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Nutritional And Biochemical Studies On Obesity And Its Complications In Population Of Sharkia And Qalyubia Governorates, Egypt

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Abstract: Obesity is nowadays considered as a top risk factor in the development of many diseases and is causative of morbidity of patients suffering from metabolic syndrome. Some studies have examined the association many chronic diseases with overweight and obesity. However, for a number of populations, including the Egyptian, information on this relationship is missing. Therefore, purpose of the present study was to analyze the socio-demographic characteristics, nutritional and biochemical parameters and oxidative stress and antioxidant defense systems on obese and control adult groups in a trial to investigate the correlation between obesity and many other diseases that may be the reason, or the so-called obesity complications. Socio-demographic characteristics of the studied groups found that persons from rural areas (58.33%), low and middle social class (79.76%), illiterate to middle education (76.19%) and big family size (4-5 persons, 35.71%) were the significant risk factors for obesity. Biochemical analysis data indicated that obese adult group recorded significant ($p \leq 0.05$) higher values for the liver enzymes activities (ALT, 29.44%; AST, 27.95% and AP, 34.35), kidney functions (uric acid, 26.64% and creatinine, 29.62%) and serum glucose (28.24%) than the normal group. For blood lipids profile, obese group exhibited significant ($p \leq 0.05$) higher values for TG (33.63%) TC (34.80%), LDL (84.05) and VLDL (33.63) while significant decreased ($p \leq 0.05$) in HDL (-28.03) than the normal groups. Additionally, obese group adults recorded significant ($p \leq 0.05$) higher values for oxidative stress parameter (TBARS, 36.05%; NO_2 , 30.29 and $NO_3 + NO_2$, 28.54) and significant ($p \leq 0.05$) lower values for antioxidative defense systems [glutathione fractions, GSH (-33.36%), GSSG (-19.61%), GSH/GSSG (-17.11%) and antioxidant vitamins, A (-39.33), C (-13.25) and E (-23.68%)] than the normal groups. Such data confirmed the correlation between the obesity and the pathogenesis of many diseases that may be the reason including liver, kidney, heart and diabetes diseases. This may be useful in the design and dissemination of awareness of nutritional and health programs mandated by authorities.

Keywords: Liver functions, kidney functions, blood lipids profile, serum glucose, oxidative stress, albumin, selenium, glutathione fractions, antioxidant vitamins**.**

Introduction

Obesity is defined as an excessive accumulation of body fat mass to the extent that individual's health will be negatively affected. Although often viewed as equivalent to increased body weight, this need not be the case-lean but very muscular individuals may be overweight by numerical standards without having increased adiposity. Body weights are distributed continuously in populations, so that choice of a medically meaningful distinction between lean and obese is somewhat arbitrary. Obesity is therefore more effectively defined by assessing its linkage to morbidity or mortality. Although not a direct measure of adiposity, the most widely used method to gauge obesity is the *body mass index* (BMI), which is equal to weight/height2 (in kg/m2). Using data from the Metropolitan Life Tables, BMIs for the midpoint of all heights and frames among both men and women range from 19–26 kg/m2; at a similar BMI, women have more body fat than men. Based on data of substantial morbidity, a BMI of 30 is most commonly used as a threshold for obesity in both men and women (WHO, 2000).

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. According to the WHO, (2006), there are more than one billion overweight adults in the world. At least 300 million of them are clinically obese and of these about 115 million come from developing countries (WHO and Dini, 2006). Furthermore, in the past 20 years, the rates of obesity have tripled in developing countries (Hossain *et al.,* 2007). 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).

Obesity is a worldwide escalating problem caused by a complex interaction of genetic, socio-demographic, behavioral and environmental factors. Underlying factors associated with overweight and obesity, as shown in different population-based studies, are

physical inactivity (Swallen *et al.,* 2005; Al-Nozha *et al.,* 2007 and Al-Hazzaa, 2004) , diabetes(El-Hazmi and Warsy, 2000 and Warsy and El-Hazmi, 1999) dietary habits (Amin *et al.,* 2013) , gender (Al-Hazzaa, 2007) , (Al-Othaimeen *et al.,* 2007 and Al-Almaie , 2005) employment and education (Amin *et al.,* 2008 and Al-Baghli *et al.,* 2008).

Additionally the previous risk factors of obesity, systemic oxidative stress is part of the numerous biological alterations reported during chronic obesity (Roberts and Sindhu, 2009). Oxidative stress is a general term for cellular damage caused by an imbalance between pro-oxidants such as ROS and/or reactive nitrogen species (RNS) antioxidants. ROS are oxidizing agents generated during cellular metabolism when the chemical reduction of oxygen forms unstable free radicals, characterized by an unpaired electron (Montezano and Touyz (2012). ROS are essential for physiological functions such as gene expression, cellular growth, infection defense, and modulating endothelial function (Vider et al., 2001). However, to maintain a physiologically beneficial level of ROS within cells, antioxidants are necessary. Antioxidants are enzymatic and nonenzymatic molecules which significantly delay or prevent the oxidizing damage of ROS through the inhibition of ROS formation and action or by repairing cells which have been damaged by ROS (Kunwar and Priyadarsini, 2011). Evidences regarding obesity-induced oxidative stress are derived from several clinical studies, which have established correlations of biomarkers, or end-products of free radicals-mediated oxidative stress (lipid peroxidation or protein carbonylation products) with body mass index (BMI) (Sankhla *et al.,* 2012). In contrast, an inverse relationship exists between body fat, visceral obesity, and antioxidant defense markers in obese individuals (Chrysohoou *et al.,* 2007). So, many studies assume that most of the complications of obesity or its causes of disease come through the path of oxidative stress.

Some studies have examined the association of type 2 diabetes, cardiovascular, liver, immunodeficiency and aging diseases with overweight and obesity (Lakka *et al.,* 2002 and Sankhla *et al.,* 2012). However, for a number of populations, including the Egyptian, information on this relationship is missing. Therefore, the first purpose of the present study was to analyze Socio-demographic characteristics

differences between in a sample of Egyptian adults. A second purpose was to analysis the nutritional and biochemical parameters on the obese and control samples in a trial to investigate the correlation between obesity and many other diseases that may be the reason, or the so-called obesity complications. A third purpose was to analyze whether oxidative stress and antioxidant defense systems varies between obesity and noral subjects. Investigation of such research point may be useful in the design and dissemination of awareness of nutritional and health programs mandated by authorities.

Materials and Methods

Study protocol and criteria

This study was conducted from June 2015 to March 2016, in seven hospitals and Medical Care Centers of Sharkia and Qalyubia governorates, Egypt. These hospitals and Medical Care Centrs serve most adults from low and middle socioeconomic standards. The study protocol was carried out in accordance with the Helsinki Declaration as revised in 1989 and approved by the Local Committee for Scientific Research Ethics. Study groups participated were 84 adult persons (46.43% male and 53.57 female), 28 normal and 56 obese. All subjects were informed about the study protocol and written consent was obtained from all participants. A field pretested interviewing questionnaire was used for data collection which covering the following points: age, sex, residence (urban or rural) and family size. Socio-demographic status of participants and their families and socioeconomic score, which contained social variables were calculated and classified. Participants reported their height and weight from which we calculated body mass index (BMI) and categorized individuals as being not overweight/ obese, overweight, or obese (WHO, 2000).

Chemicals and Equipment

Vitamins standards (A, C, and E) 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.

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.

Hematological analysis

Blood samples were withdrawn from the antecubital vein 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 hematological analysis (Stroev and Makarova, 1989).

Serum glucose

Enzymatic determination of serum glucose was carried out colorimetrically according to Yound, (1975).

Liver functions

Aspartate aminotransferase (AST), alanine aminotransferase (ALT) were determined according to Yound, (1975) and Tietz, (1976) respectively by using specific kits supplied by Biocon Company, Cairo, Egypt.

Albumin

Albumin was determined in plasma using kits purchased from El-Nasr Pharmaceutical Chemicals Company, Cairo, Egypt.

Kidney functions

Serum creatinine and urea concentration was determined using specific kits supplied by Biocon Company, Cairo, Egypt, respectively.

Blood lipids profile

Triglycerides (TG), Total cholesterol (TC) and HDL-Cholesterol were determined in serum using specific kits purchased from El-Nasr Pharmaceutical Chemicals Company, Cairo, Egypt. Low density lipoprotein cholesterol (LDL-c) and very low density lipoprotein cholesterol (VLDL-c) were assayed according to the equations of Fniedewald *et al.,* (1972) *as* follow:

Very low density lipoprotein (VLDL cholesterol) = TG/5

LDL cholesterol = Total cholesterol – HDL cholesterol – V LDL cholesterol

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*-carboxymethyl 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 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 untill 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 92.32 ± 2.65, 86.55± 4.95 and 88.43 ± 5.77, respectively.

Selenium content

Selenium content in plasma samples were determined by the adaptation the method mentioned by Singh *et al*., (1991). One hundred µl of plasma sample were transferred into a digested glass tube and 2 ml of tri-acids mixture (containing nitric acid : perchloric acid : sulfuric acid in the ratio of 20 : 4 : 1 v/v respectively) were added to each tube. The tubes content were digested gradually as follow, 30 min at 70 ^{0}C; 30 min at 180 ^{0}C and 30 min at 220 ^{0}C. After digestion, the mixture was cooled, dissolved in MilliQ water, and the volume was increased to 10 ml in volumetric beaker. After filtration in ashless filter paper, aliquots were analyzed for selenium content using of atomic absorption spectrophotometer, type Perkin - Elmer, Model 2380.

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 $FeSO_4.7H_2O$ 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

Statistical analysis were performed by using computer program statistical package for social science (SPSS) and compared with each other using the suitable tests. Statistical analysis has been achieved using MB-PC computer by SPSS program (Levesque, 2007).

Results and discussions

Socio-demographic characteristics of the studied groups

Data in Table (1) showed socio-demographic characteristics of the studied group. From such data it could be noticed that there was

Table 1. Socio-demographic characteristics of the studied groups

Variables	Control		Obese		Total Number	
	Number	Percentage (%)	Number	Percentage (%)	Number	Percentage (%)
Sex:						
– Male	16	57.14	23	41.07	39	46.43
– Female	12	42.86	33	58.93	45	53.57
Total	28	100	56	100	84	100.00
Age (years):						
– 18-39	4	14.29	7	12.50	11	13.10
– 40-49	11	39.29	16	28.57	27	32.14
– 50-59	6	21.43	18	32.14	24	28.57
– 60-69	3	10.71	11	19.64	14	16.67
– ≥ 70	4	14.29	4	7.14	8	9.52
Total	28	100	56	100	84	100.00
Education:						
– Illiterate/can read and write	6	21.43	8	14.29	14	16.67
– Primary/prepar-tory	4	14.29	15	26.79	19	22.62
– Secondary school	12	42.86	19	33.93	31	36.90
– University or higher	6	21.43	14	25.00	20	23.81
Total	28	100	56	100	84	100.00
Family size (person):						
– 1	3	10.71	9	16.07	12	14.29
– 2-3	7	25.00	17	30.36	24	28.57
– 4-5	10	35.71	20	35.71	30	35.71
– >5	8	28.57	10	17.86	18	21.43
Total	28	100	56	100	84	100.00
Residence:						
– Urban	11	39.29	24	42.86	35	41.67
– Rural	17	60.71	32	57.14	49	58.33
Total	28	100	56	100	84	100.00
Social class:						
– Low	9	32.14	20	35.71	29	34.52
– Middle	12	42.86	26	46.43	38	45.24
– High	7	25.00	10	17.86	17	20.24
Total	28	100	56	100	84	100.00

considerable variability with regard to the age, level of education, family size, residence and social class of the study participants. Study groups participated were 84 adult persons (46.43% male and 53.57 female), 28 normal and 56 obese. A logistic regression model was used to assess the effects of the significant explanatory variables in order to distinguish predictors of obese. It was found that persons from rural areas (58.33%), those from low and middle social class (79.76%), those of illiterate to middle education (76.19%) and those from big family size (4-5 persons, 35.71%) were the significant risk factors for obesity in these adults. In similar studies, resechers found that sex, age, smoking, physical activity level, educational level, body image, health consciousness and social desirability , sports participation are other factors beside dietary intake were associated with increased risk of obesity (Johansson *et al.,* 1998 and 2001 and Nathalie *et al.,* 2007).

Classification of obese group according to body mass index (BMI)

Classification of obese group according to body mass index (BMI) was shown in Table (2). Such data indicated that almost of the obese subjects (41.07% and 35.71) were classified as obese I and obese II. According to WHO (2000) classification such groups have moderate and severe for Risk of co-morbidities, respectively. Such as mentioned by Lakka *et al.,* (2002) and Nathalie *et al.,* (2007) associations between obesity with type 2 diabetes, cardiovascular disease and mortality were recorded. Therefore, our data probably illustrated the important role that obesity plays as one of the factors that strongly influence quality of life in Egypt.

Liver function enzymes activity and albumin in serum of control and obese studied groups

The liver inflammation and functions-improving effects were evaluated according to serum alanine aminotransferase (ALT) , serum aspartate aminotransferase (AST) and serum alkaline phosphatase (AP) activities (serum biochemical indicators for liver inflammation), as well as serum albumin (Alb) level (liver cell regeneration indicators). Mean ±SD values of liver function enzymes activity and albumin levels in serum of control and obese studied groups were shown in Table (3). From such data it could be noticed that obesity group recorded a significant ($p \leq 0.05$) increasing in ALT (29.44%), AST (27.95%) and AP (34.35%) compared to normal group. On the other side, an opposite

Table 2. Studied obese group classification according to body mass index (BMI)

BMI (Classification, Risk of co-morbidities) According to WHO, (2000)	Control		Obese		Total	
	Number	Percentage (%)	Number	Percentage (%)	Number	Percentage (%)
18.5-24.9 (Normal range, Average)	28	100	------	------	28	33.33
25-29.9 (Overweight, Increased)	------	------	5	8.93	5	5.95
30-34.9 (Obese I, Moderate)	------	------	23	41.07	23	27.38
35-39.9 (Obese II, Severe)	------	------	20	35.71	20	23.81
≥ 40 (Obese III, Very severe)	------	------	8	14.29	8	9.52
Total	28	100	56	100	84	100.00

Table 3. Mean ±SD values of liver function enzymes activity and albumin in serum of control and obese studied groups

Groups		ALT (U/L)	AST (U/L)	AP (U/L)	Alb (g/L)
Control study population	Range	51.21-66.23	30.54-37.98	114.20-133.14	42.67-60.45
	Mean	60.56^{b}	32.39^{b}	125.67^{b}	49.09^{b}
	SD	7.99	4.39	8.95	6.03
Obese study population	Range	65.78-88.32	35.87-51.05	144.01-177.45	30.74-41.71
	Mean	78.39^{a}	41.44^{a}	168.83^{a}	36.90^{a}
	SD	11.15	6.01	19.50	7.44
% of change		29.44	27.95	34.35	-24.83

* Mean values with the different superscript letters in the same column are significantly different ($p \leq 0.05$).

direction was observed for the Alb levels in serum. Such data are in accordance with that recorded in experimental animals by Sayed Ahmed (2106).

Aminotransferases are normally intracellular enzymes. Thus, the presence of elevated levels of aminotransferase in the serum indicates damage to cells rich in these enzymes. For example, physical trauma or a disease process can cause cell lysis, resulting release of intracellular enzymes into the blood. Two amino transferases were found in plasma

are of particular diagnostic value AST and ALT. AST enzyme is one of the enzymes tested in the cardiac enzyme series. This enzyme is found in very high concentration within the heart muscles, skeletal muscle cells, and to a lesser degree in the kidney and panaceas (Pagana and pagana, 1997). ALT is found predominately in the liver leaser quantities are found in the kidneys, heart and skeletal muscles (Pagana and pagana, 1997). These enzymes are elevated in nearly all liver diseases, but are particularly high in conditions that the causes extensive cell necrosis, such as severe viral hepatitis and prolongated circulatory collapse. Serial enzyme measurements are often useful in determining the course of liver damage (Abd El-Aziz, 1990 and Pagana and pagana, 1997). Also, aminotransferases may be elevated in nonhepatic disease, such as myocardial infraction and muscle disorders; however, these disorders can usually be distingushed clinically from liver disease (Champe and Harvey, 1994). Alkaline phosphatatse (AP) is an enzyme which catalyzes the hydrolysis of phophate esters at an alkaline pH to give pi and the corresponding alcohol, phenol or sugar. Although AP is found in many tissues, the highest concentrations are found in the liver, biliary tract , epithelium and bone. The intestinal mucosa and placenta also contain AP (Pagana and pagana, 1997). Also, elevated serum and leukocytic AP leaves in patients with Hodgkin`s, non-Hodgkin`s lymbhoma, myloprolifrative and lymphoproliferative disorders, were reported by several investigators.

Alb is an important metal binding protein. It is a sacrificial antioxidant that can bind copper tightly and iron weakly to its surface serving as a target for their related free radical reactions. Thus it inhibits copper ion dependent lipid peroxidation (Gutteridge and Wilkins, 1983). It was reported that hypo-albuminaemia is most frequent in the presence of advanced chronic liver diseases. Hence decline in total protein and albumin can be deemed as a useful index of the severity of cellular dysfunction in chronic liver diseases.

Data of the present study with the other reported that aminotransferases and AP activities as well as Alb level may be elevated significantly in additionally nonhepatic disease such as obesity in human and experimental animals (Elhassaneen and Salem, 2015 and Sayed Ahmed, 2016).

Kidney function parameters of control and obese studied groups

Data in Table (4) indicated the mean ±SD values of kidney function parameters in serum of control and obese studied groups were shown in Tables (6-8) and Figure (2). From such data it could be noticed that obese group recorded a significant ($p \leq 0.05$) increasing in uric acid (26.64%) and creatinine (29.62%) compared to normal group. Such data are in accordance with that recorded in experimental animals by Sayed Ahmed (2106). Urea is formed in the liver as the end product of protein metabolism. During ingestion, protein is broke down into amino acids. In the liver, these amino acids are catabolized and free ammonia is formed. The ammonia is combined to form urea (Pagana and pagana, 1997). Urea, the major product of protein catabolism measuring urea is the most popular laboratory procedure for assessing renal function (Pagana and pagana, 1997). Creatinine is a catabolic product of creatine phosphate, which is used in skeletal muscle concentration (Pagana and pagana, 1997). In the skeletal muscle serum creatinine levels are elevated by renal disease and dehydration. The possible mechanism (s) of action of kidney parameters -increasing activity of the obesity is still not fully understood. But, our data suppose that obese group are more susceptible to liver disorders subsequently risk of kidney disease when compared with the normal group.

Table 4. Mean ±SD values of some kidney function parameters of control and obese studied groups

Groups		Uric acid (mmol/L)	Creatinine (μmol/L)
Control study population	Range	0.14-0.23	74.78-94.76
	Mean	0.19 [a]	85.02 [b]
	SD	0.04	9.93
Obese study population	Range	0.22-0.30	96.08-126.98
	Mean	0.24 [a]	110.21 [a]
	SD	0.05	14.37
% of Change		26.64	29.62

* Mean values with the different superscript letters in the same column are significantly different (p≤0.05).

Blood lipids profile parameters in serum of control and obese studied groups

Mean ±SD values of some blood lipid profile parameters in serum of control and obese studied groups were shown in Table (5). From such data it could be noticed that obesity group recorded a significant ($p \leq 0.05$) increasing in triglycerides (TG, 33.63%), total cholesterol (34.80, TC) and low density lipoprotein (LDL, 84.05%) and very low density lipoprotein (VLDL, 33.63%) while significant ($p \leq 0.05$) decreasing in high density lipoprotein (HDL, -28.03%) compared to normal groups. Such data are in accordance with that recorded by (Elhassaneen and Salem, 2015). Such as mentioned by Lakka *et al.,* (2002) and Nathalie *et al.,* (2007) associations between obesity with cardiovascular disease as the result of alterations in blood lipids profile and mortality was recorded. On the same direction, modeling studies suggests that modest and sustained weight loss (5 kg –10 kg) in patients with overweight or obesity is associated with reductions in LDL, TC and TG and with increased levels of HDL (Avenell *et al.,* 2004; Christensen *et al.,* 2007; Bales and Buhr, 2008 and Williamson *et al.,* 2009). Also, many survey studies revealed that obesity was an independent risk factor for the 26-year incidence of cardiovascular disease in men and women [including coronary disease, stroke, and congestive heart failure (CHF)] (Wells, 2009). When the additional effects of hypertension and glucose intolerance associated with

Table 5. Mean ±SD values of some blood lipids profile parameters in serum of control and obese studied groups

Groups		TG (mg/dl)	TC (mg/dl)	HDL (mg/dl)	LDL (mg/dl)	VLDL (mg/dl)
Control study population	Range	70.32-88.23	110.01-138.54	42.09-54.87	60.76-70.65	11.05-16.32
	Mean	79.06 [b]	129.67 [b]	49.87 [a]	63.99 [b]	15.81 [b]
	SD	8.03	10.98	3.34	5.67	2.98
Obese study population	Range	89.09-117.23	160.06-184.98	29.87-41.65	105.87-121.87	17.60-23.79
	Mean	105.65 [a]	174.79 [a]	35.89 [b]	117.77 [a]	21.13 [a]
	SD	11.14	11.67	5.09	8.94	3.76
% of change		33.63	34.80	-28.03	84.05	33.63

* Mean values with the different superscript letters in the same column are significantly different (p≤0.05).

obesity are included, the adverse impact of obesity is even more evident. The effect of obesity on cardiovascular mortality in women may be seen at BMIs as low as 25. Obesity, especially abdominal obesity, is associated with an atherogenic lipid profile; with increased LDL, VLDL and TG; and with decreased HDL and decreased levels of the vascular protective adipokine adiponectin. Obesity is also associated with hypertension. Measurement of blood pressure in the obese requires use of a larger cuff size to avoid artifactual increases. Obesity-induced hypertension is associated with increased peripheral resistance and cardiac output, increased sympathetic nervous system tone, increased salt sensitivity, and insulin-mediated salt retention; it is often responsive to modest weight loss (Montezano and Touyz, 2012).

Serum glucose of control and obese studied groups

Mean ±SD values of serum glucose in serum of control and obese studied groups were shown in Table (6). From such data it could be noticed that obese group recorded a significant ($p \leq 0.05$) increased in serum glucose (28.24%) compared to normal group. Such data are in accordance with that recorded in experimental animals by Sayed Ahmed (2106). Also, Avenell *et al.,* (2004) reported that in patients with type 2 diabetes, weight loss of around 5 kg is associated with a reduction in fasting blood glucose of between 0.17 mmol/L to 0.24 mmol/L at 12 months. Furthermore, Williamson *et al.,* (2009) mentioned that overweight or obese patients with type 2 diabetes who received an intensive lifestyle intervention which yielded significant weight loss (9 kg), had improved physical fitness, reduced physical symptoms and experienced significant improvements in health-related quality of life compared with those who received diabetes support and education who lost less than 1 kg. Insulin resistance and type 2 diabetes mellitus inrelationship with obesity were studied by many authors who reported that hyperinsulinemia and insulin resistance are pervasive features of obesity, increasing with weight gain and diminishing with weight loss (Jiajj, 2009 and Van and Astrup, 2009). Insulin resistance is more strongly linked to intra-abdominal fat than to fat in other depots.The molecular link between obesity and insulin resistance in tissues such as fat, muscle, and liver has been reported. Finally, Kershaw (2004) reviewed that obesity, however, is a major risk factor for diabetes, and as many as 80% of patients with type 2 diabetes

mellitus are obese. Weight loss and exercise, even of modest degree, are associated with increased insulin sensitivity and often improve glucose control in diabetes. Several oxidative enzymes such as myeloperoxidase and lipoxygenases have been shown to involve in LDL oxidation and are associated with the development of obesity along with inflammation and insulin resistance (Carr et al., 2000 and Wang *et al.,* 2014).

Table 6. Mean±SD values of glucose in serum of control and obese studied groups

Groups		Glucose (mmol/L)
Control study population	Range	2.76-3.22
	Mean	2.91 [b]
	SD	0.26
Obese study population	Range	3.30-4.45
	Mean	3.73 [a]
	SD	0.64
% of change		28.24

* Mean values with the different superscript letters in the same column are significantly different (p≤0.05).

Oxidative stress in serum of control and obese studied groups

Oxidative stress parameters i.e. thiobarbituric acid reactive substances (TBARS) and nitric oxides (nitrite, NO_2 and nitrate, NO_3) were assayed in normal and obese groups (Table 7). From such data it could be noticed that obese group recorded a significant ($p \leq 0.05$) increasing in TBARS, NO_2 and NO_2/NO_3concentration in serum by 36.05, 30.29 and 28.54% compared to normal group, respectively. 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). On the same context, clinical evidences for obesity-associated oxidative stress have been provided by measurement of either biomarkers or end-products of free radical-mediated oxidative processes (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 (Elhassaneen and Salem, 2015). Under normal physiological conditions, nitric oxide (NO) is a critical homeostatic regulator of the vessel wall and plays a role in the maintenance of vascular tone and reactivity (Verma and Anderson, 2002). However, when ROS production is elevated, the process of cell damage occurs and can possibly facilitate the development of CVD (Ji *et al.,* 2006) which is largely attributed to oxidation of low-density lipoprotein (LDL) (Zhang *et al.,* 2014). Several oxidative enzymes such as myeloperoxidase and lipoxygenases have been shown to involve in LDL oxidation (Carr *et al.,* 2000) and are associated with the development of obesity along with inflammation and insulin resistance (Wang *et al.,* 2014).

Table 7. Mean ±SD values of oxidants concentration in serum of control and obese studied groups

Groups		TBARS, (nmol/mL)	NO_2 (nmol/L)	NO_2/NO_3, (nmol/L)
Control study population	Range	2.41-3.10	2.12-2.79	3.52-4.29
	Mean	2.73 [b]	2.41 [b]	3.96 [b]
	SD	0.31	0.35	0.38
Obese study population	Range	3.34-4.19	2.71-3.69	4.31-6.11
	Mean	3.71 [a]	3.14 [a]	5.09 [a]
	SD	0.52	0.41	0.61
% of change		36.05	30.29	28.54

* Mean values with the different superscript letters in the same column are significantly different ($p \leq 0.05$).

Biological antioxidant macromolecules in serum of control and obese studied groups

Biological antioxidant macromolecules i.e. glutathione fractions concentration (reduced glutathione, GSH and oxidized glutathione, GSS) and selenium (Se) levels in serum of control and obese studied groups were assessed (Table 8). From such data it could be noticed that obese group recorded a significant ($p \leq 0.05$) decreased in GSH, GSSG and Se concentrations in serum by -33.36, -19.61 and -43.03% compared to

Table 8. Mean ±SD values of glutathione fractions in serum of control and obese studied groups

Groups		GSH (µmol /L)	GSSG (µmol /L)	GSH/GSSG ratio	Se (µmol/L)
Control study population	Range	10.02-12.54	0.82-1.33	9.12-12.23	1.22-2.02
	Mean	11.21 [a]	1.02 [a]	10.99 [a]	1.65 [a]
	SD	1.05	0.27	1.12	0.34
Obese study population	Range	6.11-9.02	0.64-1.10	7.76-10.70	0.62-1.20
	Mean	7.47 [b]	0.82 [a]	9.11 [b]	0.94 [b]
	SD	1.78	0.21	1.54	0.28
% of change		-33.36	-19.61	-17.11	-43.03

* Mean values with the different superscript letters in the same column are significantly different ($p \leq 0.05$).

normal group, respectively. The same behavior was observed for the GSH/GSSG ratio.

GSH (tripeptide-thiol, γ-glutamyl cysteinyl-glycine) is highly abundant in all cell compartments and is the major soluble antioxidant. GSH/GSSG ratio is a major determinant of oxidative stress. GSH shows its antioxidant effects in several ways (Masella *et al.,* 2005). It detoxifies hydrogen peroxide and lipid peroxides via action of GSH-Px. GSH donates its electron to H_2O_2 to reduce it into H_2O and O_2. GSSG is again reduced into GSH by GSH reductase that uses NAD(P)H as the electron donor. GSH-Pxs are also important for the protection of cell membrane from lipid peroxidation. Reduced glutathione donates protons to membrane lipids and protects them from oxidant attacks (Curello *et al.,* 1985). GSH is a cofactor for several detoxifying enzymes, such as GSH-Px and transferase. It has a role in converting vitamin C and E back to their active forms. GSH protects cells against apoptosis by interacting with proapoptotic and antiapoptotic signaling pathways. (Masella *et al.,* 2005). Decreasing in GSH fractions observed in obese 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. A marked decreased level of GSH is reported in the plasma of obese patients (reviewed in Rahman *el al.,* 2012). By other meaning, obesity could be induced its

pathogenesis through depression the antioxidant defense systes including GSH.

On the other side, Se plays a key role in the maintenance of normal health in human populations (Safaralizadeh *et al.,* 2005). It is part of the active site of glutathione peroxidase (GSH-Px), an antioxidant enzyme. GSH-Px controls the interacellular level of hydrogen peroxide, reducing the formation of reactive oxygen species that can induce lipid peroxidations with consequent damage to the cellular membranes (Rotruck *et al.,* 1973). There is increasing evidence that selenium deficiency may have several serious short- and long-term medical implications, including impaired immune response, or even cancer (Kohrle *et al.,* 2000). Basing on this basis, the decreasing in serum Se level recorded in obese group could be affected in the pathogenesis of obese related diseases.

Antioxidant vitamins concentration in serum of control and obese studied groups

The reducing in biological antioxidant macromolecules (GSH and GSSG) was contrary with significant decreasing (p>0.05) in antioxidant vitamins in human serum as a consequence of obesity injury (Table 9). The rate of decreasing in vitamins A, C and E were recorded -39.33, -13.25 and -23.68 % in obese group compared to normal group. The decreasing rate was higher in fat soulbe vitamins (A and E) than water soluble vitamin (C). According to these results, the decreasing in antioxidant vitamins in serum could be attributed to their consumption in scavenge, quench and/or trap different ROS resulted from obese injuries.

Vitamins include A, E and C, the non-enzymatic antioxidants that prevent or retards the oxidation of sensitive molecules found in the body. Vitamin E is considered as primarily intracellular antioxidants associated with cell membranes (krinskey, 1992). It is a potent peroxyl radical scavenger and can protect polyunsaturated fatty acids (PUFA) within phospholipids of biological membranes and in plasma lipoproteins (Jialal *et al*., 1995). It is react directly with a variety of oxyradicals including the peroxyl radical (LOO), trichloromethyl radical (CCl_3) and hydroxyl radical ($OH^.$) (McCay, 1985). Furthermore vitamin E reacts with superoxide radical and singlet oxygen (Burton and Inglod, 1981). Vitamin E donates hydrogen to chain propagating

Table 9. Mean ±SD values of antioxidant vitamins in serum of control and obese studied groups

Groups		Vitamin A (retinol) (μmol/L)	Vitamin C (ascorbic acid) (μmol/L)	Vitamin E (tocopherol) (μmol/L)
Control study population	Range	1.13-2.16	56.04-66.54	28.17-36.88
	Mean	1.62 [a]	61.43 [a]	33.70 [a]
	SD	0.43	4.89	4.11
Obese study population	Range	0.59-1.30	45.28-60.17	20.87-28.67
	Mean	0.98 [b]	53.29 [b]	25.72 [b]
	SD	0.42	6.93	3.94
% of change		-39.33	-13.25	-23.68

* Mean values with the different superscript letters in the same column are significantly different ($p \leq 0.05$).

lipid peroxyle radicals, and thus convert them to less reactive forms (Heffner and Repine, 1989). *β*-carotene i.e. precursor of vitamin A and other carotenoids belong to the large family of conjugated polyenes. Carotenoids are bleached when exposed to radicals such as those that arise during lipid peroxidation, which indicates that these pigments; must also intercept active oxygen species. Their long, conjugated double bond systems make them excellent substrates for radical attack (Kennedy *et al*., 1991). They have antioxidant activity through its property as singlet oxygen (1 O_2) quenchers and their ability to trap peroxyl radicals (Truscott, 1990; Stahl and Sies 1993). They are also able to inhibit free radical reactions (Palozza and Krinsky, (1992). The products of the reaction between β-carotenes and radicals are primarily carbonyl derivatives of β-carotene along with some expoxides (Handelman *et al*., 1991). Vitamin C is an important antioxidant. Its water solubility allows it to be widely available in both the extracellular and intracellular spaces in most biological systems (Halliwell and Gutteridge 1990). Antioxidant roles of ascorbic acid can be' summarized in the following*:* scavenge $O_2^{.-}$ and $OH^{.}$ with the formation of the sernidehydro-ascorbate free radical that is subsequently reduced by GSH to generate dehydroascorbate and GSSG, as most cells contain a GSH-dependent dehydro ascorbate reductase that generates ascorbate and GSSG scavenges water-soluble peroxyl (RO_2) radicals (Frei, 1991), reduces nitroxide radicals, e.g. the

radicals formed by attack of O_2 or OH upon desferrioxamine (Hoffman and Garewell 1995) and it also protects plasma lipids against peroxidation induced by activated neutrophils (Frei. 1991).

Regarding obesity, Brinkmann *et al.,* (2012) suggested that oxidative stress induced as the result of obesity injuries leads to decrease the availability of plasma vitamins C and E. The decreasing in vitamins in obese group could be affective in the all functions performed by them including antioxidant defence systems, detoxification process etc. Such attention was confirmed by Shanmugam and Ayyalasomayajula (2015) reviewed that vitamin A as a key regulator of obesity and its associated disorders in obese rat model. Also, Olga *et al..,* (2013) reported that low concentrations of zinc, vitamins A and E in children who were overweight and obese were associated with lipids, inflammation and insulin resistance.

Correlation studies

In the correlation analysis, important differences were found between oxidative stress and antioxidant defense systems in obese group subjects (Table 10). From such data it could be noticed that there was a strong negative significant ($p\leq 0.05$) relationship between antioxidant macromolecules in serum [GSH (r^2 = -0.910) and GSSG (-0.854), antioxidant vitamins in plasma [vitamin A (r^2 = -0.775) and vitamin E (r^2 = - 0.874)], and TBARS concentration in serum. While, moderate negative significant ($p\leq 0.05$) relationship between water soluble antioxidant vitamins in serum [vitamin C (r^2 = -0.634) and TBARS concentration in serum. On the same time, there was a strong negative significant ($p\leq 0.05$) relationship between antioxidant macromolecules in plasma [GSH (r^2 = -0.851) and GSSG (r^2 = -0.815) antioxidant vitamins in serum [vitamin A (r^2 = -0.796) and vitamin E (r^2 = -833], and NO_2 concentration in serum. While, moderate negative significant ($p\leq 0.05$) relationship between water soluble antioxidant vitamins in serum [vitamin C (r^2 = -0.613) and NO_2 concentration in plasma. These correlations confirm that if there were no change in the antioxidant defense systems (antioxidant macromolecules in plasma and antioxidant vitamins) of obese subjects, it would be difficult to observe high oxidative stress parameters concentrations (TBARS and NO_2). In similar study, Lepage *et al*., (1996) reported that high levels of MDA (main compound in TBARS) in the plasma of obese patients were

Table 10. Correlation between oxidative stress and antioxidant defense systems in obese group

Parameters	R^{2*}	Parameters	R^{2*}
TBARS/GSH	- 0.910	NO2/GSH	- 0.851
TBARS/GSSG	- 0.854	NO2/GSSG	- 0.815
TBARS/Vit A	- 0.775	NO2/Vit A	- 0.796
TBARS/Vit C	- 0.634	NO2/Vit C	- 0.613
TBARS/Vit E	- 0.874	NO2/Vit E	- 0.833

* $P \leq 0.05$

associated with rather low levels of β-carotene. Evidences regarding obesity-induced oxidative stress are derived from several clinical studies, which have established correlations of biomarkers, or end-products of free radicals-mediated oxidative stress (TBARS) with BMI (Sankhla *et al.,* 2012). On the same side, an inverse relationship exists between obesity and antioxidant defense system markers in obese individuals (Chrysohoou *et al.,* 2007).

Conclusion

Obesity is nowadays considered as a top risk factor in the development of many diseases and is causative of morbidity of patients suffering from metabolic syndrome. Socio-demographic characteristics of the studied group found those adults from rural areas, low and middle social class, illiterate to middle education big family size were the significant risk factors for obesity. Biochemical analysis data indicated that obese adult group recorded significant ($p \leq 0.05$) higher values for the liver enzymes activities (ALT, AST and AP), kidney functions (uric acid and creatinine) and serum glucose than the normal group. For blood lipids profile, obese group exhibited significant ($p \leq 0.05$) higher values for TG, LDL and VLDL while significant decreased ($p \leq 0.05$) in HDL than the normal groups. Additionally, obese group adults recorded significant ($p \leq 0.05$) higher values for oxidative stress parameter (TBARS, NO_2 and NO_3) and significant ($p \leq 0.05$) lower values for antioxidative defense systems (glutathione fractions, GSH, GSSG, GSH/GSSG and antioxidant vitamins, A, C and E) than the normal groups. Such data confirmed the correlation between the obesity and the pathogenesis of many diseases that may be the reason including liver, kidney, heart and diabetes diseases. This may be useful in the design and dissemination of awareness of nutritional and health programs mandated by authorities.

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دراسات غذائية وبيوكيميائية على السمنة ومضاعفاتها في سكان محافظتى الشرقية والقليوبية، جمهورية مصر العربية

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الملخص:
تعتبر السمنة في الوقت الحاضر عامل خطر كبير يساهم بدرجة كبيرة في ظهور وتطوير العديد من الأمراض، بل وتعد سببا رئيسيا فى إنتشار المرض للمرضى الذين يعانون من متلازمة التمثيل الغذائي .وقد إختبرت بعض الدراسات العلاقة بين العديد من الأمراض المزمنة وزيادة الوزن والسمنة. ومع ذلك، بالنسبة لعدد من السكان، بما في ذلك المصريين، فمازالت المعلومات عن هذه العلاقة مفقودة. لذلك كان الغرض من هذه الدراسة هو تحليل الخصائص الاجتماعية والديموجرافية والمقاييس الغذائية والكيموحيوية والإجهاد التأكسدي وأنظمة الدفاع المضادة للأكسدة فى الأشخاص المصابون بالسمنة فى محاولة لإستبيان العلاقة بين العلاقة بين السمنة والعديد من الأمراض الأخرى التي قد تكون السمنة سببا فيها أو ما يسمى بمضاعفات السمنة. ووجدت الخصائص الاجتماعية الديموغرافية للمجموعات المدروسة أن الأشخاص من المناطق الريفية (٥٨,٣٣٪) والطبقة الاجتماعية المنخفضة والمتوسطة (٧٩,٧٦٪) والأميين إلى التعليم المتوسط (٧٦,١٩٪) وحجم الأسرة الكبير (٤-٥ أشخاص، ٣٥,٧١%) كانت تمثل عوامل الخطر الكبيرة المسببة للسمنة. أظهرت نتائج التحليل الكيموحيوى أن مجموعة البالغين الذين يعانون من السمنة المفرطة قد سجلت قيم معنوية ($p\leq0.05$)لأنشطة انزيمات الكبد (ALT ، ٢٩.٤٤٪، AST ، 27.95٪، AP ، ٣٤.٣٥)، وظائف الكلى (حمض اليوريك، ٢٦.٦٤٪ والكرياتينين، ٢٩.٦٢ ٪) ومستوى الجلوكوز في الدم (٢٨.٢٤٪) مقارنة بالمجموعة الطبيعية (الصحيحة). أما فيما يتعلق بصورة دهون الدم فقد اظهرت النتائج ان نسبة البدناء قد أظهرت إرتفاعا ملحوظا ومعنويا ($p\leq0.05$) فيما يتعلق بقيم الدهون الثلاثية (٣٣,٦٣%)، الكولستيرول الكلى (٣٤,٨٠%) ، الليبوبروتينات منخفضة الكثافة (٨٤.٠٥%)، الليبوبروتينات منخفضة الكثافة جدا (٣٣,٦٣%) بينما سجل إنخفاضا ملحوظا ($p\leq0.05$) فى الليبوبروتينات عالية الكثافة (-٢٨,٠٣%) مقارنة بالمجموعة الضابطة (الطبيعية). بالإضافة إلى ذلك، سجلت مجموعة البالغين من البدناء قيم عالية بدرجة معنوية ($p\leq0.05$) لمقاييس الإجهاد التأكسدي (TBARS ، ٣٦.٠٥٪ - NO2، ٣٠,٢٩% - NO3 + NO2 ، ٢٨,٥٤%) وقيم منخفضة بدرجة معنوية ($p\leq0.05$) فيما يتعلق بأنظمة الدفاع المضادة للأكسدة [كسور الجلوتاثيون ، GSH (-٣٣.٣٦٪)، GSSG (-١٩.٦١٪)، نسبة GSH/GSSG (-١٧.١١٪) والفيتامينات المضادة للأكسدة التى تشمل فيتامين أ (-٣٩,٣٣%) ، فيتامين جـ (-١٣,٢٥%) ، فيتامين هـ (-٢٣,٦٨%). ولقد أكدت النتائج العلاقة بين السمنة والتسبب في العديد من الأمراض بما في ذلك أمراض الكبد والكلى والقلب والسكري .وقد تكون تلك النتائج مفيده في تصميم ونشر الوعي بالبرامج التغذوية والصحية التي تكلف بها الهيئات المختلفة.

الكلمات المفتاحية: وظائف الكبد، وظائف الكلى، صورة دهون الدم، جلوكوز الدم، الإجهاد التأكسدى، الألبيومين، السيلينيوم، أجزاء الجلوتاثيون، الفيتامينات المضادة للأكسدة.