

Association of Anthropometric Measurements With Oxidant-Antioxidant Status Among Young Saudi Females

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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 ; $p=0.007$), 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

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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 mm Hg, diastolic BP ≥ 90 mm Hg) (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 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).

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

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).

$$\% \text{ Body Fat} = \left(\frac{495}{\text{Body Density}} \right) - 450$$

Blood samples obtained after an overnight fast were clotted, centrifuged and frozen at -80 °C. 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's test. Where the number of groups was two, the Student's 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's *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; $p=0.007$).

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	<0.001	<0.001	<0.001
BMI (kg/m ²)	20.14 ± 0.24	24.98 ± 0.26	32.54 ± 0.83	<0.001	<0.001	<0.001
WC (cm)	67.08 ± 0.61	74.99 ± 1.48	91.20 ± 2.57	<0.001	<0.001	<0.001
WHR	0.72 ± 0.01	0.73 ± 0.02	0.79 ± 0.02	0.93	<0.001	<0.001
BD	1.04 ± 0.00	1.02 ± 0.00	1.01 ± 0.00	<0.001	<0.001	<0.001
BAI	29.04 ± 0.44	35.43 ± 1.32	40.56 ± 1.17	<0.001	<0.001	<0.001
Body Fat %	28 ± 0.79	33 ± 0.58	38 ± 0.58	<0.001	<0.001	<0.001
FBS (mg/dl)	91.23 ± 1.68	94.04 ± 1.47	95.56 ± 2.50	0.57	0.88	0.26
Systolic BP (mm Hg)	114.75 ± 1.78	116.04 ± 2.19	123.84 ± 2.79	0.91	0.08	0.01
Diastolic BP (mm Hg)	71.19 ± 1.16	71.04 ± 1.36	75.60 ± 1.61	0.10	0.12	0.06
Total physical activity (MET- minutes/week)	1,543.46 ± 189.9	1,585.46 ± 414.4	1,718.24 ± 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	<0.001	<0.001
Subscapular skinfold (mm)	17.1 ± 0.08	24.1 ± 0.12	34.1 ± 0.08	<0.001	<0.001	<0.001
Suprailiac skinfold (mm)	13.3 ± 1.35	20.7 ± 1.39	33.7 ± 1.60	<0.001	<0.001	<0.001

Values expressed as the Mean ± S.E.M. Results were compared using the One-way ANOVA test followed by the *post hoc* Tukey's 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	1.000	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 dismutase.

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 ; $p \leq 0.001$) (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 differed 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).

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	< 0.001
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 \pm S.E.M. Results were compared using the Student's t-test for TBARS (normally distributed data) and Mann-Whitney U-test for 8-OH-2dG, SOD and Antioxidants (normally distributed data). $p \leq 0.05$ 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 \pm S.E.M. Results were compared using the Student's t-test for TBARS (normally distributed data) and Mann-Whitney U-test for 8-OH-2dG, SOD and Antioxidants (normally distributed data). $p \leq 0.05$ 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.

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 defenses or state of chronic inflammation which coexists in obesity (Monteiro and Azevedo 2010). In our study, SOD and serum total antioxidant capacity did not 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.

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

<i>Variables</i>		TBARS (μM)	8-OH-2dG (pg/ml)	SOD (U/ml)	Total antioxidant capacity (mM)
<i>BMI (kg/m²)</i>	Correlation coefficient	-0.072	0.286	-0.050	0.175
	Significance	0.472	0.004*	0.623	0.079
<i>WC (cm)</i>	Correlation coefficient	-0.109	0.280	-0.096	0.091
	Significance	0.279	0.005*	0.340	0.364
<i>WHR</i>	Correlation coefficient	-0.143	0.104	-0.171	-0.102
	Significance	0.155	0.300	0.089	0.843
<i>BAI</i>	Correlation coefficient	-0.005	0.263	-0.092	0.219
	Significance	0.958	0.008*	0.361	0.128
<i>BD</i>	Correlation coefficient	0.058	-0.273	0.127	-0.163
	Significance	0.561	0.006*	0.210	0.102
<i>% Body Fat</i>	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.

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.

Conflict of Interest

There is no conflict of interest.

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