPP + CLP+ EPP by equal parts.



Long time ago, interest in the possible significance of MDA on human health has been stimulated by reports that are mutagenic and carcinogenic compound (Elhassaneen and Tawfik, 1998). Nitric oxide synthase catalyzes the conversion of L-arginine to citrulline and highly reactive free radical species, nitric oxide (NO) (Manahan, 1989). NO, in turn, can react with molecular oxygen and water to form nitrite and nitrate; with hemoglobin to form iron-nitrosyl adducts and/or nitrate in blood, with superoxide anion to make nitrate, and with the amino and thiol groups of protein to produce nitrosylated species (Manahan, 1989; Misko *et al.*, 1993). The excess production of nitric oxides has been implicated in the pathogenesis and tissue destruction of a growing number of immunological and inflammatory diseases including septic shock, arthritis, graft rejection and diabetes (Jacob *et al.*, 1992).

The positive effects of plant parts on oxidants formation/concentration of obese rats could be attributed to several mechanisms induced by their bioactive components content. In this context, Jung *et al.*, (2011) reported that hepatic oxidant stress was reduced by 1% onion peel extract, as assessed by increasing superoxide dismutase (SOD) activity and blocking MDA formation. Also, Coskun *et al.*, (2005) found that quercetin, dominant flavonoid such as found in our selected plant parts, have anti-oxidative and anti-inflammatory activities. Such dietary phenolics found in the selected plant by-products are metabolized in liver, inhibiting liver injury induced by diabetes i.e. enhancing lipid metabolism, reducing OS may be particularly effective, consequently. Additionally, the mixture treatment gave maximum reduction yield of plasma oxidants concentration when compared with the tested plant by-products plant separated. Such notice means that a combination of different plant by-products may be more efficient for reducing plasma oxidants concentration which probably due to the interactive effects occurred by their content of different categories of bioactive compounds.

Effect of feeding the selected food processing by-products on plasma glutathione fractions concentration of obese rats

Effect of feeding the selected food processing by-products on plasma glutathione fractions, biological antioxidant macromolecules, concentration of obese rats were assessed in Table (2) and Figure (3). From such data it could be noticed that consumption of DIO induced a significant ($p \leq 0.05$) decreased in GSH and GSSG concentrations and GSH/GSSG ratio in plasma by -35.45, -18.07 and -21.21% compared to normal control group, respectively. Supplementation of the rat diets with 7.5% w/w by 7.5% w/w by TPP, PPP, CLP, EPP and their mixture induced significant ($p \leq 0.05$) increasing on these parameters concentration in plasma by the ratio of 31.45, 17.97, 19.80, 26.79 and 38.94%; 11.76, 5.88, 7.35, 4.41 and 17.65%; and 17.61, 11.42, 11.59, 21.43 and 18.09%, respectively. The higher effect in plasma GSH and GSSG concentrations and GSH/GSSG ratio was recorded for the by-product mixtures treatment followed by TPP, EPP, CLP and PPP, respectively.

Such as mentioned in different studies, reduced glutathion (GSH) is a tripeptide-thiol (γ -glutamylcysteinyl-glycine) that has received considerable attention in terms of its biosynthesis, regulation, and various intracellular functions (Reed and Beatty, 1980; Larsson *et al.*, 1983). Among of these function are two constructing roles in detoxifications: (1) as a key conjugate of electrophilic intermediates,

Table 2. Effect of feeding the selected food processing by-products on plasma glutathione fractions concentration of obese rats*

Value	Control (-)	Control (+)	By-products (7.5%, W/W)				
			TPP	PPP	CLP	EPP	Mix
Reduced glutathione concentration (GSH, $\mu\text{mol/L}$)							
Range	7.10-12.76	5.01-8.30	6.29-8.77	5.71-8.20	6.03-8.94	5.21-9.52	5.87-10.63
Mean	9.31 ^a	6.01 ^c	7.90 ^{ab}	7.09	7.20 ^{ab}	7.62 ^{ab}	8.35 ^{ab}
SD	2.11	1.78	0.96	0.69	1.14	1.51	2.01
% of Change	0.00	-35.45	31.45	17.97	19.80	26.79	38.94
Oxidized glutathione concentration (GSSG, $\mu\text{mol/L}$)							
Range	0.73-0.96	0.54-0.91	0.55-0.71	0.63-0.77	0.61-0.88	0.60-0.78	0.67-1.05
Mean	0.83 ^a	0.68 ^{ab}	0.76 ^{ab}	0.72 ^{ab}	0.73 ^{ab}	0.71 ^{ab}	0.80 ^a
SD	0.11	0.20	0.21	0.13	0.19	0.10	0.23
% of Change	0.00	-18.07	11.76	5.88	7.35	4.41	17.65
GSH/GSSG ratio							
Range	9.76-13.76	5.98-9.65	9.01-12.65	6.23-12.67	7.50-10.33	8.56-11.65	8.55-13.87
Mean	11.22 ^a	8.84 ^{ab}	10.39 ^a	9.85 ^{ab}	9.86 ^{ab}	10.73 ^a	10.44 ^a
SD	2.66	3.21	0.89	2.08	3.05	2.56	1.73
% of Change	0.00	-21.21	17.61	11.42	11.59	21.43	18.09

*TPP, Tomato pomace powder; PPP, potato peel powder; CLP, cauliflower leaves powder; EPP, Eggplant peel powder and Mix, mixture of TPP + PPP + CLP+ EPP by equal parts. Means in the same row with different superscript letters are significantly different at $p \leq 0.05$.

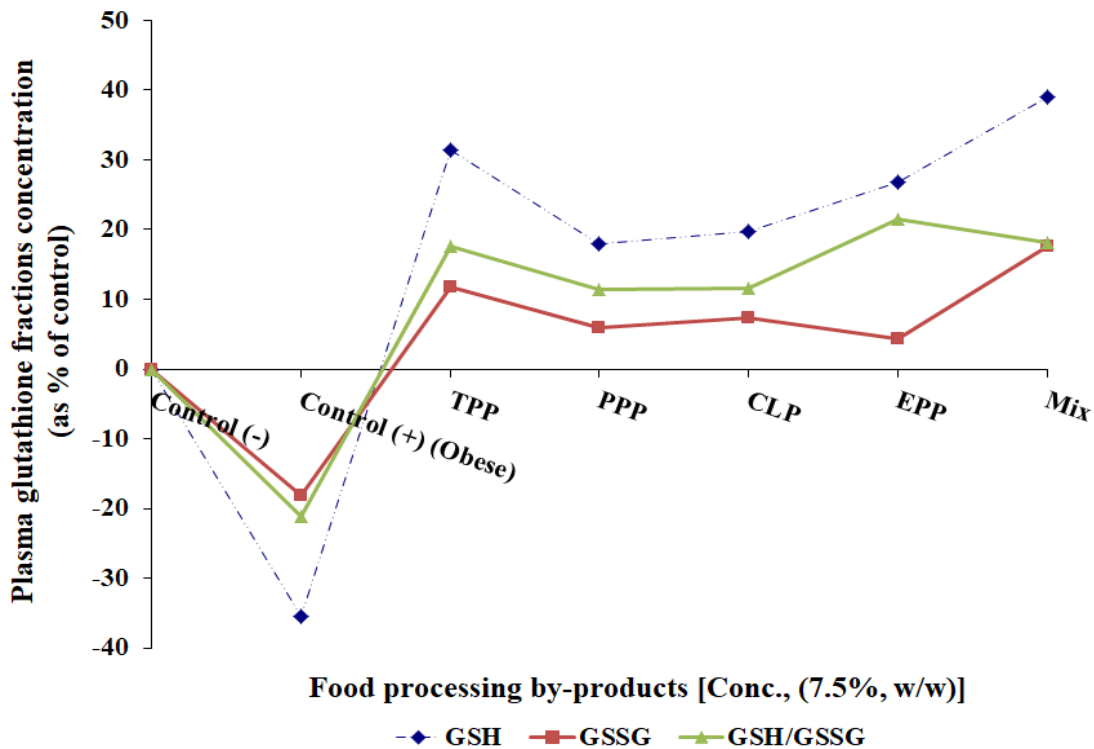


Figure 3. Effect of feeding the selected food processing by-products on plasma glutathione fractions concentration of obese rats*

*TPP, Tomato pomace powder; PPP, potato peel powder; CLP, cauliflower leaves powder; EPP, Eggplant peel powder and Mix, mixture of TPP + PPP + CLP+ EPP by equal parts.

principally via glutathione-*s*-transferase activities in phase II metabolism, and (2) as an important antioxidant. The antioxidant functions of GSH includes its role in the activities of GSH enzymes family including glutathione peroxidase (GSH-Px) and peroxiredoxins (PRXs). In addition, GSH can apparently serve as a nonenzymatic scavenger of oxyradicals (Halliwell and Gutteridge, 1985 and Elhassaneen *et al.*, 2016-a).

A fall in glutathione fractions observed in obese rats group generally accompanied by a concomitant decreased in the ratio of GSH/GSSG. In this context, Di Giulio (1991) mentioned that a more fundamental effect of oxyradical-generating compounds as the obesity development, however, is their effect on what can be referred to as the redox status (GSH/GSSG) of cells or tissues. Also, Elhassaneen, (2004) mentioned that increased fluxes of oxyradicals might be decreased in the GSH/GSSG ratio, due either to direct radical scavenging or to increased peroxidase activity. This effect could also occur indirectly due to reduced NADPH availability [necessary for glutathione reductase (GSH-Rd) activity] resulting, for example, from oxidations in the first step of the redox cycle (Champe and Harvey, 1994 and Bedard, and Krause, 2007). In regarding studies, Bedard and Krause (2007) reported that various enzymes inside the cells including adipocytes can also produce ROS. Particularly, the family of NADPH oxidases (NOX) is considered to be an important source of ROS generation. Such effect could be one of the most important reasons for reducing the GSH/GSSG ratio in obese rats. Therefore, we could expected that the food processing by-products selected in the present study and their mixtures feeding are rich in bioactive compounds which exhibited antioxidant effects against ROS formation as the obesity development through several mechanism of action including the raising of redox status (GSH/GSSG ratio) in the body.

Effect of feeding the selected food processing by-products on erythrocytes antioxidant enzymes activities of obese rats

Antioxidant status (glutathione peroxidase, GSH-Px, glutathione reductase, GSH-Rd, catalase, CAT and superoxide dismutase, SOD) in erythrocytes of obese rats feeding the selected food processing by-products was illustrated in Table (3) and Figure (4). From such data it could be noticed that DIO induced a significant ($p \leq 0.05$) increased in GSH-Px, GSH-Rd, CAT and SOD activities in erythrocytes by -39.35, -31.52, -29.65 and -25.03% compared to normal control group, respectively. Supplementation of the rat diets with 7.5% w/w by TPP, PPP, CLP, EPP and their mixture induced significant ($p \leq 0.05$) decreasing on these parameters concentration in plasma by the ratio of 36.05, 22.35, 26.53, 28.12 and 40.37%; 30.11, 20.74, 23.01, 24.74 and 35.64; 29.87, 22.29, 23.88, 26.34 and 31.95%; and 20.26, 12.99, 14.38, 17.77 and 23.87%, respectively. The higher effect in GSH-Px, GSH-Rd, CAT and SOD activities of erythrocytes was recorded for the by-product mixtures treatment followed by TPP, EPP, CLP and PPP, respectively.

Several years ago, Thomas *et al.*, (1990) mentioned that to prevent free radical damages/OS activities, the organism has developed antioxidant defense system largely based on antioxidant enzymes which able to scavenge ROS. Such system consists of SODs which are responsible for the reduction of O_2^- to H_2O_2 and multiple enzymes will remove H_2O_2 including GSH-Px and CAT. Additionally, GSH-Rd enzyme which catalyze the reaction: $GSSG + NADPH + H^+ \rightarrow 2GSH + NADP^+$. GSH-Rd can also catalyse reduction of certain mixed disulphides such as that between



Table 3. Effect of feeding the selected food processing by-products on erythrocytes antioxidant enzymes activities of obese rats*

Value	Control (-)	Control (+)	By-products (7.5%, W/W)				
			TPP	PPP	CLP	EPP	Mix
Glutathione peroxidase (GSH-Px, U/g Hb)							
Range	19.03-24.10	11.67-15.52	14.71-22.04	10.62-20.62	14.01-20.65	15.98-18.55	14.50-22.99
Mean	22.87 ^a	13.87 ^c	18.87 ^{ab}	16.97 ^{ab}	17.55 ^{ab}	17.77 ^{ab}	19.47 ^{ab}
SD	2.89	3.77	3.89	5.21	3.54	1.45	4.11
% of Change	0.00	-39.35	36.05	22.35	26.53	28.12	40.37
Glutathione reductase (GSH-Rd, U/g Hb)							
Range	12.01-16.03	7.04-12.78	8.51-16.66	9.01-13.05	9.01-15.98	8.63-15.21	9.02-17.03
Mean	14.00 ^a	9.58 ^c	12.47 ^{ab}	11.57 ^{ab}	11.79 ^{ab}	11.96 ^{ab}	13.00 ^a
SD	1.32	2.09	3.21	2.87	3.77	4.10	3.80
% of Change	0.00	-31.52	30.11	20.74	23.01	24.74	35.64
Catalase (CAT, U/g Hb)							
Range	166.63-226.76	120.78-178.76	153.89-194.73	140.73-198.03	135.78-205.78	154.02-191.02	160.54-210.45
Mean	197.36 ^a	138.85 ^d	180.32 ^{ab}	169.80 ^{ab}	172.00 ^c	175.42 ^c	183.21 ^{ab}
SD	21.76	30.76	18.45	23.74	32.87	20.54	19.92
% of Change	0.00	-29.65	29.87	22.29	23.88	26.34	31.95
Superoxide dismutase (SOD, U/g Hb)							
Range	3.19-4.22	2.56-3.61	3.11-3.90	2.39-3.88	2.56-4.11	2.89-4.22	3.13-4.55
Mean	4.02 ^a	3.01 ^{ab}	3.62 ^a	3.40 ^a	3.45 ^a	3.55 ^a	3.73 ^a
SD	0.42	0.41	0.29	0.71	1.02	0.62	0.54
% of Change	0.00	-25.03	20.26	12.99	14.38	17.77	23.87

*TPP, Tomato pomace powder; PPP, potato peel powder; CLP, cauliflower leaves powder; EPP, Eggplant peel powder and Mix, mixture of TPP + PPP + CLP+ EPP by equal parts. Means in the same row with different superscript letters are significantly different at $p \leq 0.05$.

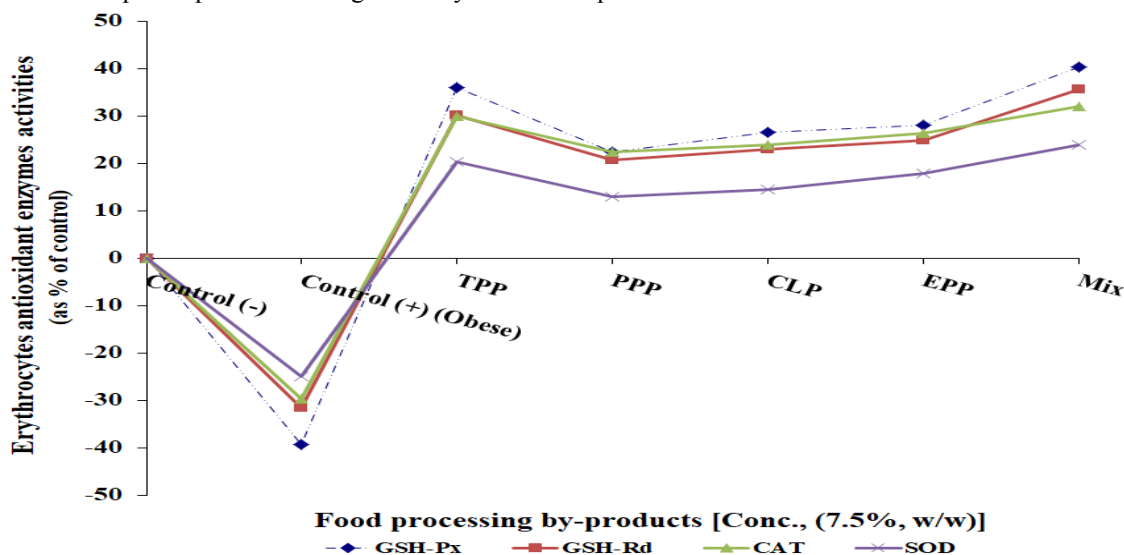


Figure 4. Effect of feeding the selected food processing by-products on erythrocytes antioxidant enzymes activities of obese rats*

*TPP, Tomato pomace powder; PPP, potato peel powder; CLP, cauliflower leaves powder; EPP, Eggplant peel powder and Mix, mixture of TPP + PPP + CLP+ EPP by equal parts.

GSH and Co-enzyme. Many studies such Galinier *et al.*, (2004) and Cao, (2014) reported that antioxidant enzymes systems are active in fat cells isolated from rats, although their activities are lower than in liver. Their activities might moreover depend on fat pad localization in as much as redox status differences have been reported between epididymal and inguinal fat pads from obese Zucker rats (Galiniere *et al.*, 2006). Despite the presence of active antioxidant systems, obese state is associated with a decrease in antioxidant defenses including the enzymatic system. For instance, a decrease in expression and activities of antioxidant enzymes such as SOD, GSH-Px or CAT have been reported in WAT from obese mice models (Furukawa *et al.*, 2004).

The food processing by-products selected in the present study and their mixtures feeding are rich in bioactive compounds such phenolics, organosulphur compounds, carotenoids etc which exhibited antioxidant activities in different biological systems (Kadivec *et al.*, 2015 and Elhassaneen *et al.*, 2016-a and Salama *et al.*, 2017). Such antioxidant properties are important in manipulation of the obesity development through increasing the antioxidant enzymes activities in fat cells subsequently increasing the ROS scavenging processes.

Effect of feeding the selected food processing by-products on plasma antioxidant vitamins concentration of obese rats

Data of the effect of feeding the selected food processing by-products on plasma antioxidant vitamins concentration of obese rats were shown in Table (4) and Figures (5). From such data it could be noticed that DIO induced a significant ($p \leq 0.05$) decreased in vitamins A, C and E in plasma by -27.43, -18.39 and -23.04% compared to normal control group, respectively. Supplementation of the rat diets with 7.5% w/w by TPP, PPP, CLP, EPP and their mixture induced significant ($p \leq 0.05$) increasing on these parameters concentration in plasma by the ratio of 25.20, 16.89, 18.92, 20.32 and 29.32%; 11.68, 7.35, 9.30, 10.07 and 13.40; and 17.88, 11.12, 13.94, 16.70 and 21.86%, respectively. The higher effect in plasma vitamins A, C and E concentration was recorded for the by-product mixtures treatment followed by TPP, EPP, CLP and PPP, respectively.

Such data indicated that as a consequence of obesity injury the reducing in antioxidant enzymes defense potential of erythrocytes was contrary with significant decreasing ($p > 0.05$) in antioxidant vitamins in plasma. Beside the erythrocytes, adipose tissue represents also a preferential storage site for natural antioxidants compounds, as lipid soluble vitamins (e.g., vitamins A and E) or β -carotenoids (Landrier *et al.*, 2012). However, obese people present generally a relatively low total antioxidant status characterized by lower levels of serum vitamins A, E, C and β -carotene (Neuhouser *et al.*, 2001; Elhassaneen and Salem 2015). Although adipose tissue storage generally equilibrates with circulating levels of molecules (Parker, 1989 and Blum *et al.*, 2008), fat can also act as sink concentrating vitamins in adipocyte lipid droplets therefore limiting their bioavailability (Traber and Kayden, 1987). Similar studies indicated that food processing by-products selected in the present study and their mixtures are rich in bioactive compounds including carotenoids and vitamins which exhibited antioxidant activities in different biological systems (Elhassaneen *et al.*, 2016-a,b). Such antioxidant properties are important in manipulation of the obesity development through ROS scavenging processes subsequently excess the bioavailability of the lipid soluble vitamins in fat cells.

Table 4. Effect of feeding the selected food processing by-products on plasma antioxidant vitamins concentration of obese rats*

Value	Control (-)	Control (+)	By-products (7.5%, W/W)				
			TPP	PPP	CLP	EPP	Mix
Vitamin A (Retinol, $\mu\text{mol/L}$)							
Range	1.231-1.412	0.995-1.239	0.982-1.351	1.052-1.351	1.056-1.352	0.894-1.472	1.053-1.610
Mean	1.397 ^a	1.014 ^{ab}	1.270 ^a	1.185 ^{ab}	1.206 ^{ab}	1.220 ^{ab}	1.311 ^a
SD	0.230	0.110	0.310	0.120	0.173	0.310	0.231
% of Change	0.00	-27.43	25.20	16.89	18.92	20.32	29.32
Vitamin C (Ascorbic acid, $\mu\text{mol/L}$)							
Range	48.88-70.53	40.76-61.78	52.87-61.34	51.87-61.20	50.72-66.20	50.53-66.09	44.87-63.99
Mean	62.90 ^a	51.33 ^b	57.32 ^a	55.10 ^a	56.11 ^a	56.50 ^a	58.21 ^a
SD	10.54	9.74	5.12	4.88	9.53	8.29	10.62
% of Change	0.00	-18.39	11.68	7.35	9.30	10.07	13.40
Vitamin E (Tocopherol, $\mu\text{mol/L}$)							
Range	36.11-46.14	28.55-37.88	29.76-44.52	30.65-37.10	33.78-38.72	32.00-41.75	34-51-44.84
Mean	40.78 ^a	31.38 ^c	36.99 ^a	34.87 ^{ab}	35.75 ^{ab}	36.62 ^{ab}	38.24 ^a
SD	4.76	5.12	6.26	3.88	2.54	4.81	6.45
% of Change	0.00	-23.04	17.88	11.12	13.94	16.70	21.86

*TPP, Tomato pomace powder; PPP, potato peel powder; CLP, cauliflower leaves powder; EPP, Eggplant peel powder and Mix, mixture of TPP + PPP + CLP+ EPP by equal parts. Means in the same row with different superscript letters are significantly different at $p \leq 0.05$.

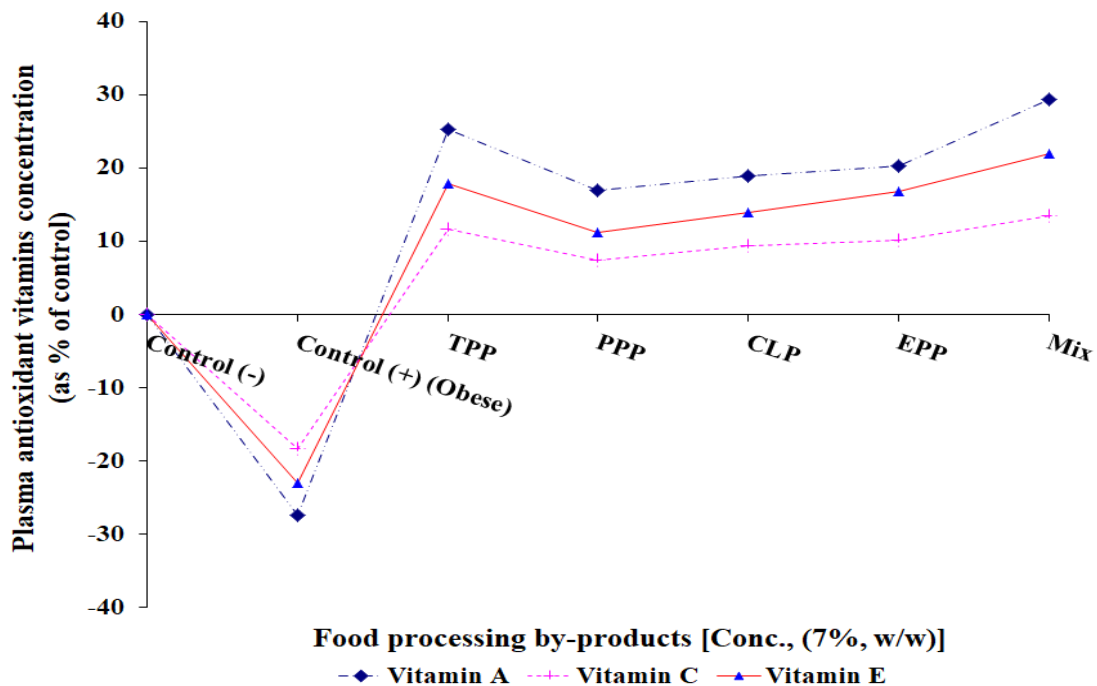


Figure 5. Effect of feeding the selected food processing by-products on plasma antioxidants vitamins concentration of obese rats*

*TPP, Tomato pomace powder; PPP, potato peel powder; CLP, cauliflower leaves powder; EPP, Eggplant peel powder and Mix, mixture of TPP + PPP + CLP+ EPP by equal parts.



Correlation studies

Correlation analysis indicated that important differences were found between oxidant and antioxidant status in obese rats plasma feeding the selected food processing by-products (TPP, PPP, CLP, EPP and their mixture) (Table 5). From such data it could be noticed that there was a strong negative significant ($p \leq 0.01$) relationship between GSH concentration in plasma ($r^2 = -0.9192$), antioxidant vitamins in plasma [vitamin A ($r^2 = -0.7515$) and vitamin E ($r^2 = -0.8683$)], antioxidant enzymes in RBC's [GSH-Px ($r^2 = -0.9076$), CAT ($r^2 = -0.8442$) and SOD ($r^2 = -0.8062$)] and TBARS concentration in plasma. While, moderate negative significant ($p \leq 0.01$) relationship between water soluble antioxidant vitamins in plasma [vitamin C ($r^2 = -0.6256$) and TBARS concentration in plasma. On the same time, there was a strong negative significant ($p \leq 0.01$) relationship between GSH concentration in plasma ($r^2 = -0.8554$), antioxidant vitamins in plasma [vitamin A ($r^2 = -0.7730$) and vitamin E ($r^2 = -0.8392$)], antioxidant enzymes in RBC's [GSH-Px ($r^2 = -0.8538$), CAT ($r^2 = -0.8053$) and SOD ($r^2 = -0.8641$)] and NO_2 concentration in plasma. While, moderate negative significant ($p \leq 0.01$) relationship between water soluble antioxidant vitamins in plasma [vitamin C ($r^2 = -0.6142$) and NO_2 concentration in plasma. These correlations confirm that if there were no change in the antioxidant status of obese rats, it would be difficult to observe high concentrations of TBARS and NO_2 . In similar study, Lepage *et al.*, (1996) reported that high levels of MDA in the plasma of obese patients were associated with rather low levels of β -carotene. Also, in some model systems, a combination of α -tocopherol and β -carotene interact synergistically to inhibit lipid peroxidation subsequently increased TBARS (Bohm *et al.*, 1997).

Table 5. Correlation between oxidant stress and antioxidant defense system parameters in obese rats feeding the selected food processing by-products

Parameters	R ^{2*}	Parameters	R ^{2*}
TBARS/GSH	- 0.9192	NO2/GSH	- 0.8554
TBARS/ Vit A	- 0.7515	NO2/ Vit A	- 0.7730
TBARS/Vit C	- 0.6256	NO2/Vit C	- 0.6142
TBARS/Vit E	- 0.8683	NO2/Vit E	- 0.8392
TBARS/GSH-Px	- 0.9076	NO2/GSH-Px	- 0.8538
TBARS/CAT	- 0.8442	NO2/CAT	- 0.8053
TBARS/SOD	- 0.8062	NO2/SOD	- 0.8641

* $P \leq 0.01$

Conclusion

Associations between obesity and markers of oxidant/antioxidant status have been observed in rats. This is confirmed by the results of this study, which showed that obese development causes' reduction in the enzymatic antioxidant defense potential of erythrocytes and non-enzymatic antioxidant in plasma. It was accompanied by a concomitant high concentration of different oxidants in plasma including TBARS and nitric oxides. Feeding of plant processing by-products has been proven to be essential in the treatment and/or prevention of obesity but also beneficial for oxidant status (OS) reduction. Overall, the present study supports the benefits of dietary modification, including bioactive compounds in plant parts supplementation, in alleviating OS associated obesity.

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تأثير التغذية ببعض النواتج الثانوية للتصنيع الغذائي على حالة الأكسدة ومضادات الأكسدة لدم الفئران المصابة بالسمنة

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الملخص

تهدف الدراسة الحالية إلى استكشاف تأثير التغذية ببعض النواتج الثانوية للتصنيع الغذائي على حالة الأكسدة ومضادات الأكسدة لدم الفئران المصابة بالسمنة. لذلك تم تقسيم عدد ٤٢ فأر (متوسط أوزانهم 139 ± 5.3 جم للفأر الواحد) إلى مجموعتين رئيسيتين، المجموعة الأولى (مجموعة ١، ٦ فئران) تم تغذيتها على الغذاء الأساسي، والمجموعة الرئيسية الأخرى (٣٦ فأر) تم تغذيتها على نظام غذائي يسبب السمنة (DIO) لمدة ٨ أسابيع، تم تقسيمها فيما بعد إلى ستة مجموعات فرعية على النحو التالي: المجموعة (٢) تم تغذيتها على غذاء (DIO) كمجموعة ضابطة موجبة، اما المجموعات (٣ - ٧) تم تغذيتها على غذاء DIO يحتوي على ٧.٥٪ من مسحوق قفل الطماطم، قشر البطاطس، مسحوق اوراق القنبيط، مسحوق قشر الباذنجان، ومسحوق مخلوطهم (بنسب متساوية) على التوالي. وفي نهاية فترة التجربة (٨ اسابيع) سجلت أوزان الفئران المصابة بالسمنة زيادة في الوزن مقدارها ١٤٩.٠٢٪ من خط البداية. بينما أدت التغذية على المساحيق السابقة الى حدوث نقص غى النسبة السابقة لتسجل ١٢٠.٨٣، ١٣٨.٣٢، ١٣٥.٥١، ١٢٧.٦٥، ١١٩.٠٢ بالنسبة لمساحيق قفل الطماطم، قشر البطاطس، اوراق القنبيط، قشر الباذنجان، ومخلوطهم على التوالي. كما أظهرت نتائج التحاليل الحيوية الى أن الاصابة بالسمنة قد ادت إلى ارتفاع معنوي في تركيز المؤكسدات بالبلازما (حمض الثايوبريبوتك بمعدل ٣٩.١٠٪، النيتريت بمعدل ٣١.٠٢٪ وتركيز النيتريت/النترات ٢٧.١٠٪)، وانخفاض معنوي في كل من مضادات الاكسدة الغير انزيمية (الجلوتاثيون في صورته المختزلة بمعدل ٣٥.٤٥٪ والجلوتاثيون في صورته المؤكسدة بمعدل ١٨.٠٧٪)، والفيتامينات المضادة للاكسدة (فيتامين (أ) بمعدل ٢٧.٤٣٪، فيتامين (ج) بمعدل ١٨.٣٩٪ و فيتامين (هـ) بمعدل ٢٣.٠٤٪) وكذلك في مستوي الانزيمات المضادة للأكسدة في كرات الدم الحمراء (انزيم الجلوتاثيون بيروكسيداز بمعدل ٣٩.٣٥٪، انزيم الجلوتاثيون ريداكثيز بمعدل ٣١.٥٢٪، انزيم الكالتيك بمعدل ٢٩.٦٥٪ وانزيم السوبرأكسيد ديسميوتاز ٢٥.٠٣٪ بمعدل) بالمقارنة بالمجموعة الضابطة السالبة. الا ان التغذية على وجبات تحتوى على ٧.٥٪ من مساحيق قفل الطماطم، قشر البطاطس، اوراق القنبيط، قشر الباذنجان، ومخلوطهم قد ادي الي تحسن في مستويات جميع التقديرات الحيوية السابقة. كما كانت افضل التأثيرات الإيجابية لجميع التقديرات الحيوية السابقة من نصيب مخلوط المساحيق السابقة يليه قفل الطماطم ثم مسحوق قشر الباذنجان ثم مسحوق اوراق القنبيط ، وأخيرا مسحوق قشر البطاطس على التوالي. وأيدت نتائج الدراسة أهمية تعديل/تدعيم الوجبات الغذائية لتشتمل على الأجزاء النباتية الغنية بالمركبات النشطة حيويًا للحد من الاجهاد التأكسدي المرتبط بمرض السمنة.

الكلمات المفتاحية: قفل الطماطم، قشر البطاطس، ورق القنبيط ، قشر الباذنجان، المواد الفعالة لحمض الثيوباربيتوريك، أجزاء الجلوتاثيون، الإنزيمات المضادة للأكسدة، الفيتامينات المضادة للأكسدة.