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Oxidative Stress and antioxidant defense systems status in obese rats feeding some selected food processing by-products applied in bread

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Abstract: Oxidative stress plays a pathological role in the development of various diseases including obesity. Systemic oxidative stress results from an imbalance between oxidants derivatives production and antioxidants defenses. The present study aims to investigate the oxidative stress and antioxidant defense system status in obese rats feeding some selected food processing by-products applied in bread. Forty two (140±10g 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 5 % potato peel powder (PPP), cauliflower leaves powder (CLP), red onion skin powder (ROSP), mango peel powder (MPP) and their mixture, respectively. At the end of the experiment (8 weeks), rats of the obese group recorded 148.70% of the normal group for the BW. Biochemical analysis data indicated that obesity induced a significant increased ($p \leq 0.05$) in plasma oxidants concentration (TBARS, 41.95%; NO_2, 31.02% and NO2/NO3, 26.45%) and significant decreased ($p \leq 0.05$) in plasma non-enzymes antioxidant (GSH, 30.83% and GSSG, 11.27%), plasma antioxidant vitamins (vitamin A, 27.43%; vitamin C, 20.98% and vitamin E, 31.50%) as well as RBC's antioxidant enzymes (GSH-Px, 37.66%; GSH-Rd, 28.66%; CAT, 19.51% and SOD, 25.26%) as a percent of normal

group. Feeding on 5% of PPP, CLP, ROSP, MPP and their mixture induced significant exhibited a significant improvement ($p \leq 0.05$) in all of these parameters by different rates. The higher amelioration effects were recorded for the by-product mixtures treatment followed by ROSP, MPP, PPP and CLP, respectively. In conclusion, the present data support the benefits of dietary modification, including bioactive compounds and antioxidant vitamins supplementation, in alleviating oxidative stress associated obesity.

Keywords: TBARS, NO_2, GSH, antioxidant vitamins, antioxidant enzymes, cauliflower leaves, onion skin, mango peel, potato peel.

Introduction

Oxidative stress was initially defined by **Sies (1985, 1986)** 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. Oxidative stress 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, oxidative stress can cause disruptions in normal mechanisms of cellular signaling **(Evans *et al.*, 2005).**

In humans, oxidative stress 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 contribute 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 modifycation have been observed in humans **(Van Gaal *et al.*, 1998).**

Obesity is defined as an excessive accumulation of body fat mass to the extent that individual's health will be negatively affected. Indeed, obesity is considered as a top risk factor to develop deleterious associated pathologies as liver and coronary heart diseases, osteoarthritis and asthma, and a combination of medical disorders which includes: type 2 diabetes, high blood pressure, high blood cholesterol, and high triglyceride levels **(Jenner, 2003; Grundy, 2004; Dut *et al.*, 2008 and Esra *et al.*, 2012; Le Lay *et al.*, 2014 and Sayed Ahmed, 2016)**.The prevalence of obesity over the past years has been in constant progression leading the World Health Organization (WHO) to consider it as an epidemic pathology and one of the leading preventable causes of death worldwide. For example, obesity Large-scale American and European studies have found that mortality risk is lowest at normal body weight, a body mass index (BMI) of 20–25 kg/m^2 in non-smokers and at 24–27 kg/m^2 in current smokers, with risk increasing along with changes in either direction. Obesity grade I, a BMI above 32 kg/m^2 has been associated with a doubled mortality rate among women over a 16-year period. In the United States obesity is estimated to cause 111,909 to 365,000 deaths per year, while 1 million (7.7%) of deaths in Europe are attributed to excess weight. On average, obesity reduces life expectancy by six to seven years, Obesity grade I, a BMI of 30–35 kg/m^2 reduces life expectancy by two to four years, while severe obesity (BMI > 40 kg/m2) reduces life expectancy by ten years **(Whitlock *et al.*, 2009)**

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

therapeutical strategies used currently to treat obesity-associated metabolic disorders have the potential to decrease oxidative stress, which might, at least partially, participate in their beneficial effect. Unfortunately, data related to this issue is still in dearth. So, one of the aims of the present study is to observe the oxidative stress and antioxidant defense status in obesity, which represents one of the leading preventable causes of death worldwide. Also, the effect of feeding some selected food processing by-products rich in bioactive compounds (acts as antioxidants) applied in bread on that status in obese rats will be in the scope of this investigation.

Material and Methods

Materials

Wheat flour: wheat (*Triticum vulgare*) flour samples (extraction rate, 82%) was purchased from Damietta Milling Company, Damietta, Egypt.

Food processing by-products: Red onion (*Allium cepa* L.) skin (ROS) was obtained from the New Beni Suef company for Preservation, dehydration and Industrialization of Vegetables, Beni Suef Elgudida City, Nile East, Beni Suef, Egypt; potato (*Solanum tuberosum* L.) peel (PP) from SFCO For Manufacturing & Export Agricultural Products, El Negila, Kom Hamada, Behira Government , Egypt. Mango (*Mangifera indica* L. cv Copania) peel (MP) was obtained from Faragalla Company for Food Industries, Borg El-Arab, Alexandria, Egypt. Cauliflower (*Brassica oleracea* L. cv Copania) leaves were obtained by special arrangement with some farmers in Damietta, Damietta Governorate, Egypt. The collected samples were transported immediately in cooling state o the laboratory and used immediately for dehydration process and powders preparation.

Bread manufacturing components: Salt, bicarbonate soda, sugar and yeast were purchased from the local markets of Damietta, Damietta Governorate, Egypt.

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). De-ionized water (Milli-Q 18.2 MΩ) was used in the preparation of the mobile phases, reagent solutions and standards.

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 µm, 150 x 4.6 mm I.d.) for glutathione fractions ; a reversed-phase water Adsorbosil C_{18} (5 µM, 100 mm × 4.6 mm I.d.) for vitamin C; and normal Ultrasphere Si (5 µM, 250 mm × 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 peel powder

Unripe mango peel were soaked in 0.1% sodium metabisulphite solution for 30 min, washed, sliced and dried in two stages at 60 ^{0}C for 12 and 40 ^{0}C for 12 hours in hot air oven (AFOS Mini Smoker, England). ROS and PP were washed and then dried in a hot air oven (Horizontal Forced Air Drier, Proctor and Schwartz Inc., Philadelphia, PA) at 55 ^{0}C for 14. Cauliflower leaves were washed and then dried in a hot air oven (Horizontal Forced Air Drier, Proctor and Schwartz Inc., Philadelphia, PA) at two stages 50 ^{0}C for 6 hrs followed by 40 ^{0}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.

Balady bread processing

The balady bread samples were prepared according to the modified method of **Saba, (1985).** Formulation of the bread is applied as follow: wheat flour, 1000 g; salt, 20g; and dries yeast, 2 g; and water 500 g. Yeast was mixed with water (25 ^{0}C) to form a suspension, to which the other ingredients were then added and kneaded to form smooth dough. Substitution of wheat flour with potato peel powder (PPP), cauliflower leaves powder (CLP), red onion skin powder (ROSP) and mango peel powder (MPP), were conducted based on 5% of the weight of the wheat flour. The dough was later proofed for 2 hours in a

proofer (Bakbar E81, New Zealand), then cut into loafs 120 g prior to baking at 170 ^{0} C for 10 min. Sensory evaluation for color, taste, overall acceptability was carried out with 10 panelists comprising of postgraduate students from Damietta University, Damietta, Egypt. Under such evaluation conditions, all the bread samples, wheat flour and wheat flour blended with the food industrial by-products were accepted for the all panelists. Wheat flour breads have the highest values of sensory evaluation process followed by wheat flour blended with MPP, CLP, ROSP and PPP, respectively.

Biological Experiments

Materials

Casein was obtained from Morgan Chemical Co., Cairo, Egypt. The rest of chemicals, reagents and solvents were of analytical grade and purchased from El-Ghomhorya for Drugs, Chemicals and Medical Instruments Trading Co. (Cairo, Egypt).

Animals

Animals used in this study, adult male albino rats (140± 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 H_2O (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), 160-170g per each, were housed individually in wire cages in a room maintained at 25 ± 2 ^{0}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 5 % PPP; group (4), fed on DIO containing 5 % CLP; group (5), fed on DIO containing 5 % ROSP, group (6), fed on DIO containing 5 % MPP and group (7): fed on DIO containing 5 % mixture, PPP + CLP+ ROSP + MPP by equal parts. Body weight gain (as percent of initial weight) was assayed every week in rats.

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) – $KHCO_3$ (2.4 M) and allowed to incubate in the dark at room temperature for 1 hr to obtain S-carbooxymethyl derivatives. The N-nitrophenol derivatization of the samples were taken overnight at 4 ^{0}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 ($INTH_2$) reduction rate.

Antioxidant vitamins

All vitamins (A, C, and E) were extracted and analyzed by HPLC techniques as follow: Vitamin A was extracted by adaptation the method of **Epler *et al*., (1993).** A 0.3 ml of serum were saponified by 0.1 ml of sodium hydroxide solution (60%) and 1- 2 ml ethyl alcohol; heated on a water bath at 85 - 90 ^{0}C under reflux for two hrs until the serum components were completely dissolved; 1 - 2 ml of ethyl alcohol and about 2 to 4 ml of distilled water were added. The unsuponified portion was extracted three times by ether using 5 ml in the first and second extraction and 2.5 ml in the third one. The ether extraction was washed 3 to 4 times with water until the washed water became neutral, 0.6 – 0.8 grams sodium sulphate were added and the mixture was left for 90 min and filtrated. After removing the ether from the solution, the residual matter was dissolved in one ml chloroform and diluted to 2.5 ml with the

same solvent, 0.1 ml of the chloroform extract was transferred to a small screw-captest tube. The chloroform solution was dried under a stream of nitrogen, re-dissolved in exactly 0.1 or 0.2 ml isopropanol -hexane (1: 99, v/v), and used for HPLC injection.

Vitamin E (α-tocopherol) was extracted by adaptation the method of **Hung *et al*., (1980).** Approximately 50 µl of serum were homogenized in 1.5 ml dioxane-isooctane (20 + 80, v/v) for 1 min, using a Polytron homogenizer (Beckman, Toronto). The homogenate was centrifuged at 10000 rpm for 5 min, and 0.5 ml supernate was placed in a 25 ml round-bottom flask. Remainder of the supernate was discarded. The residue was homogenized with another 1.5 ml dioxane-isooctane solution, centrifuged as before, and 0.5 ml supernate was pooled with the previous supernate and dried under vacuum in a rotary evaporator to near dryness. The residue was extracted 3 times with 0.5 ml acetonitrile and the pooled acetonitrile extracts were filtered through glass wool into a 5 ml screw-cap test tube. The filtrate then was extracted with 1.5, 1, and 0.5 ml isooctane. The isooctane extracts were pooled in a 25 ml round-bottom flask and dried under vacuum in a rotary evaporator. The residue was dissolved in 0.2 - 0.4 ml petroleum ether (bp 30 - 60 ^{0}C) and transferred to a small screw-captest tube. The petroleum ether solution was dried under a stream of nitrogen, re-dissolved in exactly 0.1 or 0.2 ml isopropanol -hexane (1: 99, v/v), and used for HPLC injection.

Vitamin C (ascorbic acid) was extracted according to the method of **Moeslinger *et al*., (1994).** One hundred µl of plasma were deproteinized by 400 µl ice-cold 8% perchloric acid which was described as stabilizing ascorbic acid in biological samples, centrifuged at 10000 g for 10 min at 4 ^{0}C, and neutralized by the addition of 4 M potassium hydroxide. The residues was dried under a stream of nitrogen, re-dissolved in exactly 0.1 or 0.2 ml methanol and used for HPLC injection.

The chromatographic conditions for vitamins A and E were flow rate, 1.5 ml/min; detection, UV absorption at 265 nm, volume of injection, 20 µl; temperature, room temperature; and the mobile phase composition was an isocratic system of isopropanol : hexane (1:99) while in vitamin C were flow rate, 1 ml/min; detection, UV absorption at 254 nm, volume of injection, 20 µl; temperature, room temperature,

and mobile phase composition was an isocratic system of 100 % methanol. Retention times and absorbance ratio against those of standards were used to identify the separated vitamins. 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 ±SD values of vitamins A, C and E recoveries were 90.1 ± 5.3, 88.9 ± 4.4, and 84.7 ± 5.2, respectively.

Nitrite determination

Nitrite was determined flourometric such as described by **Misko *et al*., (1993)**. Ten µl of freshly prepared 2, 3-diaminonaphthalene (DAN, 0.05 mg/ml in 0.62 M HCl, protected from light) is added to100 µl of sample and mixed immediately. Nitrate standards (> 98% pure, Sigma) are routinely made fresh, dissolved in DI H_2O, and kept on ice prior to use. After 10 min incubation at 20 ^{0}C, the reaction was terminated with 5 µl 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 µM NADPH (to initiate the reaction) and 14 mU of enzyme in a final volume of 50 µl of 20 mM Tris buffer (pH, 7.6). The reaction is terminated after 5 min at 20 ^{0}C by dilution with 50 µl 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.

Thiobarpituric 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±SD. Statistical analysis was performed with the Student *t*-test and MINITAB-12 computer program (Minitab Inc., State College, PA).

Results and Discussion

The effect of food processing by-products applied in bread on body weight of obese rats

The effect of food processing by-products applied in bread on body weight (BW, g) 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 148.70% of the control (normal) group for the BW. Replacement of wheat flour with potato peel powder (PPP), cauliflower leaves powder (CLP), red onion skin powder (ROSP), mango peel powder (MPP) and their mixture induced significant

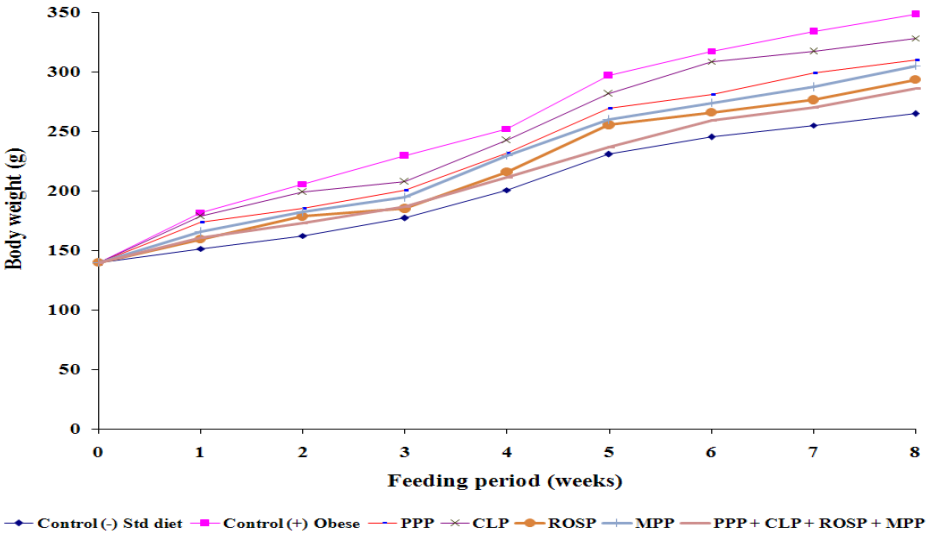

Figure 1. The effect of food processing by-products applied in bread on body weight (g) of obese rats*

* PPP, potato peel powder, CLP; cauliflower leaves powder; ROSP, red onion skin powder; MPP, mango peel powder and Mixture, PPP + CLP+ ROSP + MPP by equal parts.

decreasing on BW of the obese rats which recorded 121.71, 134.60, 109.66 , 118.00 and 104.55% as a percent of control, respectively. The higher effect on weigh decreasing was recorded for the by-product mixtures followed by ROSP, MPP, PPP and CLP, respectively. Such date are in agreement with that observed by **Bedawy, (2008) and Le Lay *et al.*, (2014).** The positive effects of the selected plant parts i.e.ROSP, MPP, PPP and CLP and their mixture regarding the control of the obesity could be attributed to their high level content of different classes bioactive compounds called phytochemicals **(Le Lay et *al.*, 2014).** Our previous studies with the others indicated that such selected plant parts were rich in several classes of phytochemicals including flavonols, phenolic acids, anthocyanins, alkaloids, carotenoids, phytosterols and organosulfur compounds **(Beattic *et al.*, 2005, Mashal, 2016 and Elhassaneen *et al.*, 2016 a-b)**. 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 oxidative stress responses; and scavenged of reactive species such ROD and RNS **(Constance *et al.*, 2003; Bonet *et al.*, 2015 and Le Lay *et al.*, 2014) .**

Effect of food processing by-products applied in bread on oxidants concentration of obese rats

Oxidative stress status in obese rats feeding some selected food processing by-products applied in bread was assessed by measuring some oxidants concentration in plasma including thiobarbituric acid reactive substances (TBARS) and nitric oxides (nitrite, NO_2 and nitrate, NO_3) (Table 1 and Figure 2). From such data it could be noticed that obesity induced a significant increased ($p \leq 0.05$) in TBARS, NO_2 and NO2/NO3concentration in plasma by 41.95, 31.02 and 26.45% compared to normal controls, respectively. Supplementation of the rat diets with 5% w/w by PPP, CLP, ROSP, MPP and their mixture induced significant decreasing on these parameters concentration in plasma by the ratio of 19.10, 19.59 and 18.64; 24.72, 25.71 and 22.67; 7.87, 10.61 and 14.36;

Table 1. Plasma oxidants concentration in obese rats feeding some selected food processing by-products applied in bread*

Value	Control (-)	Control (+)	Food processing by-products (5%, w/w)				
			PPP	CLP	ROSP	MPP	Mixture
Thiobarbituric acid reactive substances (TBARS, nmol/mL)							
Mean	2.67 [b]	3.79[a]	3.18 [a]	3.33 [a]	2.88 [b]	3.08 [ab]	2.79 [b]
SD	0.37	0.65	0.98	1.12	0,66	1.05	0.52
% of Change	0.00	41.95	19.10	24.72	7.87	15.36	4.49
Nitrite (NO_2, nmol/L)							
Mean	2.45 [b]	3.21 [a]	2.93 [a]	3.08 [a]	2.71[b]	2.86 [ab]	2.63[b]
SD	0.65	1.17	0.62	0.45	0.79	0.32	0.18
% of Change	0.00	31.02	19.59	25.71	10.61	16.73	7.35
Nitrite/Nitrate (NO_2/NO_3, nmol/L)							
Mean	3.97 [b]	5.02 [a]	4.71 [a]	4.87 [a]	4.54	4.68 [a]	4.37 [ab]
SD	0.45	0.98	1.34	0.80	0.53	0.43	1.52
% of Change	0.00	26.45	18.64	22.67	14.36	17.88	10.08

*PPP, potato peel powder, CLP; cauliflower leaves powder; ROSP, red onion skin powder; MPP, mango peel powder and Mixture, PPP + CLP+ ROSP + MPP by equal parts. Means in the same row with different litters are significantly different at $p < 0.05$.

15.36, 16.73 and 17.88; and 4.49, 7.35 and 10.08%, respectively. The higher amelioration effect in plasma TBARS, NO_2 and NO2/NO3concentration rising induced by obesity in rats was recorded for the by-product mixtures treatment followed by ROSP, MPP, PPP and CLP, respectively.

In similar studies, clinical evidences for obesity-associated oxidative stress have been provided by measurement of either biomarkers or end-products of free radical-mediated oxidative processes **(Elhassaneen and Salem, 2014 and Sayed Ahmed, 2016).** 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).** Systemic metabolic alterations associated with obesity contribute to the increase in oxidative stress have been reported by many authors. For example,

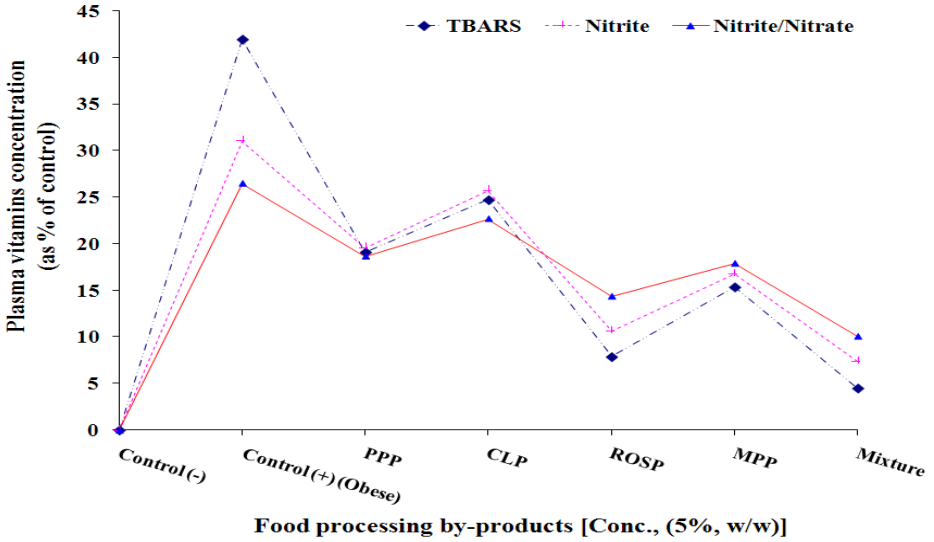

Figure 2. Plasma oxidants concentration in obese rats feeding some selected food processing by-products applied in bread*

*PPP, potato peel powder, CLP; cauliflower leaves powder; ROSP, red onion skin powder; MPP, mango peel powder and Mixture, PPP + CLP+ ROSP + MPP by equal parts.

hyperglycemia as a hallmark of type II diabetes, a metabolic complication of obesity, induces oxidative stress 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).** 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).** Altogether, dysregulation of metabolic parameters occurring with fat mass expansion will contribute to inducing oxidative-stress damages notably at the vascular level **(Brandes and Kreuzer, 2005).**

Regarding the RNS, Endothelial NO synthase- (eNOS-) and inducible NO synthase- (iNOS-) dependent NO are abundant in adipocytes. iNOS expressionhas 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).**

Several decades ago, interest in the possible significance of MDA on human health has been stimulated by reports that are mutagenic and carcinogenic compound **(Shamberger *et al.*, 1974)**. Nitric oxide synthase catalyzes the conversion of L-arginine to citrulline and highly reactive free radical species, nitric oxide (NO) **(Manahan, 1989).** Nitric oxide, 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, **Coskun *et al.*, (2005)** found that quercetin, domainant flavonoid such as found in our selected plant parts, have anti-oxidative and anti-inflammatory activities. Such dietary phenolics found in tested plant by-products are metabolized in liver, inhibiting liver injury induced by diabetes i.e. enhancing lipid metabolism, reducing oxidative stress may be particularly effective, consequently. Additionally, the mixture treatment gave maximum reduction yield of plasma MDA when compared with the tested plant by-products plant separated. It could be mean that a combination of different plant by-products may be more efficient for reducing plasma MDA level, the biomarkers of oxidative stress and inflammation in the body, because the interactive effects occurred by different categories of bioactive compounds of different plant parts by-products used.

Effect of food processing by-products applied in bread on glutathione fractions concentration in plasma of obese rats

Biological antioxidant macromolecules i.e. glutathione fractions concentration in plasma of obese rats consumed food processing by-products applied in bread were assessed (Table 2 and Figure 3). From such data it could be noticed that obesity induced a significant decreased ($p \leq 0.05$) in GSH and GSSG concentrations and GSH/GSSG ratio in plasma by 30.83, 11.27 and 22.04% compared to normal controls, respectively. Supplementation of the rat diets with 5% w/w by PPP, CLP, ROSP, MPP and their mixture induced significant increasing on these parameters concentration in plasma by the ratio of 16.99, 9.86 and 7.91; 19.42, 9.86 and 10.60; 12.86, 7.04 and 6.26; 14.81, 8.45 and 6.94; and 11.17, 7.04 and 4.44, respectively. The higher amelioration effect in plasma GSH and GSSG concentrations and GSH/GSSG ratio rising induced by obesity in rats was recorded for the by-product mixtures treatment followed by ROSP, MPP, PPP and CLP, respectively.

Table 2. Plasma glutathione fractions concentration in obese rats feeding some selected food processing by-products applied in bread*

Value	Control (-)	Control (+)	Food processing by-products (5%, w/w)				
			PPP	CLP	ROSP	MPP	Mixture
Reduced glutathione concentration (GSH, µmol /L)							
Mean	8.24 [a]	5.70 [bc]	6.84 [ab]	6.64 [ab]	7.18 [a]	7.02 [a]	7.32 [a]
SD	1.67	0.86	0.98	0.61	1.05	0.33	0.64
% of Change	0.00	-30.83	-16.99	-19.42	-12.86	-14.81	-11.17
Oxidized glutathione concentration (GSSG, µmol /L)							
Mean	0.71 [a]	0.63 [b]	0.64 [ab]	0.64 [ab]	0.66 [ab]	0.65 [a]b	0.66 [ab]
SD	0.08	0.17	0.10	0.21	0.04	0.11	0.07
% of Change	0.00	-11.27	-9.86	-9.86	-7.04	-8.45	-7.04
GSH/GSSG ratio							
Mean	11.61 [a]	9.05 [b]	10.69 [a]	10.38	10.88 [a]	10.80 [a]	11.09 [a]
SD	1.76	0.99	2.00	1.76	2.33	1.34	0.55
% of Change	0.00	-22.04	-7.91	-10.60	-6.26	-6.94	-4.44

*PPP, potato peel powder, CLP; cauliflower leaves powder; ROSP, red onion skin powder; MPP, mango peel powder and Mixture, PPP + CLP+ ROSP + MPP by equal parts. Means in the same row with different litters are significantly different at $p < 0.05$.

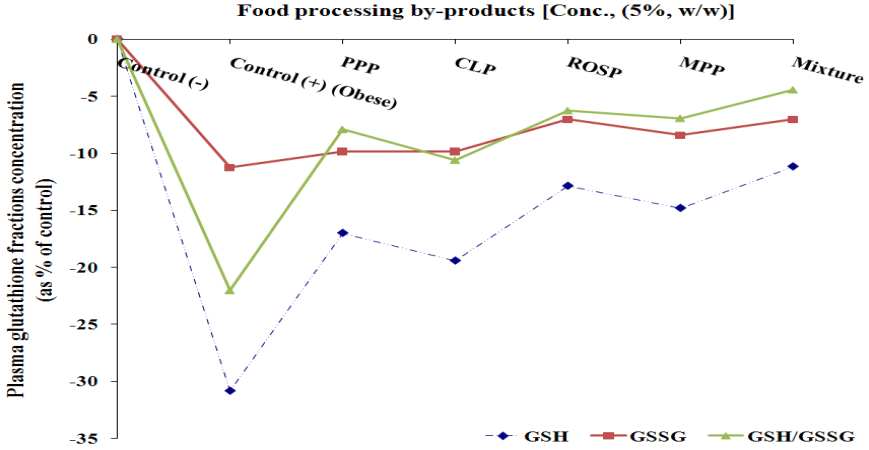

Figure 3. Plasma glutathione fractions concentration in obese rats feeding some selected food processing by-products applied in bread*

*PPP, potato peel powder, CLP; cauliflower leaves powder; ROSP, red onion skin powder; MPP, mango peel powder and Mixture, PPP + CLP+ ROSP + MPP by equal parts.

Reduced glutathion (GSH) is a tripeptide-thiol (γ-glutamyl cysteinyl-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 these function are two constructing roles in detoxifications: (1) as a key conjugate of electrophilic intermediates, 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 fall in glutathione fractions observed in obese rats group generally accompanied by a concomitant decreased in the ratio of GSH/GSSG. **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. Few studies have been addressed directly the issue of effects of pro-oxidants on redox status. **Elhassaneen *et al.*, (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 this context, **Bedard and Krause (2007)** reported that various enzymes inside the cells including adipocytes can also produce ROS. Particularly, the family of NAPDH 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. 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 food processing by-products applied in bread on antioxidant enzymes activities in RBCs of obese rats

Antioxidant defense system in RBCs in obese rats feeding some selected food processing by-products applied in bread was assessed by measuring antioxidant enzymes activities including glutathione peroxidase (GSH-Px), glutathione reductase (GSH-Rd), catalase (CAT) and superoxide dismutase (SOD) (Table 3 and Figure 4). From such data it could be noticed that obesity induced a significant increased ($p \leq 0.05$) in GSH-Px, GSH-Rd, CAT and SOD activities in RBC's in by 37.66, 28.66, 19.51 and 25.26% compared to normal controls, respectively. Supplementation of the rat diets with 5% w/w by PPP, CLP, ROSP, MPP and their mixture induced significant decreasing on these parameters concentration in plasma by the ratio of 23.27, 20.17, 12.95 and 20.10; 27.36, 24.77, 17.04 and 23.45; 17.49, 15.19, 8.63 and 13.66; 22.43, 18.15,

Table 3. Erythrocytes antioxidant enzymes activities in obese rats feeding some selected food processing by-products applied in bread*

Value	Control (-)	Control (+)	Food processing by-products (5%, w/w)				
			PPP	CLP	ROSP	MPP	Mixture
Glutathione peroxidase (GSH-Px, U/g Hb)							
Mean	19.04 a	11.87 c	14.61 b	13.83 bc	15.71 ab	14.77 ab	16.07 a
SD	3.76	2.32	1.79	2.19	2.55	0.97	3.44
% of Change	0.00	-37.66	-23.27	-27.36	-17.49	-22.43	-15.60
Glutathione reductase (GSH-Rd, U/g Hb)							
Mean	12.84 a	9.16 bc	10.25 ab	9.66 ab	10.89 ab	10.51 ab	11.21 a
SD	2.06	1.11	2.62	1.80	2.00	1.34	2.77
% of Change	0.00	-28.66	-20.17	-24.77	-15.19	-18.15	-12.69
Catalase (CAT, U/g Hb)							
Mean	170.65 a	137.35 bc	148.55 ab	141.57	155.92 a	150.82 ab	158.42 a
SD	11.67	8.94	17.32	10.43	5.22	6.29	4.87
% of Change	0.00	-19.51	-12.95	-17.04	-8.63	-11.62	-7.17
Superoxide dismutase (SOD, U/g Hb)							
Mean	3.88 a	2.90 ab	3.1 ab	2.97 ab	3.35 a	3.14 ab	3.41 a
SD	0.74	0.52	0.460	0.51	1.08	0.17	0.49
% of Change	0.00	-25.26	-20.10	-23.45	-13.66	-19.07	-12.11

*PPP, potato peel powder, CLP; cauliflower leaves powder; ROSP, red onion skin powder; MPP, mango peel powder and Mixture, PPP + CLP+ ROSP + MPP by equal parts. Means in the same row with different litters are significantly different at $p < 0.05$.

11.62, 19.07; and 15.60, 12.69, 7.17 and 12.11% , respectively. The higher amelioration effect in plasma GSH-Px, GSH-Rd, CAT and SOD activities in RBC's rising induced by obesity in rats was recorded for the by-product mixtures treatment followed by ROSP, MPP, PPP and CLP, respectively.

To prevent free radical damages (oxidative stress activities), the organism has developed antioxidant defenses largely based on antioxidant enzymes able to scavenge ROS. SODs are responsible for the reduction of $O_2^{\cdot-}$ to H_2O_2 and multiple enzymes will remove H_2O_2 including GSH-Px and CAT. Also, the GSH reduces the selenium and the reduced form of the enzyme then react with hydrogen peroxide. The ratio of nine *GSH lGSSG* in normal cells are kept high. So there must be a mechanism

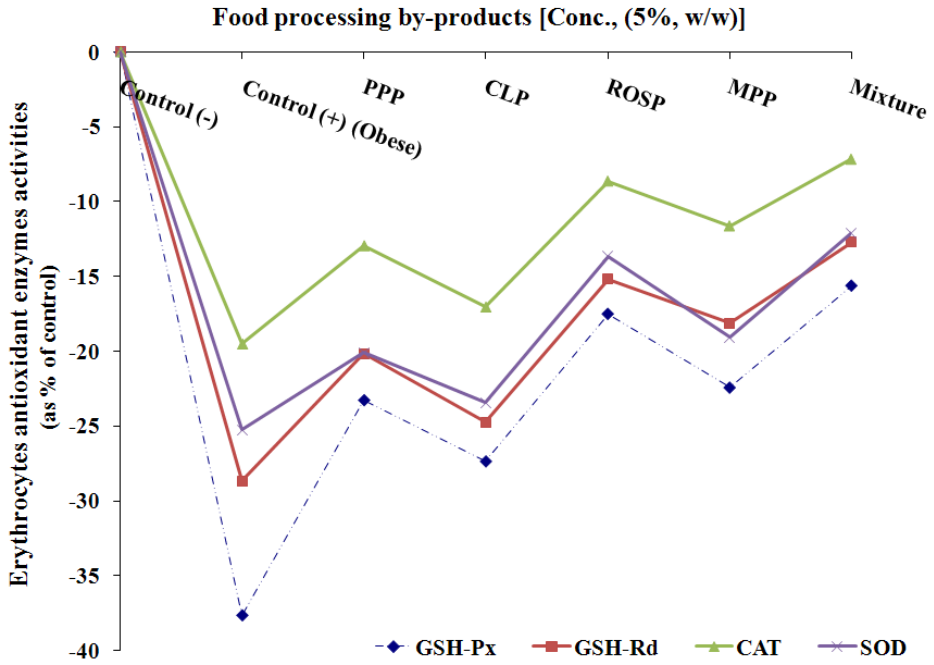

Figure 4. Erythrocytes antioxidant enzymes activities in obese rats feeding some selected food processing by-products applied in bread*

*PPP, potato peel powder, CLP; cauliflower leaves powder; ROSP, red onion skin powder; MPP, mango peel powder and Mixture, PPP + CLP+ ROSP + MPP by equal parts.

of reducing GSSG back to GSH. This is achieved by GSH-Rd enzyme which catalyze the reaction:GSSG + NADPH + H^+ → 2GSH + $NADP^+$. GSH-Rd can also catalyse reduction of certain mixed disulphides such as that between GSH and Co-enzyme A **(Thomas *et al.*, 1990).** Mammalian erythrocytes operate the pentose phosphate pathway in order to provide NADPH for glutathione reduction **(Harmon, 1986).** 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 **(Galinier *et al.*, 2006).** Silencing of the antioxidant enzyme glutathione-*S*-transferase (GSTA4) in cultured adipocytes or its invalidation in mice both results in increased ROS production and mitochondrial dysfunction **(Curtis *et al.*, 2010).** 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 **(Elhassaneen *et al.*, 2016 and and Mashal, 2016).** Such antioxidant properties are important in manipulation of the obesity development through ROS scavenging processes in fat cells.

Effect of food processing by-products applied in bread on antioxidant vitamins concentration in plasma of obese rats

Antioxidant vitamins concentration in plasma of obese rats consumed food processing by-products applied in bread were shown in Table (4) and Figures (5). From such data it could be noticed that obesity induced a significant decreased ($p \leq 0.05$) in vitamins A, C and E in plasma by 27.43, 20.98 and 31.50% compared to normal controls, respectively. Supplementation of the rat diets with 5% w/w by PPP, CLP, ROSP, MPP and their mixture induced significant increasing on

these parameters concentration in plasma by the ratio of 19.47, 11.37 and 19.93; 23.01, 15.57 and 24.58; 14.16, 7.62 and 14.29; 17.70, 10.17 and 19.14; and 9.73, 7.42 and 11.21%, respectively. The higher amelioration effect in plasma vitamins A, C and E concentration rising induced by obesity in rats was recorded for the by-product mixtures treatment followed by ROSP, MPP, PPP and CLP, respectively.

The reducing in antioxidant enzymes defense potential of erythrocytes was contrary with significant decreasing ($p>0.05$) in antioxidant vitamins in rats plasma as a consequence of obesity injury. Beside the erythrocytes, adipose tissue represents also a preferential storage site for natural antioxidants compounds, as liposoluble vitamins (e.g., vitamins A and E) or β-carotenoids **(Landrier *et al.*, 2012).**

Table 4. Plasma antioxidants vitamins concentration in obese rats feeding some selected food processing by-products applied in bread*

Value	Control (-)	Control (+)	Food processing by-products (5%, w/w)				
			PPP	CLP	ROSP	MPP	Mixture
Vitamin A (Retinol, μmol/L)							
Mean	1.13 [a]	0.82 [b]	0.91 [ab]	0.87 [ab]	0.97 [a]	0.93 [a]	1.02 [a]
SD	0.23	0.08	0.04	0.11	0.20	0.08	0.16
% of Change	0.00	-27.43	-19.47	-23.01	-14.16	-17.70	-9.73
Vitamin C (Ascorbic acid, μmol/L)							
Mean	55.67 [a]	43.99 [b]	49.34 [a]	47.00 [ab]	51.43 [a]	50.01 [a]	51.54 [a]
SD	4.65	3.98	1.59	6.05	7.25	11.05	7.46
% of Change	0.00	-20.98	-11.37	-15.57	-7.62	-10.17	-7.42
Vitamin E (Tocopherol, μmol/L)							
Mean	30.51 [a]	20.90 [b]	24.43	23.01 [ab]	26.15 [a]	24.67 [ab]	27.09 [a]
SD	2.76	4.52	6.76	1.65	3.54	1.63	2.65
% of Change	0.00	-31.50	-19.93	-24.58	-14.29	-19.14	-11.21

*PPP, potato peel powder, CLP; cauliflower leaves powder; ROSP, red onion skin powder; MPP, mango peel powder and Mixture, PPP + CLP+ ROSP + MPP by equal parts. Means in the same row with different litters are significantly different at $p < 0.05$.

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).** Our previous studies indicated that food processing by-products selected in the present study and their mixtures feeding are rich in bioactive compounds including caroteoids and vitamins which exhibited antioxidant activities in different biological systems **(Elhassaneen *et al.*, 2016-a,b,c).** Such antioxidant properties are important in manipulation of the obesity development through ROS scavenging processes subsequently excess the bioavailability of the vitamins in fat cells.

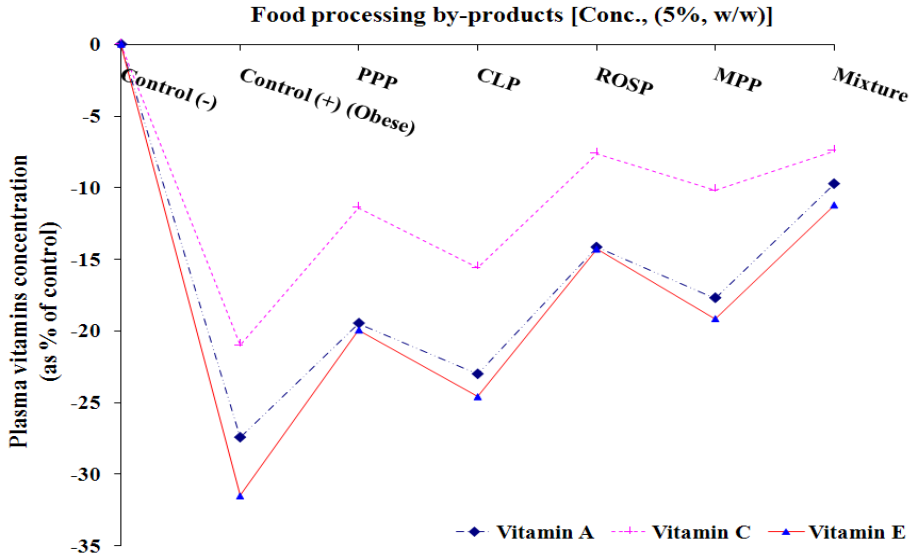

Figure 5. Plasma antioxidants vitamins concentration in obese rats feeding some selected food processing by-products applied in bread*

*PPP, potato peel powder, CLP; cauliflower leaves powder; ROSP, red onion skin powder; MPP, mango peel powder and Mixture, PPP + CLP+ ROSP + MPP by equal parts.

Correlation studies

In the correlation analysis, important differences were found between oxidative and antioxidant defense system in obese rats feeding some selected food processing by-products (PPP, CLP, ROSP and MPP) applied in bread (Table 5 and Figures 6-11). 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 = -0.937), antioxidant vitamins in plasma [vitamin A (r^2 = -0.779) and vitamin E (r^2 = -0.779)], antioxidant

Table 5. Correlation between oxidant stress and antioxidant defense system in obese rats feeding some selected food processing by-products (PPP, CLP, ROSP and MPP) applied in bread*

Parameters	Correlation	R^{2*}
TBARS/GSH	GSH (µmol /L) = - 2.203 (TBARS, nmol/mL) + 13.85	0.937
TBARS/ Vit A	Vit A (mmol/L) = -0.202 (TBARS, nmol/mL) + 1.592	0.779
TBARS/Vit C	Vit C (µmol /L) = -5.900 (TBARS, nmol/mL) + 68.68	0.618
TBARS/Vit E	Vit E (µmol /L) = -6.447 (TBARS, nmol/mL) + 45.43	0.870
TBARS/GSH-Px	GSH-Px (U/g Hb) = -5.481 (TBARS, nmol/mL) + 32.55	0.899
TBARS/CAT	CAT (U/g Hb) = -17.90 (TBARS, nmol/mL) + 207.7	0.838
TBARS/SOD	SOD (U/g Hb) = -0.94 (TBARS, nmol/mL) + 6.408	0.815
NO2/GSH	GSH (µmol /L) = - 2.262 (NO2, nmol/mL) + 13.11	0.863
NO2/ Vit A	Vit A (mmol/L) = -0.629 (NO2, nmol/mL) + 2.789	0.803
NO2/Vit C	Vit C (µmol /L) = -8.244 (NO2, nmol/mL) + 72.40	0.608
NO2/Vit E	Vit E (µmol /L) = -4.933 (NO2, nmol/mL) + 39.51	0.854
NO2/GSH-Px	GSH-Px (U/g Hb) = -6.009 (NO2, nmol/mL) + 32.33	0.879
NO2/CAT	CAT (U/g Hb) = -22.67 (NO2, nmol/mL) + 212.9	0.849
NO2/SOD	SOD (U/g Hb) = -1.203 (NO2, nmol/mL) + 6.753	0.859

** $P \leq 0.01$*

enzymes in RBC's [GSH-Px (r^2 = -0.899), CAT (r^2 = -0.838) and SOD (r^2 = -0.815)] 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 = -0.618) 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 = -0.863), antioxidant vitamins in plasma [vitamin A (r^2 = -0.803) and vitamin E (r^2 = -0. 0.854)], antioxidant enzymes in RBC's [GSH-Px (r^2 = -0. 0.879), CAT (r^2 = -0.849) and SOD (r^2 = -0.859)] 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 = -0.608) and NO_2 concentration in plasma. These

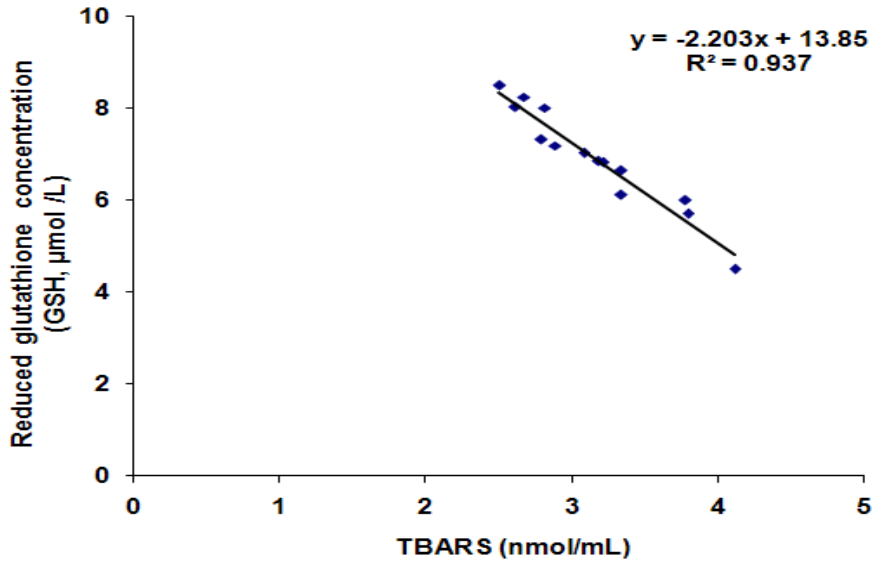

Figure 6. Correlation between TBARS content detected and glutathione in obese rats feeding some selected food processing by-products (PPP, CLP, ROSP and MPP) applied in bread*

*PPP, potato peel powder, CLP; cauliflower leaves powder; ROSP, red onion skin powder; MPP, mango peel powder.

correlations confirm that if there were no change in the antioxidant defense system 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).**

In conclusion, obesity is nowadays considered as a top risk factor in the development of several diseases and is causative of morbidity of patients suffering from metabolic syndrome. Oxidative stress appears as a major contributor in the development of many metabolic complications associated obesity. Lowering oxidative stress to prevent such metabolic disorders and complications therefore constitutes an interesting target.

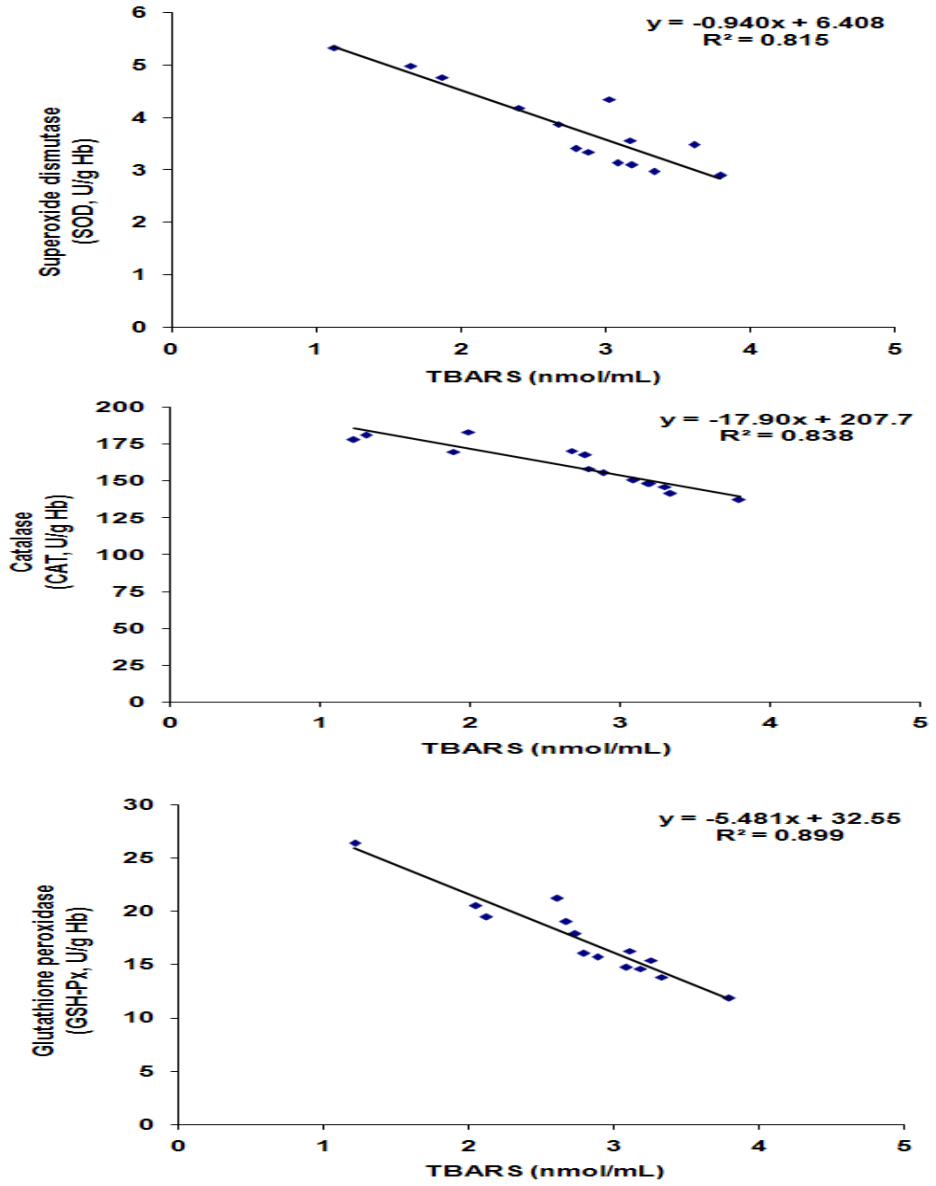

Figure 7. Correlation between TBARS content detected and antioxidant enzymes in obese rats feeding some selected food processing by-products (PPP, CLP, ROSP and MPP) applied in bread*

*PPP, potato peel powder, CLP; cauliflower leaves powder; ROSP, red onion skin powder; MPP, mango peel powder.

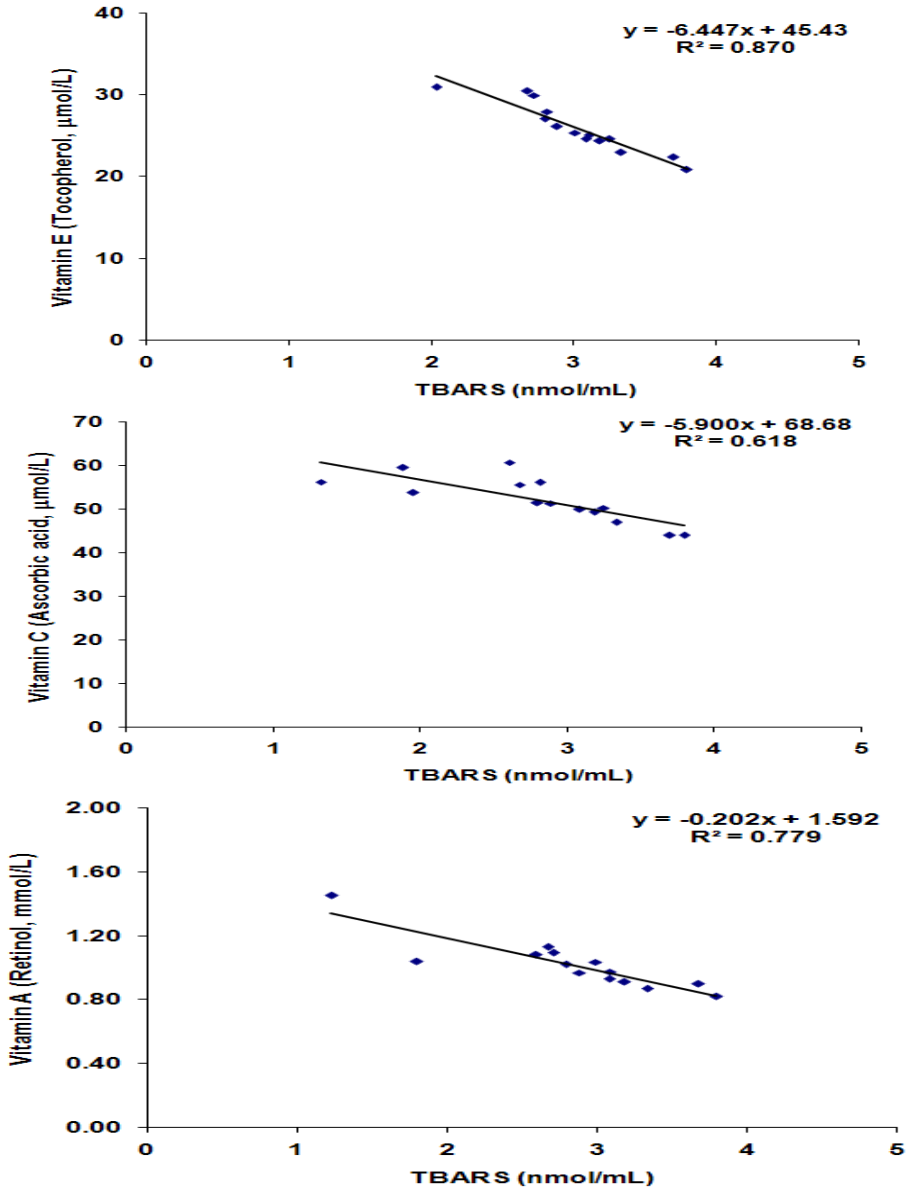

Figure 8. Correlation between TBARS content detected and antioxidant vitamins in obese rats feeding some selected food processing by-products (PPP, CLP, ROSP and MPP) applied in bread*

*PPP, potato peel powder, CLP; cauliflower leaves powder; ROSP, red onion skin powder; MPP, mango peel powder.

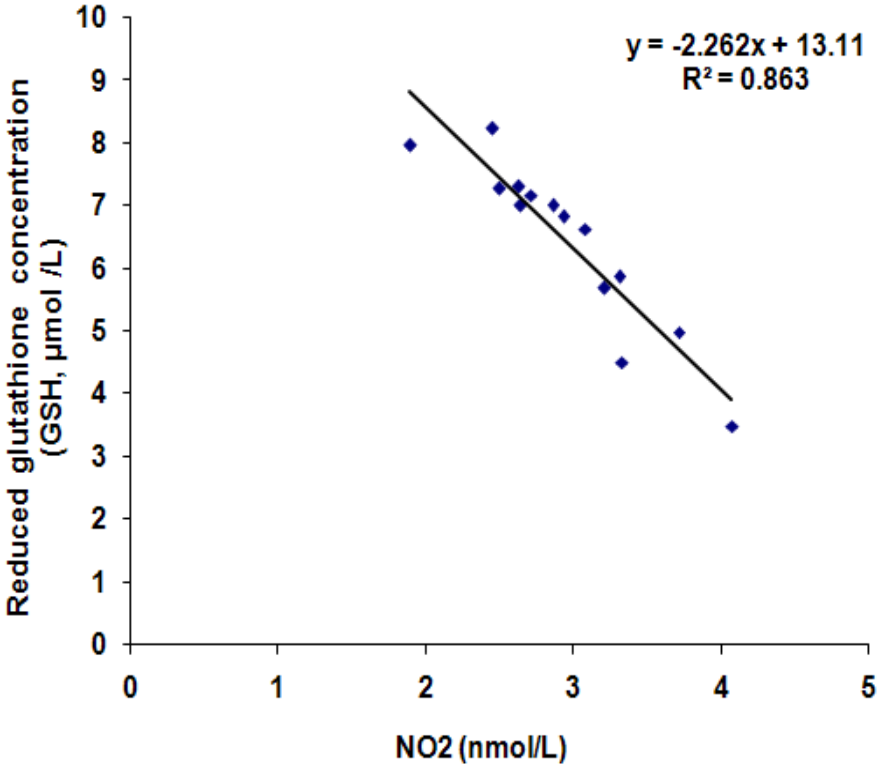

Figure 9. Correlation between NO_2 content detected and glutathione in obese rats feeding some selected food processing by-products (PPP, CLP, ROSP and MPP) applied in bread*

*PPP, potato peel powder, CLP; cauliflower leaves powder; ROSP, red onion skin powder; MPP, mango peel powder.

Feeding of some selected food processing by-products applied in foods has been proven to be essential in the treatment and/or prevention of obesity but also beneficial for oxidative stress reduction. Overall, the present study support the benefits of dietary modification, including bioactive compounds (e.g. phytochemicals) and antioxidant vitamins (e.g. A, E and C) supplementation, in alleviating oxidative stress associated obesity.

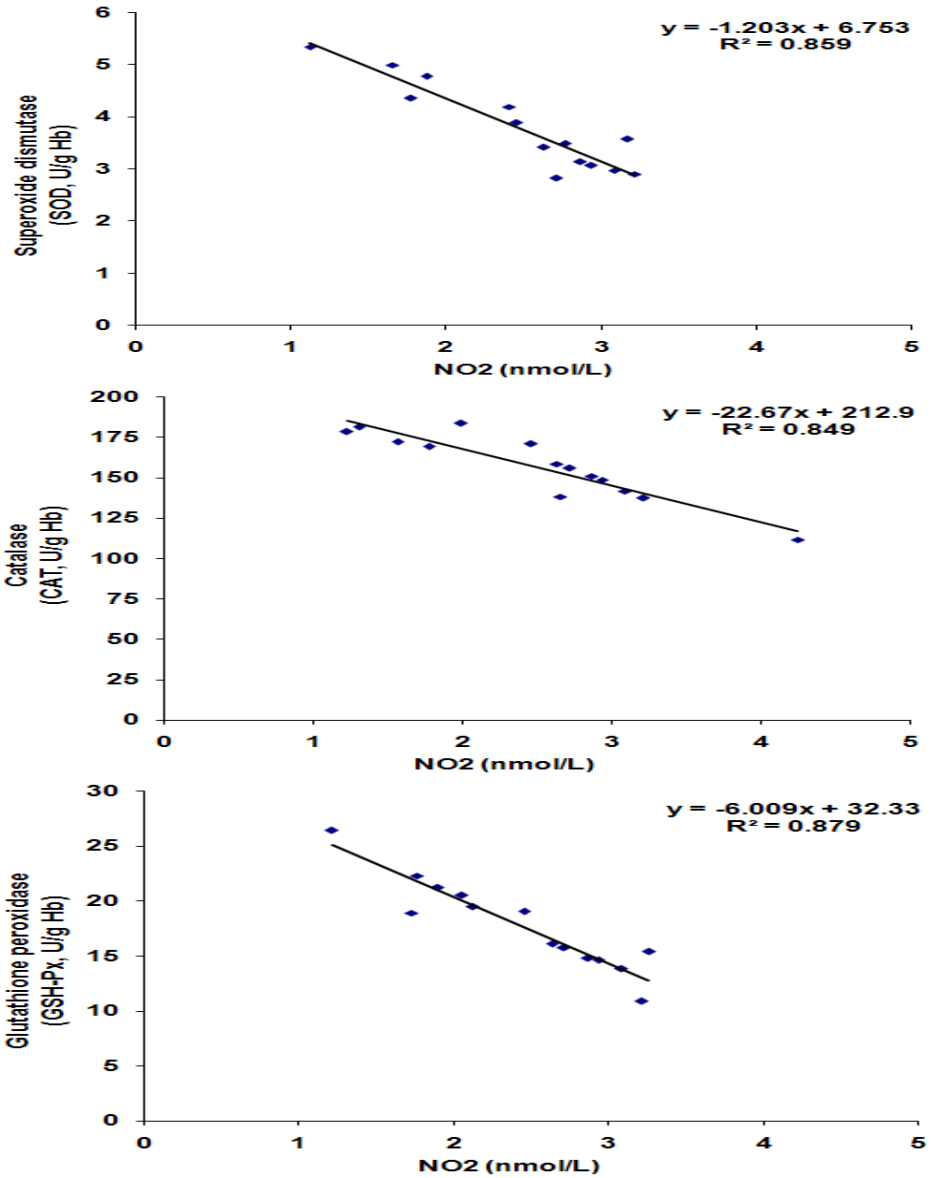

Figure 10. Correlation between NO_2 content detected and antioxidant enzymes in obese rats feeding some selected food processing by-products (PPP, CLP, ROSP and MPP) applied in bread*

*PPP, potato peel powder, CLP; cauliflower leaves powder; ROSP, red onion skin powder; MPP, mango peel powder.

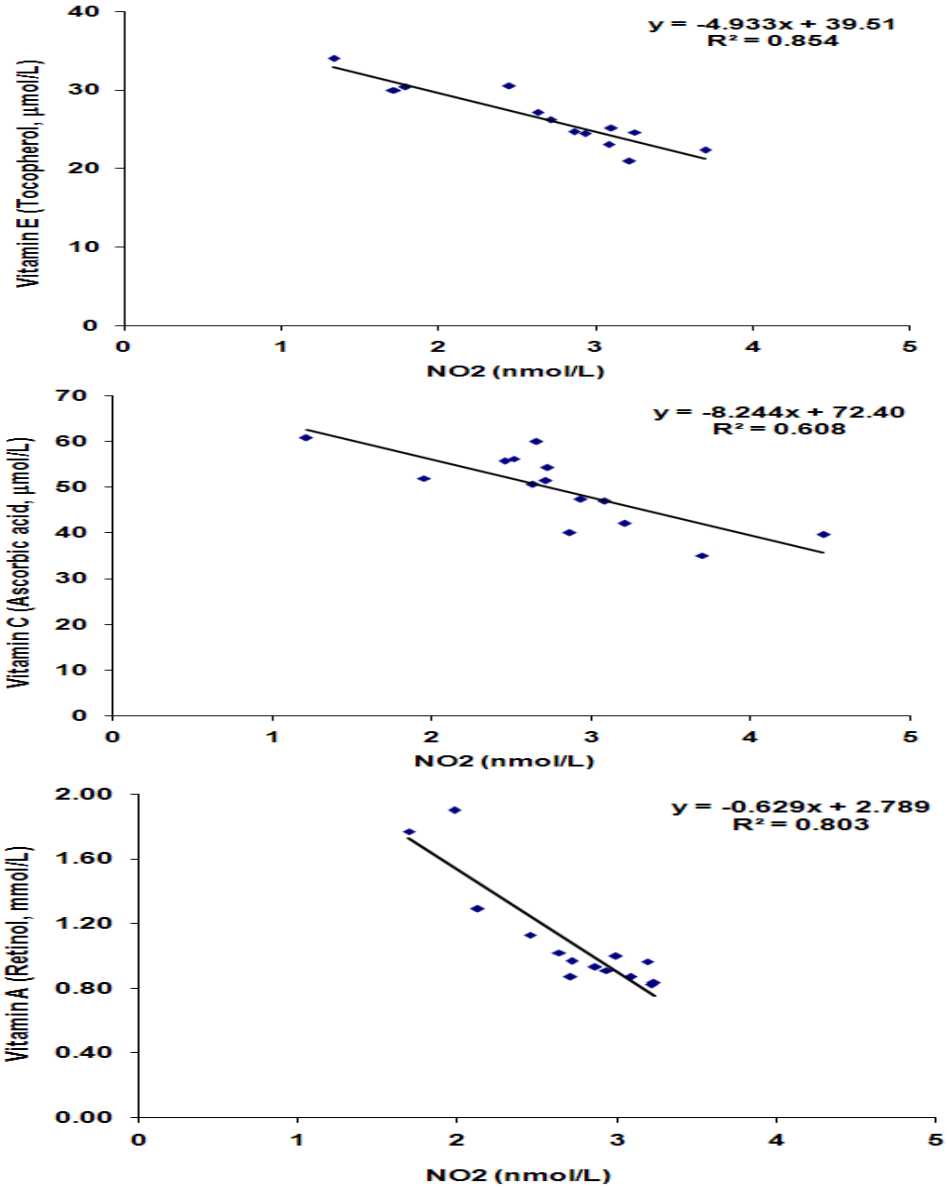

Figure 11. Correlation between NO2 content detected and antioxidant vitamins in obese rats feeding some selected food processing by-products (PPP, CLP, ROSP and MPP) applied in bread*

*PPP, potato peel powder, CLP; cauliflower leaves powder; ROSP, red onion skin powder; MPP, mango peel powder.

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حالة جهد التأكسد ونظم الدفاع المضاد للأكسدة فى الفئران المصابة بالسمنة التى تم تغذيتها على بعض النواتج الثانوية لمصانع الأغذية المضافة للخبز

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يلعب الإجهاد التأكسدى دورا هاما فى تطور وظهور العديد من الأمراض بما فيها مرض السمنة. ينشأ نظام الإجهاد التأكسدى نتيجة لاختلال التوازن بين انتاج كل من مشتقات الأكسدة والمواد التي تمثل نظم الدفاعات المضادة للاكسدة. لذلك، تهدف الدراسة االحالية إلى التعرف على حالة جهد التأكسد ونظم الدفاع المضاد للاكسدة في الفئران المصابة بالسمنة والتي تم تغذيتها علي بعض النواتج الثانوية لمصانع الأغذية المضافة للخبز. لذلك تم تقسيم اثني وأربعون فأر (١٤٠ ± ١٠جم) إلى مجموعتين رئيسيتين، المجموعة الأولى (مجموعة ١، ٦ فئران) تم تغذيتها على الغذاء الأساسي، والمجموعة الرئيسية الأخرى (٣٦ فار) تم تغذيتها علي نظام غذائي يسبب السمنة (DIO) لمدة ٨ أسابيع، تم تقسيمها فيما بعد إلي ستة مجموعات فرعية على النحو التالي: المجموعة (٢) تم تغذيتها على غذاء (DIO) كمجموعة ضابطة موجبة، اما المجموعات (٣ - ٧) تم تغذيتها علي غذاء DIO يحتوي على ٥٪ من مسحوق قشر البطاطس، مسحوق اوراق القنبيط، مسحوق قشر البصل الاحمر، مسحوق قشر المانجو ومسحوق مخلوطهم على التوالي. وفي نهاية فترة التجربة (٨ اسابيع) سجلت أوزان الفئران المصابة بالسمنة زيادة في الوزن بنسبة ١٤٨.٧٠٪ مقارنة بالفئران السليمة (الغير مصابة). كما أشارت النتائج الى أن الاصابة بالسمنة قد ادت إلي ارتفاع معنوي في تركيز المؤكسدات بالبلازما (حمض الثايوبربيوتك بمعدل ٤١.٩٥٪، النيتريت بمعدل ٣١.٠٢٪ وتركيز النتيريت/النترات ٢٦.٤٥ بمعدل ٪)، وانخفاض معنوي في كل من مضادات الاكسدة الغير انزيمية (الجلوتاثيون فى صورته المختزلة ٣٠.٨٣ بمعدل ٪ و والجلوتاثيون فى صورته المؤكسدة بمعدل ١١.٢٧٪)، والفيتامينات المضادة للاكسدة (فيتامين (أ) بمعدل ٢٧.٤٣٪، فيتامين (ج) بمعدل ٢٠.٩٨٪ و فيتامين (هـ) بمعدل ٣١.٥٠٪) وكذلك في مستوي مضادات الاكسدة الانزيمية في كرات الدم الحمراء (انزيم الجلوتاثيون بيروكسيداز بمعدل ٣٧.٦٦٪، انزيم الجلوتاثيون ريداكتيز بمعدل ٢٨.٦٦٪، انزيم الكالتيز بمعدل ١٩.٥١٪ وانزيم السوبرأكسيد ديسميوتاز٢٥.٢٦ بمعدل ٪) بالمقارنة بالمجموعة الضابطة السالبة. الا ان التغذية علي ٥٪ من مسحوق قشر البصل الاحمر، قشرالمانجو، قشر البطاطس و اوراق القنبيط، ومخلوطهم قد ادي الي تحسن في مستويات جميعالتقديرات السابقة، وقد سجلت افضل التأثيرات لمخلوط النواتج ثم مسحوق قشر البصل الاحمر، مسحوق قشرالمانجو، مسحوق قشر البطاطس و مسحوق اوراق القنبيط، علي التوالي. واستنتجت الدراسة ضرورة تعديل النظام الغذائي بحيث يحتوي علي المركبات النشطة بيولوجيا والفيتامينات المضادة للاكسدة للحد من الاجهاد التأكسدي المرتبط بمرض السمنة.

الكلمات المفتاحية: حمض الثايوبربيوتك، النيتريت، الفيتامينات المضادة للاكسدة، مضادات الاكسدة الانزيمية، ورق القرنبيط، قشر البصل، قشر المانجو، قشر االبطاطس.