Research Communication

Oxidative stress and inflammation, two features associated with a high percentage body fat, and that may lead to diabetes mellitus and metabolic syndrome

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Abbreviations: BMI, body mass index; WC, waist circumference; MONW, metabolically obese normal weight; ROS, reactive oxygen species; PAB, pro-oxidant-antioxidant balance; SOD1, superoxide dismutase 1; MASHAD Study, Mashhad stroke and heart association disorder study

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Abstract

Obesity is an important feature of the metabolic syndrome and is associated with an increased risk of type 2 diabetes mellitus, cardiovascular disease, and some cancers. The aim of this study was to determine the relationship between body fat percentage and an imbalance of the prooxidant/antioxidant balance (PAB), serum superoxide dismutase (SOD1) and inflammation (serum hs-CRP) and increase risk of metabolic syndrome and diabetes mellitus. In this study, 9154 individuals were recruited as part of the Mashhad Stroke and Heart Association Disorder (MASHAD) study. Subjects were categorized into two groups according to body fat percentage as defined >25% in male and > 30% in female, according to gender. Biochemical factors, including serum PAB, SOD1, and hs-CRP were measured in all subjects. SPSS version 18 was used for statistical analyses for all. GraphPad Prism 6 for figures was used. Of total number of subjects (9154), 6748 (73.7%) were found to have a high body fat (BF) percentage. Serum hs-CRP and PAB were significantly higher in individuals with a high BF percentage ($P < 0.05$) but SOD1 was not significantly different between the two groups ($P > 0.05$). BF percentage, serum PAB and serum hs-CRP were significantly higher in individuals with metabolic syndrome and diabetes versus those without metabolic syndrome and diabetes mellitus ($P < 0.05$), however serum SOD1 was significantly lower in individuals with metabolic syndrome ($P < 0.005$). Oxidative stress and inflammation are two factors that may link the presence of high BF percentage with the development of metabolic syndrome, diabetes, and cardiovascular disease. © 2018 BioFactors, 00(00):1–8, 2018

Keywords: *oxidative stress; metabolic syndrome; SOD1*

1. Introduction

Obesity is defined as excess body fat, which increases the risk of several metabolic abnormalities. Obesity is one of the most common features of metabolic syndrome and is associated with an increased risk of type 2 diabetes, cardiovascular disease, and some cancers [1]. Although the WHO and many countries define obesity based on body mass index (BMI) and waist circumference (WC), these measures have some limitations [2–4]. There are some people who are not obese based on the BMI definition of obesity (BMI >30 Kg/m^2), have a normal weight, but still have metabolic abnormalities and they have recently been categorized into a subgroup called metabolically obese normal weight individuals (MONW). These people have impaired glucose tolerance and are insulin resistant and prone to type 2 diabetes and cardiovascular disease [5]. Therefore, studies have tended to add body fat percentage factor to BMI and WC and consider all of the above mentioned factors for assessing for the presence of obesity [6,7].

Obesity leads to various changes in inflammatory processes that increase reactive oxygen species (ROS) production and cause oxidative stress. Obesity leads to a disruption of the Krebs cycle and the mitochondrial respiratory chain, which causes an impaired mitochondrial function and higher ROS production. Increased ROS production by the respiratory chain leads to oxidative stress, which may intensify inflammatory processes in obesity [8]. Another possible reason for increased inflammation and oxidative stress in obese people is the production of adipocytokines. Body fat adipocytes are the most important source of adipocytokines such as adiponectin that may play an important role in the metabolism of lipids and glucose, inflammation and oxidative stress. Altered adiponectin can also lead to insulin resistance, type 2 diabetes and cardiovascular diseases [9].

Metabolic syndrome comprises a cluster of abnormalities that include: obesity, insulin resistance, high blood pressure, glucose intolerance, and dyslipidemia (high triglyceride and low HDL) [10]. Obesity and insulin resistance are also common important risks for type 2 diabetes. Studies show that inflammation caused by the loss of insulin protection (insulin resistance) in obese people is one of the main causes of metabolic syndrome and diabetes [11,12]. Interference of insulin signal pathway transduction with inflammatory mechanisms can be a major reason for obesity to be a pro-inflammatory state. Oxidative stress in accumulated fat is also one of the important mechanisms of obesity in the progression of metabolic syndrome [13,14]. In the diabetes mellitus, oxidative stress impairs glucose uptake in muscle and fat [19,20] and decreases insulin secretion from pancreatic β cells. Insulin resistance is also a major cause of oxidative stress that main lead to metabolic syndrome, diabetes, and cardiovascular disease [15–17].

The primary aim of the current study was to determine the relationship between body fat percentage and imbalance of oxidant/antioxidant defense (Pro-oxidant-antioxidant balance [PAB] and superoxide dismutase [SOD1]) and inflammation (hs-CRP). We also wanted to determine whether individuals with a high BF percentage, imbalance of oxidant/antioxidant defense and inflammation are more susceptible to metabolic syndrome and diabetes mellitus.

2. Methods

In this study, 9154 subjects, 3639 male, and 5515 female, without a CVD history, were recruited as a part of MASHAD study using a cluster randomized sampling approach in 2007–2008, as previously described. [18] The MASHAD study is a 10-years

cohort study, which aims to investigate the impact of several genetic, nutritional, psychosocial, and environmental risk factors on the incidence of cardiovascular events among a total urban population living in NorthEastern Iran.

2.1. Data collection and measurements

For each subjects participating in the study, height (cm), weight (kg), body mass index (kg/m^2) and waist circumference were evaluated. SBP and DBP by sphygmomanometer twice on the left arm when the individuals remained seated at rest for 15 min [19]. Biochemical parameters, consisting total cholesterol (TC), high-density lipoprotein cholesterol (HDL-C), low-density lipoprotein cholesterol (LDL-C) and triglyceride (TG), C-reactive protein (CRP) and fasting blood glucose (FBG) were determined as described previously [20]. BF percentage was determined by using a calibrated instrument for bioelectric impedance analysis (BIA) based on standardized protocol using a RJ Systems BIA-101A and gender-specific equations. A high BF percentage is defined >25% in men and > 30% in women, according to gender [6].

2.2. Serum prooxidant/antioxidant balance measurement

The serum PAB assay estimates the balance of oxidants and antioxidants simultaneously in a single test. The level of PAB was measured in serum samples using a modified PAB assay as previously described. [21]. In summary, the standard solutions were provided by mixing varying portions (0–100%) of 250-μM hydrogen peroxide (H_2O_2) with 3 mM uric acid (in 10 mM NaOH). Sixty milligrams of TMB powder was dissolved in 10 mL DMSO. In order to provide 3,3′,5,5′-Tetramethylbenzidine (TMB) cation, 400 μL of the TMB/DMSO was added to 20 mL of acetate buffer (0.05 M buffer, pH 4.5). Then, 70 μL of fresh chloramine T (100 mM) solution was added to this 20 mL. This was mixed and incubated for 2 h at room temperature in the dark. Next, 25 U of peroxidase enzyme solution was added to 20 mL of TMB cation solution, distributed in 1 mL and stored at −20 °C. To prepare the TMB solution, 200 μL of TMB/DMSO was added to 10 mL of acetate buffer (0.05 M buffer, pH 5.8) and the working solution was made by mixing 1 mL TMB cation with 10 mL of TMB solution. This was also incubated for 2 min at room temperature in the dark and immediately used. Ten microliters (10 μL) of each sample, standard or blank (distilled water) were mixed with 200 μL of working solution in every well of a 96 well plate, after which incubated in a dark place at 37 °C for 12 min. When the incubation was finished, 100 μL of 2 N hydrochloric acid (HCL) was added to each well, and the optical density (OD) was measured in an ELISA reader at 450 nm with a reference wavelength of 620 or 570 nm. A standard curve was drawn from the values relative to the standard samples. The serum PAB values are expressed in arbitrary HK unit, which is the percentage of hydrogen peroxide in the standard solution. The values of the unknown samples were then computed based on the values obtained from the above standard curve [21].

2.3. Serum superoxide dismutase assay

The assay buffer comprised Tris–cacodylic acid buffer and a pyrogallol solution. Tris–cacodylic acid buffer (0.05 M, pH 8.2) containing 0.001 M diethylenediamine penta acetic acid (DTPA) was made by adding Tris (0.05 M, containing 0.001 M DTPA) to cacodylic acid (0.05 M, containing 0.001 M DTPA) until pH 8.2 was achieved. Before use, the buffer was equilibrated in air for 1 h; to make the pyrogallol solution (0.2 mM) a stock 0.02 M (100) pyrogallol solution was made in water, flushed with nitrogen for 1 h to eliminate soluble oxygen, aliquoted (100 μl per aliquot), and stored frozen until use.

Each serum and control sample (20 μl/well) was added in duplicate wells. Immediately before measurement, pyrogallol stock solution (0.02 M) was diluted 1:100 with equilibrated assay buffer and added to the wells (180 μl/well) using a multichannel pipettor. The reactions were read in a plate reader at 405 nm in 5-min intervals during 1 h. The blocking of pyrogallol oxidation was graphically characterized for every dilution of SOD1. A level of SOD1 that inhibited the oxidation of pyrogallol by 50% (relative to the control) was defined as a unit of SOD1 activity under the conditions described.

2.4. Statistical analysis

SPSS version 18(SPSS Inc. Chicago, IL) was applied for all statistical analyses. The normality of the data was manifested using the Kolmogorov–Smirnov test. Descriptive statistics consisting mean, frequency, and standard deviation (SD) were determined for all variables and said as mean ± SD for variables with normally distribution or median ± IQR for not normally distributed parameters. Partial correlation and linear regression were used to investigate the relation between BF percentage, serum PAB, SOD1, and hs-CRP and metabolic syndrome and each other. Logistic regression analysis was used to appear the association of BF percentage, PAL, PAB, and SOD1 with metabolic syndrome and diabetes in high body FAT subjects. For drawing figures, we used GraphPad Prism 6.

3. Results

3.1. Prevalence of metabolic syndrome and diabetes according to BF percentage

Of the 9154 subjects, 6748 (73.7%) had a high BF percentage, 1632 (44.8%), and 5116 (92.8%) were male and female, respectively (Table 1). The prevalence of metabolic syndrome was 17.9% and 42.8% in the individuals with normal and high BF percentage, respectively. In the latter sub-group, it was present in 43.2% in women and 41.6% in men (Fig. 1). The prevalence of diabetes mellitus in the total population with high BF percentage was 15.9%; 15.2% and 18.1% in women and men, respectively (Fig. 1). There were significant differences between normal and high BF percentage in the prevalence of metabolic syndrome and diabetes mellitus.

TABLE 1 *Demographic and biochemical characteristics of subjects according to BF percentage* (n = *9154)*

		Normal BF% (n = *2406, 26.3%)*	*High BF%* (n = *6748, 73.7%)*	P-*value*
Sex	Male (*n* = 3639)	2007 (55.2%)	1632 (44.8%)	<0.001
	Female (*n* = 5515)	399 (7.2%)	5116 (92.8%)	
Age, y		46.36 ± 8.28	48.8 ± 8.49	<0001
Smoking habit	No (*n* = 6274)	1392 (22.2%)	4882 (77.8%)	<0.001
	Former (*n* = 882)	306 (34.7%)	576 (65.3%)	
	Current (*n* = 1995)	707 (35.4%)	1288 (64.6.2%)	
BMI, kg/m^2		23.13 ± 1.77	29.59 ± 1.03	<0.001
SBP, mm Hg		118.63 ± 15.94	129.67 ± 17.89	<0.001
DBP, mm Hg		77.19 ± 1.26	79.58 ± 11.05	<0.001
Waist circumference, cm		87.35 ± 8.01	98.8 ± 8.64	<0.001
Fasting Glucose, mg/dl		87.53 ± 36.37	94.58 ± 44.12	<0.001
Serum TC, mg/dl		183.37 ± 39.68	194.12 ± 40.01	<0.001
Fasting TG, mg/dl		106 (75–156)	126 (89–178)	<0.001
Serum HDL, mg/dl		41.26 ± 9.85	42.47 ± 9.83	<0.001
Serum LDL, mg/dl		113.1 ± 34.11	119.51 ± 34.85	<0.001

Data presented as Mean ± SD or inter quartile range. T-*test or Mann Whitney* U-*test has done for comparing two groups.*
High body fat percentage (High BF %):> 25 in men and > 30 in female.
BF%, body fat percentage; SBP, systolic blood pressure; DBP, diastolic blood pressure; TC, total cholesterol; TG, triacylglycerol; HDL-C,HDL cholesterol; hsCRP, high-sensitivity C-reactive protein.

3.2. General characteristic of subjects

Demographic and biochemical characteristic of subjects according to BF percentage are presented in Table 1. Mean age in the normal and high BF percentage subjects were 46.36 ± 8.28 and 48.8 ± 8.49 years, respectively (Table 1). In addition, there were no significant differences between two groups ($P > 0.05$). And there were not significant differences between two groups ($P > 0.05$). Among subjects who had high BF percentage, 19.1% were current smoker, 8.5% were former smoker and 72.4% were no smoker, and there were significant differences between smoking status and BF percentage (Table 1) ($P < 0.05$). In the high Body fat percentage subjects, BMI, waist circumference, SBP, DBP, glucose, total cholesterol and LDL, HDL, and TG were higher than normal BF percentage subjects (Table 1) ($P < 0.05$). The prevalence of metabolic syndrome and diabetes is shown in Fig. 1. The prevalence of metabolic syndrome in women with a high body fat was higher than for men ($P < 0.005$). Although, the prevalence of diabetes mellitus in men with high body fat was higher than for women but this was not significant ($P > 0.05$).

3.3. Anti-oxidant status and hs-CRP in subjects according to BF percentage, metabolic syndrome, and diabetes

We found that serum hs-CRP was significantly higher in the group with elevated BF percentage ($P < 0.05$) (Table 2). Serum PAB was significantly higher in the high BF percentage subjects ($P < 0.05$); although the mean SOD1 (superoxide dismutase) was higher in the high BF percentage subjects, this was not significant between two groups ($P > 0.05$) (Table2).

The BF percentage, hs-CRP, PAB, and SOD1 were assessed according to the presence of metabolic syndrome and diabetes mellitus and are shown in Table 3. BF percentage, serum PAB, and serum hs-CRP were significantly higher in individuals with metabolic syndrome compared to those without ($P < 0.05$), but serum SOD1 was significantly lower in metabolic syndrome subjects ($P < 0.005$). Serum PAB, hs-CRP, and BF percentage were significantly higher in diabetic subjects (Table 3).

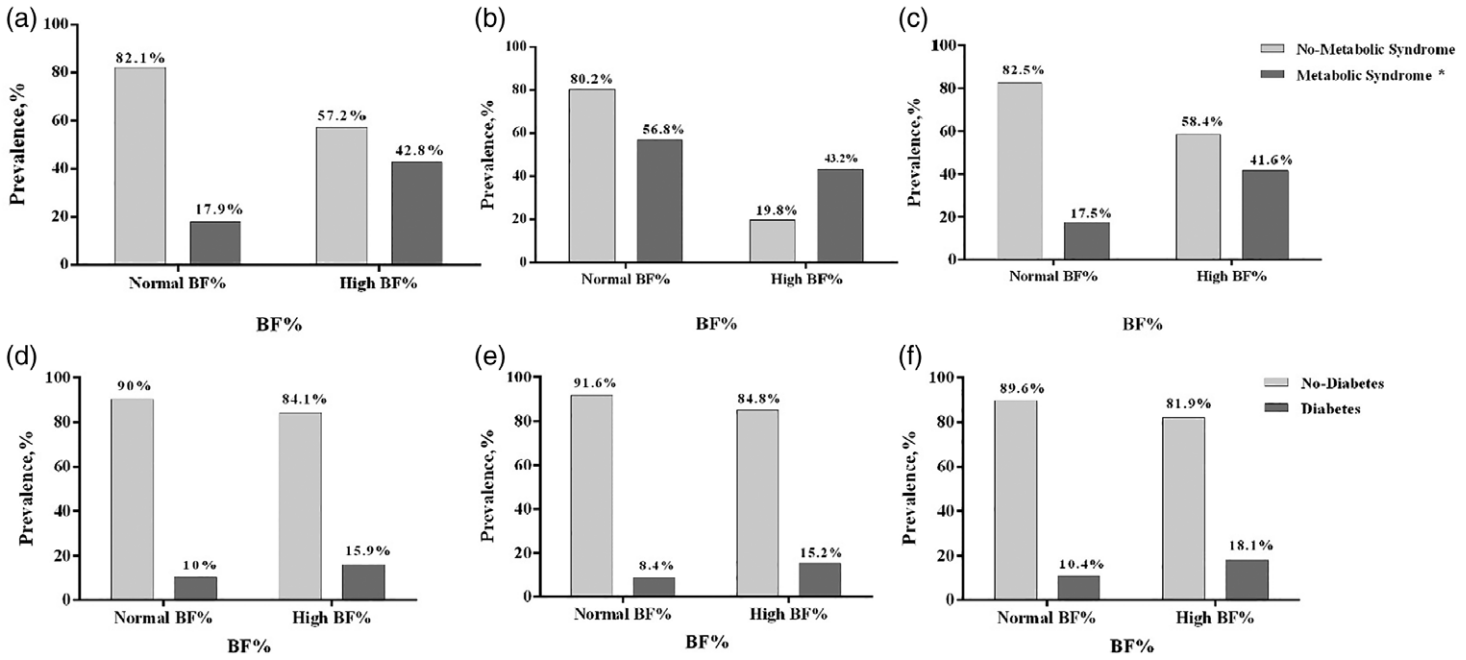

FIG 1 *Prevalence of Metabolic syndrome and diabetes according to BF percentage. (A) Total subjects; (B) Female; (C) Male.* *P *< 0.001.*

3.4. Association between BF percentage, PAB, SOD1, hs-CRP, and metabolic syndrome components and diabetes

We also sought to assess the association between BF percentage, serum PAB, SOD1, and hs-CRP as continuous variables and components of metabolic syndrome and diabetes. After controlling for age, sex, smoking, and BF percentage was positively correlated with fasting glucose, triglyceride, waist circumference and blood pressure and negative correlation with serum HDL. PAB was positively correlated with fasting glucose, and serum triglyceride, and negative correlated with serum HDL. Serum SOD1 was positive correlated with serum HDL and negative correlation with fasted serum triglycerides. Serum Hs-CRP was positively correlated with fasting glucose and waist circumference. Using univariate and multivariate analysis, we showed that there was an association between serum PAB, hs-CRP, and BF percentage.

TABLE 2 ***Serum PAB, SOD, and hs-CRP according to BF percentage***

	Normal BF% (2406, 26.3%)	*High BF% (6748, 73.7%)*	P-*value*
Serum PAB	55.24 ± 44.59	67.19 ± 48.73	<0.001
Serum SOD1, U/ml	2.18 ± 1.27	2.11 ± 1.27	0.41
Serum hs-CRP, mg/l	1.28 (0.79–2.2)	1.81 (1.08–3.8)	<0.001

Data presented as Mean ± SD. T-*test has done for comparing two groups.*
PAL, Physical Activity Level; PAB, Prooxidant-Antioxidant Balance; SOD1, Superoxide Dismutase.

3.5. Risk of metabolic syndrome and diabetes associated with antioxidant defense and hs-CRP in subjects with high BF percentage

Lastly, we determined the risk of metabolic syndrome and diabetes mellitus that were associated with measures of antioxidant defense and hs-CRP in subjects with high BF percentage. Table 4 shows the unadjusted and adjusting odds ratio of the association between serums PAB, SOD1 and hs-CRP with metabolic syndrome. Before adjusting for age, sex and smoking habit, the odds ratio for quartile 4 of PAB (> 94) was 1.17 (95% CI, 1.037–1.32), quartile 4 of SOD1 (0.56) was 0.829 (95% CI, 0.727–0.946), quartile 4 of hs-CRP (< 3.525) was 1.429 (95% CI, 1.286–1.587). After adjustment for age, sex, smoking and each other's, the results confirmed except for serum PAB.

Table 4 shows the unadjusted and adjusted odds ratio of the association between PAB, SOD1, and hs-CRP with diabetes mellitus. Before adjustment the odds ratio for quartile four of PAB (> 94) was 1.313(95% CI, 1.123–1.535), quartile four of SOD1 (0.56) was 1.086(95% CI, 0.913–1.293), quartile four of hs-CRP (< 3.525) was 1.8(95% CI, 1.576–2.056). After adjustment for age, sex, smoking, and both, the results were similar.

Our data suggest an association between serum hs-CRP, PAB and SOD1 with the presence of metabolic syndrome, but after adjustment for other factors using multivariate analysis, only serum hs-CRP and SOD1 remained as independent risk

TABLE 3 ***Serum BF percentage, PAB, SOD1, and hs-CRP according to the presence of metabolic syndrome and diabetes***

		BF%	*PAB*	*SOD1*	*hs-CRP*
Metabolic syndrome	No	32.95 ± 7.52	62.57 ± 41.43	2.31 ± 1.28	1.28 (0.79–3.12)
	Yes	37.5 ± 9.09	68.65 ± 51.31	1.99 ± 1.25	2.3 (1.729–4.12)
P-value		< 0.001	< 0.001	< 0.001	< 0.001
Diabetes	No	33.85 ± 10.4	63.63 ± 48.89	2.2 ± 1.27	1.54 (0.95–3.23)
	Yes	35.9 ± 9.7	69.88 ± 43.61	2.08 ± 1. 7	2.33 (1.3–4.88)
P-value		< 0.001	< 0.001	0.1	< 0.001

Data presented as mean (SD) or inter quartile range. T-*test or Mann–Whitney* U-*test has done for comparing two groups.*
PAB, Prooxidant-Antioxidant Balance; SOD1, Superoxide Dismutase; hs-CRP, high sensitive C reactive protein.

TABLE 4 ***Unadjusted and Multivariate-Adjusted Odds Ratios of the Metabolic syndrome and diabetes according to PAB, SOD1, and hs-CRP in subjects with high body fat percentage***

	Unadjusted	P-*value*	*Adjusted by age, sex, smoking, and each other's*	P-*value*
Metabolic syndrome	Reference, Q1,2,3		Reference, Q1,2,3	
PAB, Q4	1.17 (1.037–1.32)	0.011	1.046 (0.906–1.207)	0.53
SOD1, Q4	0.829 (0.727–0.946)	0.005	0.818 (0.706–0.947)	0.007
hs-CRP,Q4	1.429 (1.286–1.587)	<0.001	1.456 (1.267–1.673)	<0.001
Diabetes	Reference, Q1,2,3		Reference, Q1,2,3	
PAB, Q4	1.313 (1.123–1.535)	0.001	1.247 (1.033–1.505)	0.021
SOD1, Q4	1.086 (0.913–1.293)	0.35	1.038 (0.854–1.263)	0.7
hs-CRP,Q4	1.8 (1.576–2.056)	<0.001	1.599 (1.339–1.91)	<0.001

PAB, prooxidant-antioxidant balance; SO, superoxide dismutase. Reference group for PAB, SOD1, and hs-CRP was Q1, 2, 3 group; Q1, 2, 3 for PAB < 94, Q4 ≥ 94; Q1,2,3 for SOD1 <0.56, Q4 ≥ 0.56; Q1,2,3 for hs-CRP < 3.525, Q4 ≥ 3.525.

factors for metabolic syndrome in subjects with high BF% (Table 4). Our data also showed that serum PAB and hs-CRP (before and after adjustment for sex, age and smoking) were independent predictive risk factors for diabetes mellitus in subjects with high BF percentage. (Table 4).

4. Discussion

Our goal in using body fat percentage was as a more accurate assessment of obesity relative to the BMI. Previous studies have shown that people, who are classified as obese based on BMI, do not necessarily represent individuals with metabolic syndrome. As shown in this study, most of the subjects with metabolic syndrome or diabetes mellitus had a high body fat percentage, and therefore measuring body fat percentage may be a more appropriate parameter than BMI.

Serum PAB was significantly higher in subjects with a high BF percentage. Serum PAB concentrations were higher in individuals with metabolic syndrome and diabetes. Serum SOD1 activity was significantly lower in subjects with metabolic syndrome, but not significantly different in individuals with a high BF percentage and those with diabetes mellitus. Obesity, especially visceral, and subcutaneous adiposity, is a health problem that causes the development of metabolic syndrome and causes incidence of other diseases, such as diabetes mellitus, dyslipidemia,

hypertension, atherosclerosis, and cancer. Dysregulation of adipokine secretion from adipose tissue cause proinflammatory state and insulin resistance. SAT (subcutaneous adipose tissue)-secreted adipokines and plasma adipokine including CRP, PAI-1, IL-1, IL-6, IL-8, leptin, and MCP-1 may contribute to increased insulin resistance and low-grade inflammation, and promote risk of T2DM and CVD [22]. There are many mechanisms that may link obesity to the chronic diseases mentioned above, with oxidative stress being one of the most plausible [23]. One of the main reasons for ROS generation is the issue of oxidation of excess glucose and free fatty acids in the cells [24], particularly in adipocytes. It is shown that 3 T3-L1 adipocytes increase ROS production by the release of free fatty acids. Oxidative stress levels have been shown to increase in adipose tissue of obese mice but not in skeletal muscle or liver. This indicates that adipocytes are a major source of ROS and can cause obesity-related insulin resistance and type 2 diabetes. Moreover, treatment of obese mice with antioxidants has been shown to prevent the development of diabetes [13]. Mirhafez et al. have shown that diabetes mellitus is associated with an altered PAB, which may impair endothelial function, and cause atherosclerosis, insulin resistance, and functional impairment of pancreatic beta cells [25]. Ceriello et al. suggested that oxidative stress is a common pathogenic mechanism linked insulin resistance to dysfunction of pancreatic beta-cells and venous endothelial cells and develop diabetes and cardiovascular diseases; some cardiovascular drugs with intracellular antioxidant activity also prevents diabetes [16].

The natural antioxidant systems that reduce oxidative stress comprise several exogenous components, which may be obtained from foods, endogenous components, and antioxidant enzymes. The most important antioxidant enzymes involved in oxidative stress reduction are SOD, glutathione peroxidase (GPx), and catalase (CAT). Furukawa et al. showed that the level of activity and expression of SOD, GPx, and CAT enzymes in the adipose tissue of KKAy obese and diabetic mouse model was significantly less than C57BL/6 mice (control group). However, they did not find any changes in the activity and expression of these enzymes in the skeletal muscles and liver [13]. Our results of logistic regression show that higher serum SOD1 enzyme activity is associated with a lower prevalence of metabolic syndrome. As our study was cross sectional, prospective study must design to confirm this finding. This result is consistent with other studies that examine the effect of antioxidant agents or controlled dietary treatment on improving oxidative stress and antioxidant enzymes [26–28].

Serum hs-CRP was significantly higher in subjects with high BF percentage, and concentrations of serum hs-CRP were higher in individuals with metabolic syndrome and diabetes. Hs-CRP is a metameric protein released from the liver in response to the released factors from macrophages and adipocytes [29–31]. The metabolic syndrome has been shown to be associated with a pro-inflammatory state that is characterized by increasing hs-CRP level. It has also been shown that inflammation may induce insulin resistance and oxidative stress, leading to manifestations of metabolic syndrome [32] and some studies suggest that an elevated expression and activity of TLR2/TLR4 in monocytes of individuals with metabolic syndrome may contribute to an increase risk of CVD [33] . Several studies consider metabolic syndrome as a predictor of cardiovascular disease and diabetes [34–37].

Another aspect of the studies is the relationship between energy balance and diet with inflammation. Bawadi et al. showed that the positive energy balance is associated with waist circumference and truncal fat percentage as well as hs-CRP and HbA1c in diabetic patients. They showed that serum hs-CRP may increase dependent on the amount of energy derived from proteins and fat diets and does not have a significant relationship with carbohydrates [38]. In turn, Chae et al. showed that long-term mild weight loss can reduce serum levels of interleukin-1β, interleukin-6. It also reduces insulin, TG, free fatty acids, TC, LDL cholesterol, and leukocytes [39].

Measuring body fat percentage may be more appropriate than BMI to predict metabolic status in individuals. We show a positive association between percentage of BF and PAB oxidative stress and inflammatory conditions are two factors in people with a high percentage of BF that may lead to metabolic syndrome, diabetes, and cardiovascular disease.

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Conflict of Interest

The authors have no conflict of interest to disclose.

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