Association of anthropometric measurements with oxidant-antioxidant status among young Saudi females

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Short Title: Oxidative stress and Anthropometry

Summary

Present study aimed to explore the levels and correlation of oxidative stress biomarkers with anthropometry in a population of young Saudi females.

One hundred six normotensives, non-diabetic Saudi females, with minimally active lifestyle, based on their body mass index (BMI) were divided as; normal-weight (NW; n = 52), overweight (OW; n = 24) and obese (OB; n = 30). Anthropometric measurements [BMI, Waist Circumference (WC), Waist-Hip Ratio (WHR), Body Density (BD), Body Adiposity Index (BAI), % Body fat) and oxidative stress biomarkers; Thiobarbituric acid reactive substances (TBARS), 8-hydroxy-2-deoxyguanosine (8-OH-2dG: indicative of DNA/RNA damage), Superoxide Dismutase, Serum total antioxidant capacity) were recorded.

There was statistically significant higher 8-OH-2dG (pg/ml) in OB compared to NW (800.63 ± 6.19 vs 780.22 ± 3.34; *p* value = 0.007), as determined by one-way ANOVA and Tukey post hoc test. 8-OH-2dG was significantly and positively associated with BMI (r = 0.286, *p* = 0.004), WC (r = 0.280, *p* = 0.005), BAI (r = 0.26, *p* = 0.008), and % body fat (r = 0.27, *p* = 0.006).

There may be significantly increased DNA damage in normoglycemic, normotensive obese adolescent females. This can be linked to the amount of adipose tissue in the body as depicted by strong positive association between DNA damage and BMI, WC, BAI, and % body fat.

Key words: Oxidative stress, Reactive Oxygen Species, Anthropometry, Saudi Arabia, DNA/RNA damage

Introduction:

Researchers have studied the relationship between obesity and oxidative stress (OS) in healthy adults. Furokawa and his colleagues (2004) revealed that amount of body fats was linked to oxidative stress in humans and mice. Reactive oxygen species (ROS) increased, and expression of antioxidant enzymes decreased significantly in obese mice. Same results were achieved in cultured adipocytes. Similarly, Unver and his group (2015) documented increased oxidative stress in obese individuals compared to non-obese controls.

In healthy young individuals, there is a delicate balance between pro-oxidative and anti-oxidative processes. This equilibrium gets impaired with advancing age or pathological states such as hypertension or hyperglycemia etc., leading to OS. Mean age of the study participants in Furokawa et al. (2004) and Unver et al. (2015) were 56 ± 13 and 35.8 ± 7.4 years respectively. Both studies did not rule out confounding factors that may cause concomitant oxidative stress such as hypertension, hyperglycemia and physical inactivity. Brown et al. (2009) considered blood pressure, plasma glucose and lipid profile in evaluating oxidative stress in obese individuals. However, study participants' mean ages were 31, 35 and 38 years in normal-weight (NW), overweight (OW) and obese (OB) subjects respectively. Hence, it remains largely unknown whether this association between obesity and OS exists from an early age and if it has the similar trends even in the absence of hypertension, hyperglycemia and physical inactivity. Therefore, the present study was designed to explore the levels and correlation of some well-known biomarkers of oxidative stress and antioxidant enzymes with anthropometry in a population of Saudi females in late adolescent stage in the absence of hypertension, diabetes and physical inactivity.

Materials and Methods:

This cross-sectional study was carried out at Department of Physiology, College of Medicine, Imam Abdulrahman Bin Faisal University, Dammam, Saudi Arabia after getting ethical approval from institutional review board (IRB number; IRB-2015-01-087).

Five hundred young Saudi females from College of Medicine, Dentistry, Nursing and Applied Medical Sciences were invited to participate. Interested participants underwent blood pressure and fasting blood glucose measurements by Welch Allyn Spot Vital Signs monitor and glucometer (ACC-CHEK G, Roche Diagnostics GmbH, Mannheim, Germany) respectively. Self-reported physical activity data were collected by using International Physical Activity Questionnaire-short form (IPAQ-SF) (Craig et al. 2003). Responses were first converted to metabolic equivalent (MET) minutes/week and then total physical activity MET-minutes/ week were obtained by taking sum of walking, moderate and vigorous MET-minutes/week scores (Guidelines 2005). Students were categorized into inactive, minimally active and health enhancing physical activity active group as described by Kavouras et al. (2007). Minimum physical activity was defined as 5 or more days total physical activity scores of at least 600 MET-minutes/week.

Students were excluded if they were diabetics (Fasting blood sugar ≥ 126 mg/dl) or having impaired fasting glucose (Fasting blood sugar ≥ 110 mg/dl) (Expert committee 2003), hypertensive (Systolic BP ≥ 140 mmHg, Diastolic BP ≥ 90 mmHg) (Chobanian et al. 2003), falling in the category of physically inactive group, taking any antioxidant supplementation, having irregular menstruation or a smoker. In the end, 106 students fulfilling the inclusion criteria were selected, and written informed consent forms were signed.

Waist circumference (WC) was measured midway between the lowest rib and the iliac crest. Hip circumference (HC) was measured around the broadest portion of the buttocks. Body Mass Index (BMI) was calculated from subjects' body weight (kg) divided by the height square (m^2). BMI cut-off points for NW, OW and OB groups were <23, 23–27.4, ≥27.5 kg/m^2 respectively (WHO 2004). Body adiposity index (BAI) was calculated as described by Bergman et al. (2011).

$$BAI = \frac{Hip}{Height \times \sqrt{Height}} - 18$$

Skinfold thicknesses from four different anatomical sites (triceps, biceps, subscapular and suprailiac) were measured with skinfold calipers and substituted in the equation of Durnin and Womersley (1974) to calculate body density. $D = 1.1599 - (0.0717 \times L)$ where D = predicted density of the body (g/ml), and L = log of the sum of four skinfolds (mm). The density was converted to % body fat as described by Siri (1961). $\%\ Body\ Fat = \left(\frac{496}{Body\ Density}\right) - 450$

Blood samples obtained after an overnight fast were clotted, centrifuged and frozen at -80ºC. OS parameters were measured by Cayman chemicals kits 1) TBARS (Thiobarbituric acid reactive substances; Catalogue No. 10009055) having inter and intra-assay coefficient of variation of (5.1-5.9 %) and (5.5- 7.6 %) respectively; 2) 8-OH-2dG (8-hydroxy-2-deoxyguanosine; Catalogue No. 589320) inter and intra-assay coefficient of variation as 10.7 % and 11.6 % respectively; 3) Superoxide Dismutase (SOD; Catalogue No. 706002) with inter and intra-assay coefficient of variation as 3.7 % and 3.2 % respectively; 4) Antioxidant assay kit (Catalogue No. 709001) having inter and intra-assay coefficient of variation of 3 % and 3.4 % respectively. Statistical analysis was carried out with software package SPSS 20. A Shapiro–Wilk's test ($p > 0.05$) and a visual inspection of their histograms were used to check for normality of data (Shapiro and Wilk 1965; Razali and Wah 2011). Differences between mean values of three

groups (NW, OW and OB females) were evaluated by One-way ANOVA followed by Post-hoc Tukey test. Where the number of groups was two, the student t test (normally distributed data) and Mann–Whitney U-test (non-normally distributed data) were applied. Pearson's and Spearman's rank-order correlation test were employed to find association of data for normally and non-normally distributed data respectively. Difference was significant if p value was less than 0.05 at a 95 % confidence interval (CI).

Results:

Overall subject characteristics of NW, OW and OB groups have been given in Table 1. All obesity indicators (BMI, WC, WHR, BAI and % total body fat) were significantly higher in OB females ($p < 0.05$).

There was a statistically significant difference in 8-OH-2dG (pg/ml) between groups [$(F(2,103) = 4.91$, $p = 0.009$] (Table 2). A Tukey post hoc test revealed significantly higher 8-OH-2dG (pg/ml) in OB compared to NW (800.63 ± 6.19 vs 780.22 ± 3.34; p value = 0.007). Next, we explored the effects of WC and WHR (cut-off points ≥ 88 cm and ≥ 0.85 respectively) (Han et al. 1995; WHO 2000) on OS biomarkers. Interestingly, study participants with higher WC had significantly raised 8-OH-2dG (pg/ml) in contrast to participants with lower WC (812.62 ± 8.86 vs 0.75 ± 0.05; p value ≤ 0.001) (Table 3). TBARS, SOD and total antioxidant capacity did not differ significantly between the two groups. None of the OS biomarkers between two groups created based on WHR cut-off points differ significantly from each other (Table 4).

A significant positive association of 8-OH-2dG with BMI ($r = 0.286$, $p = 0.004$), WC ($r = 0.28$, $p = 0.005$), BAI ($r = 0.26$, $p = 0.008$), and % body fat ($r = 0.27$, $p = 0.006$) was revealed with Spearman rank correlation (Table 5).

Discussion:

All obesity indicators, such as BMI, WC, HC, WHR, BAI, % total body fat were significantly increased in obese females (Table 1). 8-OH-2dG (DNA oxidation products) were significantly higher in OB females; a finding in agreement with published literature (Hakkak et al. 2014; Zhang et al. 2011). The mechanism underpinning this raised DNA oxidative damage might be increased free radical formation (Vincent et al. 2001) or inadequate antioxidant defences or state of chronic inflammation which coexists in obesity (Monteiro and Azevedo 2010). In our study, SOD and serum total antioxidant capacity didn't differ significantly between three groups. Our results agree to Brown et al. (2009) who found insignificant difference in total antioxidant status, SOD and reduced glutathione among NW, OW, and OB adults. However, our results are contrary to studies in which antioxidant enzymes such as SOD were raised in association with obesity (Chrysohoou et al. 2007; Sfar et al. 2013; Erdeve et al. 2004). Koboyasi et al. (2010) and Nakao et al. (2000) reported increased SOD activity/levels in obese mice. Same findings were reported by Vincent et al. (2001) in obese rats compared to lean controls. Contrarily, Olusi (2002) and Ozata et al. (2002) reported significantly lower SOD activity in OB subjects compared to control. These inconsistencies in the results might be related to the extent/period of obesity (Brown et al. 2009). Antioxidant stimulation may occur in initial stages of obesity; followed by their normal levels initially and/or depleted levels in chronic and long-term obesity. The evidence that antioxidant enzymes may be stimulated during the development stages of obesity was given by Dobrian et al. (2000) who reported increased SOD activity, 10 weeks after diet-induced obesity in rats.

Our study showed statistically significant positive correlation of 8-OH-2dG with BMI, WC, BAI, % body fat (Table 5); a finding in line with previous researches (Brown et al. 2009; Keaney et al. 2003). The participants' mean ages in these studies were 60 years, and almost all were suffering from diabetes mellitus, myocardial infarction, hypertension etc. whereas participants in our study were young and free from any disease.

If the increase in oxidant and antioxidant levels is proportionate, oxidant-antioxidant ratio is not disturbed. However, our results show that there was increased DNA oxidative damage in OB group without a concomitant increase in serum total antioxidant capacity, even at adolescence. Therefore, due to this disproportionate increase, oxidant-antioxidant imbalance may occur and oxidative insult to the cells may occur.

In the end, we conclude that there may be significantly increased DNA damage in obese females in late adolescence, despite having normal blood glucose, blood pressure and sufficient physical activity. Furthermore, it can be linked to the amount of adipose tissue as depicted by strong positive association between DNA damage and WC, BAI, % body fat. Steps should be taken at national levels to encourage physical activity, dietary control and weight reduction programs at early ages to avoid future weight gain, fat accumulation and oxidative stress. Research may be carried out in future to find actual cause of DNA damage (e.g. inflammatory cytokines) in obese adolescents.

Limitations: All our subjects were females (due to strict gender segregation observed in the kingdom), belonging to the same university. Hence, our findings cannot be generalized. Subjects were recruited by convenience sampling.

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References:

BERGMAN RN, STEFANOVSKI D, BUCHANAN TA, SUMNER AE, REYNOLDS JC, SEBRING NG, XIANG AH, WATANABE RM: A better index of Body Adiposity. *Obesity (Silver Spring)* **19**: 1083-1089, 2011.

BROWN LA, KERR CJ, WHITING P, FINER N, MCENENY J, ASHTON T: Oxidant Stress in Healthy Normal-weight, Overweight, and Obese Individuals. *Obesity* 17: 460-466, 2009.

CHOBANIAN AV, BAKRIS GL, BLACK HR, CUSHMAN WC, GREEN LA, IZZO JL JR, JONES DW, MATERSON BJ, OPARIL S, WRIGHT JT JR, ROCCELLA EJ; NATIONAL HEART, LUNG, AND BLOOD INSTITUTE JOINT NATIONAL COMMITTEE ON PREVENTION, DETECTION, EVALUATION, AND TREATMENT OF HIGH BLOOD PRESSURE; NATIONAL HIGH BLOOD PRESSURE EDUCATION PROGRAM COORDINATING COMMITTEE: The Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure: the JNC 7 report. *JAMA* **289**: 2560-2572, 2003.

CHRYSOHOOU C, PANAGIOTAKOS DB, PITSAVOS C, SKOUMAS J, ECONOMOU M, PAPADIMITRIOU L, STEFANADIS C: The association between pre-hypertension status and oxidative stress markers related to atherosclerotic disease: the ATTICA study. *Atherosclerosis* **192**: 169-176, 2007.

CRAIG CL, MARSHALL AL, SJOSTROM M, BAUMAN AE, BOOTH ML, AINSWORTH BE, PRATT M, EKELUND U, YNGVE A, SALLIS JF, OJA P: International physical activity questionnaire: 12-country reliability and validity. *Med Sci Sports Exerc* 35: 1381–1395, 2003.

DOBRIAN AD, DAVIES MJ, PREWITT RL, LAUTERIO TJ: Development of hypertension in a rat model of diet-induced obesity. *Hypertension* **35**: 1009–1015, 2000.

DURNIN JV, WOMERSLEY J: Body fat assessed from the total body density and its estimation from skinfold thickness: measurements on 481 men and women aged from 16 to 72 years. *Br J Nutr* **32**: 77-97, 1974.

ERDEVE O, SIKLAR Z, KOCATURK PA, DALLAR Y, KAVAS GO: Antioxidant superoxide dismutase activity in obese children. *Biol Trace Elem Res* **98**: 219–228, 2004.

Expert Committee on the Diagnosis and Classification of Diabetes Mellitus: Report of the Expert Committee on the Diagnosis and Classification of Diabetes Mellitus. *Diabetes Care* **26**: S5-20, 2003.

FURUKAWA S, FUJITA T, SHIMABUKURO M, IWAKI M, YAMADA Y, NAKAJIMA Y, NAKAYAMA O, MAKISHIMA M, MATSUDA M, SHIMOMURA I: Increased oxidative stress in obesity and its impact on metabolic syndrome. *J Clin Invest* **114**: 1752-1761. 2004.

Guidelines for data processing and analysis of the International Physical Activity Questionnaire(IPAQ)—Short and Long Forms. Downloaded from http://www.ipaq.ki.se/dloads/. Published November 2005).

HAKKAK R, KOROURIAN S, PAVLIV O, EVANS T, MELNYK S: Effects of obesity on development of oxidative stress and DNA damages in liver of the obese Zucker rat (643.1). *The FASEB J* **28**: 2014.

HALLIWELL B. Biochemistry of oxidative stress. *Biochem Soc Trans* **35**: 1147–1150, 2007.

HAN TS, VAN LEER EM, SEIDELL JC, LEAN ME: Waist circumference action levels in the identification of cardiovascular risk factors: prevalence study in a random sample. *BMJ* **311**: 1401–1405, 1995.

KAVOURAS SA, PANAGIOTAKOS DB, PITSAVOS C, CHRYSOHOOU C, ANASTASIOU CA, LENTZAS Y, STEFANADIS C: Physical activity, obesity status, and glycemic control: the ATTICA study. *Med Sci Sports Exerc* **39**: 606-611, 2007.

KEANEY J, LARSON M, VASAN R, WILSON PW, LIPINSKA I, COREY D, MASSARO JM, SUTHERLAND P, VITA JA, BENJAMIN EJ; FRAMINGHAM STUDY: Obesity and systemic oxidative stress: clinical correlates of oxidative stress in the Framingham Study. *Arterioscler Thromb Vasc Biol* **23**: 434–439, 2003.

KOBOYASI R, AKAMINE EH, DAVEL AP, RODRIGUES MA, CARVALHO CR, ROSSONI LV: Oxidative stress and inflammatory mediators contribute to endothelial dysfunction in high-fat diet-induced obesity in mice. *J Hypertens* **28**: 2111–2119, 2010.

MONTEIRO R, AZEVEDO I: Chronic Inflammation in Obesity and the Metabolic Syndrome. *Mediators Inflamm* **2010**: 289645, 2010.

NAKAO C, OOKAWARA T, SATO Y, KIZAKI T, IMAZEKI N, MATSUBARA O, HAGA S, SUZUKI K, TANIGUCHI N, OHNO H: Extracellular superoxide dismutase in tissues from obese (ob/ob) mice. *Free Radical Res* **33**: 229–241, 2000.

OLUSI SO: Obesity is an independent risk factor for plasma lipid peroxidation and depletion of erythrocyte cytoprotectic enzymes in humans. *Int J Obes Relat Metab Disord* **26**: 1159–1164, 2002.

OZATA M, MERGEN M, OKTENLI C, AYDIN A, SANISOGLU SY, BOLU E, YILMAZ MI, SAYAL A, ISIMER A, OZDEMIR IC: Increased oxidative stress and hypozincemia in male obesity. *Clin Biochem* **35**: 627–631, 2002.

RAZALI N, WAH YB: Power comparisons of Shapiro-Wilk, Kolmogorov–Smirnov, Lilliefors and Anderson–Darling tests. *J Stat Model Anal* **2**: 21–33, 2011.

SFAR S, BOUSSOFFARA R, SFAR MT, KERKENI A: Antioxidant enzymes activities in obese Tunisian children. *Nutr J* **12**: 18, 2013.

SHAPIRO SS, WILK MB. An analysis of variance test for normality (complete samples). *Biometrika* **52**: 591–5611, 1965.

SIRI WE: Body composition from fluid space and density. In: *Techniques for measuring body composition*. J Brozek, A Hanschel (eds), National Academy of Science, Washington DC, 1961, pp. 223-244.

UNVER PB, KARABULUT AB, SERTKAYA AC, KIRAN TR, YAGMUR J: The Relationship between Obesity and Oxidative Stress and Cardiac Markers. *Med-Science* **4**: 2087-2097, 2015.

VINCENT H, POWERS S, DIRKS A, SCARPACE PJ: Mechanism for obesity-induced increase in myocardial lipid peroxidation. *Int J Obes* **25**: 378–388, 2001.

WORLD HEALTH ORGANIZATION: Obesity: Preventing and managing the global epidemic. Report of a WHO Consultation (TRS 894). 2000.

WORLD HEALTH ORGANIZATION: Appropriate body-mass index for Asian populations and its implications for policy and intervention strategies. *Lancet* **363**: 175–163, 2004.

ZHANG H, XIE C, SPENCER HJ, ZUO C, HIGUCHI M, RANGANATHAN G, KERN PA, CHOU MW, HUANG Q, SZCZESNY B, MITRA S, WATSON AJ, MARGISON GP, FAN CY: Obesity and hepatosteatosis in mice with enhanced oxidative DNA damage processing in mitochondria. *Am J Pathol* **178**: 1715–1727, 2011.

Table 1: Subject characteristics

Variables	Groups			*p* value		
	NW (n=52)	OW (n=24)	OB (n=30)	NW vs OW	OW vs OB	NW vs OB
Age (years)	19.17±0.07	19.17±0.10	19.20±0.10	0.99	0.97	0.97
Weight (kg)	50.07±0.83	62.24±1.33	82.02±2.28	< .001	< .001	< .001
BMI (kg/m^2)	20.14±0.24	24.98±0.26	32.54±0.83	< .001	< .001	< .001
WC (cm)	67.08±0.61	74.99±1.48	91.20±2.57	< .001	< .001	< .001
WHR	0.72±0.01	0.73±0.02	0.79±0.02	0.93	< .001	< .001
BD	1.04±0.00	1.02±0.00	1.01±0.00	< .001	< .001	< .001
BAI	29.04±0.44	35.43±1.32	40.56±1.17	< .001	< .001	< .001
Body Fat %	28±0.79	33±0.58	38±0.58	< .001	< .001	< .001
FBS (mg/dl)	91.23±1.68	94.04±1.47	95.56±2.50	0.57	0.88	0.26
Systolic BP (mmHg)	114.75±1.78	116.04±2.19	123.84±2.79	0.91	0.08	0.01
Diastolic BP (mmHg)	71.19±1.16	71.04±1.36	75.60±1.61	0.10	0.12	0.06
Total Physical activity (MET- minutes/week)	1543.46±189.9	1585.46±414.4	1718.24±494.6	0.86	0.69	0.30
Biceps skinfold (mm)	13.2±0.75	13.7±0.68	17.4±1.5	0.93	0.06	0.01
Triceps skinfold (mm)	15.84±0.80	19.95±1.45	29.42±1.74	0.05	< .001	< .001
Subscapular skinfold (mm)	17.1±0.08	24.1±0.12	34.1±0.08	< .001	< .001	< .001
Suprailiac skinfold (mm)	13.3±1.35	20.7±1.39	33.7±1.60	< .001	< .001	< .001

Values expressed as the Mean ± S.E.M.

Results were compared using the One-way ANOVA test followed by the Post HOC Tukey test.

P < 0.05 was considered significant.

NW, Normal-weight; OW, Overweight, OB, Obese; BMI, body mass index; WC; Waist circumference; WHR, waist-hip-ratio; BD, Body density; BAI, Body adiposity index; FBS, Fasting blood sugar; BP; Blood Pressure; MET, Metabolic equivalent

Table 2: Mean values of oxidative stress biomarkers in all groups based on BMI cut-off points

Variables	NW (n=52)	OW (n=24)	OB (n=30)	*p* value		
				NW vs OW	OW vs OB	NW vs OB
TBARS (µM)	3.61±0.22	3.62±0.32	3.76±0.42	1.00	0.932	0.951
8-OH-2dG (pg/ml)	780.22±3.34	790.01±6.22	800.63±6.19	0.351	0.368	0.007
SOD (U/ml)	0.78±0.07	0.77±0.08	0.64±0.06	0.997	0.54	0.377
Total antioxidant capacity (mM)	4.62±0.30	4.87±0.44	5.50±0.50	0.899	0.585	0.228

Values are expressed as the Mean ± S.E.M.

Results were compared using the One-way ANOVA test followed by the Post HOC Tukey test.

P < 0.05 was considered significant.

NW, Normal-weight; OW, Overweight, OB, Obese; TBARS, Thiobarbituric acid reactive substances; 8-OH-2dG, 8-hydroxy-2-deoxyguanosine; SOD, superoxide dismuta

Table 3: Mean values of oxidative stress biomarkers based on Waist Circumference cut-off points for study subjects

Variables	WC ≥ 0.88 (n=15)	WC < 0.88 (n=86)	*p* value
TBARS (μM)	3.24±0.51	3.67±0.19	0.39
8-OH-2dG (pg/ml)	812.62 ± 8.86	784.26±2.94	< .001
SOD (U/ml)	0.63 ± 0.09	0.75 ± 0.05	0.28
Total antioxidant capacity (mM)	5.33 ± 0.78	4.85 ± 0.25	0.62

Values expressed as the Mean ± S.E.M.

Results were compared using the student t test for TBARS (normally distributed data) and Mann–Whitney U-test for 8-OH-2dG, SOD and Antioxidants (normally distributed data).

P ≤ 0.05 was considered significant.

WC, Waist circumference; NW, Normal-weight; OW, Overweight, OB, Obese; TBARS, Thiobarbituric acid reactive substances; 8-OH-2dG, 8-hydroxy-2-deoxyguanosine; SOD, superoxide dismutase

Table 4: Mean values of oxidative stress biomarkers based on Waist-Hip-Ratio cut-off points for study subjects

Variables	WHR ≥ 0.85 (n=6)	WHR < 0.85 (n=95)	*p* value
TBARS (μM)	3.87 ± 0.88	3.58 ± 0.18	0.70
8-OH-2dG (pg/ml)	794.76 ± 7.15	788.08 ± 3.14	0.37
SOD (U/ml)	0.63 ± 0.12	0.74 ± 0.05	0.63
Total antioxidant capacity (mM)	6.72 ± 1.12	4.81 ± 0.24	0.11

Values expressed as the Mean ± S.E.M.

Results were compared using the student t test for TBARS (normally distributed data) and Mann–Whitney U-test for 8-OH-2dG, SOD and Antioxidants (normally distributed data).

P ≤ 0.05 was considered significant.

WHR, Waist-Hip-Ratio; NW, Normal-weight; OW, Overweight, OB, Obese; TBARS, Thiobarbituric acid reactive substances; 8-OH-2dG, 8-hydroxy-2-deoxyguanosine; SOD, superoxide dismutase

Table 5: Correlation coefficients between oxidative stress biomarkers and anthropometric measurements in all study participants

Variables		TBARS (μM)	8-OH-2dG (pg/ml)	SOD (U/ml)	Total antioxidant capacity (mM)
BMI (kg/m^2)	Correlation coefficient	-0.072	0.286	-0.050	0.175
	Significance	0.472	0.004*	0.623	0.079
WC (cm)	Correlation coefficient	-0.109	0.280	-0.096	0.091
	Significance	0.279	0.005*	0.340	0.364
WHR	Correlation coefficient	-0.143	0.104	-0.171	-0.102
	Significance	0.155	0.300	0.089	0.843
BAI	Correlation coefficient	-0.005	0.263	-0.092	0.219
	Significance	0.958	0.008*	0.361	0.128
BD	Correlation coefficient	0.058	-0.273	0.127	-0.163
	Significance	0.561	0.006*	0.210	0.102
% Body Fat	Correlation coefficient	-0.059	0.273	-0.127	0.163
	Significance	0.556	0.006*	0.210	0.102

Results were analyzed using the Pearson's correlation for Normally distributed data (TBARS) and Spearman rank correlation for Non-normally distributed data (8-OH-2dG, SOD, Total antioxidant capacity). Correlation is significant at the 0.01 level.

TBARS, Thiobarbituric acid reactive substances; 8-OH-2dG, 8-hydroxy-2-deoxyguanosine; SOD, superoxide dismutase**;** BMI, body mass index; WC; Waist circumference; WHR, waist-hip-ratio; BD, Body density; BAI, Body adiposity index.