ERYTHROCYTE ANTIOXIDANT ENZYMES AND BLOOD PRESSURE IN RELATION TO OVERWEIGHT AND OBESE THAI IN BANGKOK

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Abstract. The specific activities of antioxidant enzymes, [*eg* superoxide dismutases (SOD), glutathione peroxidase (GPX) and catalase (CAT)], anthropometric measurements, including waist/hip ratio of 48 male and 167 female overweight persons (body mass index (BMI) ≥25.0 kg/m^2) compared with a 26 male and 80 female control group (BMI = 18.5-24.9 kg/m^2) of Thai volunteers who attended the Out-patient Department, General Practice Section, Rajvithi Hospital, Bangkok, for a physical check-up during March-October, 1998, were investigated. There was a slightly significant difference between the median age of the sexes. The medians of height, weight, and waist/hip ratio in males were significantly higher than those in female overweight and obese subjects. The median of arm circumference (AC), mid arm muscle circumference (MAMC) in males was significantly higher than those in female overweight and obese subjects ($p < 0.05$). The prevalences of hypertension based on systolic and diastolic blood pressure of ≥160/ ≥95 mmHg, were 8.3% and 37.5% for males and 5.4% and 18.6% for females, respectively. There was no significant difference between the median of antioxidant enzymes (SOD, GPX and CAT) between the sexes. No significant differences in the antioxidant enzymes in male overweight/obese persons and normal controls were presented, whereas antioxidant enzymes in female overweight/obese persons were statistically lower than in control females ($p < 0.05$). A significantly higher SOD, GPX, and CAT status was observed in normal subjects compared with overweight/obese subjects ($p < 0.01$). A higher prevalence of SOD ≤ 2,866 U/gHb, GPX (≤ 15.96 U/gHb in females was found, compared with males. A high percentage of lower catalase (CAT≤ 19.2x10^4 IU/gHb) was found in both sexes (64.5% in males and 64.5% in females). In obese subjects (BMI ≥ 30.0 kg/m^2), there were significantly positive relationships between systolic and diastolic blood pressure, systolic blood pressure and waist/hip ratio, and SOD could be related to weight, BMI as well as GPX and CAT, whereas the opposite result was observed for age and SOD.

INTRODUCTION

Obesity and dietary fat intake are associated with the prevalence of cardiovascular diseases, diabetes mellitus and cancer. In America, among 50-60 year olds, 42% of men and 52% of women are overweight (overweight being defined as a BMI value of 27.8 kg/m^2 or greater for men and 27.3 kg/m^2 or greater for women) (Kuczmarski *et al*, 1994). According to the most recent federal statistics, these figures are more than double the percentages of overweight persons among Americans 20 to 30 years old (20.2% of men and 20.2% of women) (Kuczmarski *et al*, 1994; National Center for Health Statistics, 1997). For adults, the cut-off point used by a WHO Expert Committee to delineate obesity are: <18.5 for thinness (chronic energy deficiency), 18.5-24.99 for normal, 25.0-29.99 for overweight grade I, 30.0-39.99 for overweight grade II, and ≥40.0 for overweight grade III (WHO Expert committee, 1995). The fact is that most Americans are unable to avoid gaining weight as they age. In general, men and women gain half a pound per year between the ages of 25 and 55. Thus, there is an ever increasing problem of overweight in the United States.

Obesity is not just a disease of developed nations. Obesity levels in some lower income and transitional countries are as high as, or higher than, those reported for the United States and other developed countries, and those levels are increasing rapidly. Shifts in diet and activity are consistent with these changes, but little systematic work has

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been done to understand all the factors contributing to these high levels. There is extensive documentation of populations in Thailand, Brazil, Cuba and Vietnam, with high energy and fat intakes and above average levels of obesity among adults (Mo-suwan *et al*, 1993; INCLEN Multi-center Collaborative Group, 1994).

The reasons for overweight are genetic (data from studies of twins), overeating, smoking cessation, alcohol consumption, lack of exercise, change in lifestyle, energy intake and energy expenditure (consider resting metabolic expenditure), environmental factors, salt/potassium retention, major depression/anxiety/other psychological or medical illness, medications, during and after pregnancy, cultural (perception of obesity), and socioeconomic factors. In Thailand, the transition from the rural to the urban environment affects health in various aspects, such as changes in eating habits, drinking of alcohol and smoking (Osuntokun, 1985; Hamburg, 1987; Epstein, 1989). Diseases related to obesity, such as hypertension and diabetes mellitus, have increased in rural Thai people (Chaisiri *et al*, 1997). Physical signs of obesity (overweight) are hypertension (increase blood pressure), coronary heart disease, predisposition to diabetes, hyperlipidemia (increased cholesterol level), metabolic abnormalities, increased risk of gallbladder disease, gout, some types of cancer, and development of osteoarthritis of the weight bearing joints. In the relationship between weight and blood pressure, being overweight is a significant risk factor for the development of hypertension. The prevalence of hypertension is greatly increased by the fact that more than half of all adults (prevalence varies by study) are overweight. Although the association between higher body fat and blood pressure has been recognized for years, recent studies discovered a 50% to 30% higher incidence of hypertension among adults who consider themselves overweight, compared with those classified as being of normal weight. Similar findings are related from studies involving children and young adults, in which the correlation coefficient between weight and blood pressure has been observed to be as high as 0.4. Two proposed mechanisms underlying this correlation are the stimulation of sodium retention and increased catecholamine release, which are results of increased sodium sensitivity and hyperinsulinemia.

Age, gender and race are modifiers/confounders of obesity. It has been recently suggested that oxidative stress induced by reactive oxygen species (ROS) are capable of reacting with unsaturated lipids and of initiating chain reactions of lipid peroxidation in the membrane, leading to their oxidation, and followed by (1) a decrease in the half life of bio-molecules, (2) a loss of protein functions; enzymes, receptors, membrane phospholipids, (3) the appearance of toxic products: oxidized LDL, MDA, (4) the formation of MDA-proteins and MDA-DNA adducts. However, cells are protected against oxidative damage by the body's defense system. The first of these involves enzymes that directly metabolize ROS superoxide dismutase (SOD), glutathione peroxidase (GPX), and catalase (CAT)]. Among these enzymes, SOD plays a central role in the metabolism of ROS by directly dismuting the superoxide anion radical in hydrogen peroxide, which is scavenged by CAT and GPX. The latter enzyme requires the presence of reduced glutathione (GSH) to be effective (Fridovich, 1985). The other defense system includes molecules that interact directly with free radicals to neutralize them (*eg* ascorbic acid, α-tocopherol, retinol and glutathione).

The aim of this study was to determine trends in overweight and obesity according to BMI in relation to antioxidant enzymes and blood pressure in healthy overweight and obese Thais compared with normal subjects. This would both establish baseline values for these parameters and allow us to investigate the change in antioxidant enzymes in obese persons.

MATERIALS AND METHODS

Study population

Forty-eight male and 167 female overweight and obese Thai volunteers including 26 male and 80 female normal subjects comprised the study population. Thai volunteers who attended the Out-patient Department, General Practice Section, Rajvithi Hospital, Bangkok, for a physical check-up during the period March-October, 1998, were investigated for this study. The age, marital status, place of origin, drinking and smoking habits were assessed by standardized questionnaires. A physical examination was undertaken by a medical doctor.

Analytical methods

The nutritional status of all subjects under investigation was assessed by mean of anthropometric measurements. The body weight of each individual dressed in light clothing was measured using a carefully calibrated beam balance (Detecto®).

Height measurements were taken using a vertical measuring rod. BMI or Quetelet Index was conventionally calculated as weight in kg/ (height in meters)^2. The classifications of BMI employed were those used by the WHO Expert Committee (1995), overweight grade I: BMI=25.00-29.99; grade II (obese): BMI=30.00-39.99; grade III (obese) : BMI≥40 kg/ m^2. Waist and hip circumferences were also measured in order to calculate waist/hip ratio (normal value for female< 0.77, male<0.90)(Dowling *et al*, 1993; Seidell *et al*, 1994).

About twenty ml of venous blood from all the subjects after overnight fast were drawn in the morning before the process of physical examination. Blood for antioxidant enzymes determination were collected in heparinized tubes and the hemolysates were analyzed in the next few hours. Determination of enzymes, SOD was performed by using a Randox test combination (Randox, Grumlin, United Kingdom). Xanthine and xanthine oxidase were used to generate superoxide radicals that react with 2-(4-iodophenyl) 3-(4-nitrophenol)-5 phenyl tetrazolium chloride (INT) to form a red formazan dye. Substrate concentrations were 0.075 μmol for xanthine and 0.037 μmol for INT. A single substrate concentration is imposed by the Randox test because of first order kinetics of the enzymatic reaction. The use of these concentrations allows standardization of method. SOD inhibits this reaction by converting the superoxide radical to oxygen. One SOD unit inhibits the rate of reduction of INT by 50% at 37ºC and pH 7 for 1 minute in a complex system with xanthine and xanthine oxidase. Because of the small linear range of the test, the sample must be diluted so that the percentage inhibition falls between 30% and 60%. A standard curve is prepared, using the standard provided in the kit, and the value for the diluted sample is read from this curve. SOD activity was measured at 505 nm on a Shimadzu spectrophotometer (UV-160 A) on hemolysates of washed erythrocytes. For this, 500 μl of whole blood was treated three times with 3 ml 0.9% NaCl. After centrifugation at 3,000*g* for 10 minutes, the supernate was eliminated and 2ml of de-ionized water was added to lyse the washed erythrocytes. Hemoglobin (g/l) was measured on an STKR cell counter (Coultronics, Margency, France). Results were expressed as U SOD/g hemoglobin.

GPX activity was determined, using a Randox test combination. Erythrocyte GPX catalyzes the oxidation of glutathione (at a concentration of 5 μmol) by cumene hydroperoxide according to the method of Paglia *et al* (1967). In the presence of glutathione reductase (GR; at a concentration ≥$0.75x10^{-3}$ U) and 0.035 μmol NADPH, the oxidized glutathione is immediately converted to the reduced form with a concomitant oxidation of NADPH to $NADP^+$. The decrease in absorbance at 340 nm was measured at 37ºC. The assay was performed on a hemolysate of erythrocytes obtained from the mixing of 0.05 ml whole blood with 1 ml cold diluting agent and 1 ml Drabkin reagent. One GPX unit was defined as the enzyme activity necessary to convert 1 μmol NADPH to $NADP^+$ at 37ºC and pH 7.2 in 1 minute. The results are expressed as UGPX/g hemoglobin.

The red blood cell CAT activity was assessed according to Aebi (1984). This method is based on the decomposition of hydrogen peroxide by CAT. The decrease in absorbance at 230 nm was measured at 25ºC in the presence of 0.15 volume H_2O_2. The assay was performed on hemolysates of washed erythrocytes as described for SOD determination. A standard curve was prepared by using the CAT provided by Boehringer Mannheim (Mannheim, Germany). CAT activity is expressed as U CAT/g hemoglobin. One CAT unit was defined as the enzyme activity necessary to convert 1 μmol H_2O_2 and molecular oxygen at 25ºC and pH 7 in 1 minute.

Blood pressure was recorded in a sitting position, using a standardized automatic electronic blood pressure meter.

Statistical analysis

The statistical evaluation of data was carried out using the statistical package MINITAB (Ryan *et al*, 1985). To assess statistically significant differences between males and females, the Mann-Whitney U-Wilcoxon Rank Sum W (two-tailed) test was used.

RESULTS

Medians, ranges and 95% confidence interval (CI) of age, anthropometric variables, waist/hip ratio, blood pressure and red blood cell antioxidant enzymes (SOD, GPX, CAT) of overweight and normal subjects, and overweight and normal subjects (males and females), are shown in Table 1 and Table 2. The ages of males and females of overweight subjects were in the ranges 18-55 and 18-58 years, whereas the ages of normal subjects were in the ranges 19-54 and 18-55 years, respectively, and there was no significant difference of age between both sexes. All of the anthropometric variables, except

Table 1
Median, ranges and 95% confidence interval (CI) of age, anthropometric variable, blood pressure, and antioxidant enzymes in overweight and control subjects.

Parameter	Total				p-value[a]
	Overweight (N=215)		Control (N=106)		
	Median (range)	95%CI	Median (range)	95%CI	
Age (yrs)	38 (18-58)	37-40	36.5 (18-55)	33-40	0.233
Weight (kg)	76.7 (54.0-129.2)	74.9-79.6	54.3 (42.5-78.0)	52.6-55.9	0.000
Height (m)	1.56 (1.45-1.84)	1.55-1.57	1.59 (1.43-1.85)	1.57-1.60	0.088
BMI (kg/m^2)	31.01 (25.19-53.28)	30.48-31.64	21.85 (18.18-24.82)	20.99-22.28	0.000
SST (mm)	27.0 (10.9-55.1)	26.9-27.5	20.0 (10.1-30.1)	19.8-20.9	0.000
AC (mm)	33.0 (27.0-53.0)	32.0-33.2	27.0 (23.0-32.0)	27.0-27.8	0.000
TSF (mm)	27.1 (12.2-58.0)	25.2-27.6	21.3 (6.0-31.7)	20.9-22.1	0.000
MAMC (mm)	24.6 (16.2-39.0)	24.2-25.2	20.4 (16.9-25.4)	20.0-21.0	0.000
Waist (cm)	91.0 (66.5-127.0)	89.8-93.0	72.5 (60.0-89.0)	71.2-74.0	0.000
Hip (cm)	108.0 (86.5-151.5)	106.0-110.0	92.0 (82.0-102.0)	92.0-94.0	0.000
W/H ratio	0.84 (0.67-1.01)	0.83-0.85	0.78 (0.65-0.93)	0.77-0.80	0.000
Systolic BP (mmHg)	126.0 (96.0-190.0)	123.0-130.0	116.0 (88.0-184.0)	111.5-120.0	0.000
Diastolic BP (mmHg)	82.0 (57.0-130.0)	80.0-86.0	76.0 (58.0-109.0)	73.0-78.0	0.000
SOD (U/gHb)	1,575 (234-16,312)	1,334-1,842	2,640 (316-23,881)	2,363-3,029	0.000
GPX (U/gHb)	28.10 (4.20-232.10)	25.41-30.05	34.90 (9.30-179.40)	30.00-38.00	0.0051
CAT ($x10^4$IU/gHb)	17.61 (6.51-47.69)	16.49-18.36	19.31 (5.50-58.18)	18.35-21.30	0.0053

BMI = body mass index
AC = arm circumference
SST = subscapular skinfold thickness
MAMC = mid arm muscle circumference
W/H = waist/hip ratio
TSF = tricep skinfold thickness
GPX = glutathione peroxidase
CAT = catalase
SOD = superoxide dismutase
[a]Mann-Whitney U-Wilcoxon Rank Sum W test (Two-tailed)

the height of the overweight group, were significantly higher than those of normal subjects. The antioxidant enzyme SOD, GPX and CAT of the overweight group were shown to be significantly lower than the normal groups (Tables 1, 3).

The medians of weight and waist/hip ratio of overweight and obese males were significantly higher than those of overweight and obese females. Both median systolic and diastolic blood pressures of males were significantly higher than those of

Table 2
Medians, ranges and 95%(CI) of age, anthropometric variable and blood pressure in overweight and control subjects between male and female.

Parameter	Male				p-value	Female				p-value[a]
	Overweight (N=48)		Control (N=26)			Overweight (N=167)		Control (N=80)		
	Median (range)	95%CI	Median (range)	95%CI		Median (range)	95%CI	Median (range)	95%CI	
Age (yrs)	40.5 (18.0-55.0)	38.8 -45.2	35.5 (19.0-54.0)	30.0 -42.0	0.0689	37.0 (18.0-58.0)	25.4 -39.0	37.0 (18.0-55.0)	32.8 -40.0	0.7577
Weight (kg)	85.7 (62.4-114.8)	81.0 -91.5	61.8 (50.7-78.0)	58.9 -64.7	0.0000	74.8 (54.0-129.2)	72.7 -76.4	52.5 (42.5-67.0)	51.2 -54.0	0.0000
Height (m)	1.69 (1.50-1.84)	1.67 -1.72	1.65 (1.57-1.85)	1.64 -1.71	0.5217	1.55 (1.45-1.69)	1.54 -1.56	1.56 (1.43-1.69)	1.54 -1.58	0.0473
BMI (kg/m^2)	30.58 (25.35-38.65)	29.18 -31.93	21.74 (18.68-24.61)	20.61 -23.23	0.0000	31.13 (25.19-53.28)	31.48 -31.89	21.85 (18.18-24.82)	20.90 -22.19	0.0000
SST (mm)	26.7 (14.9-50.1)	25.4 -28.0	17.0 (10.1-30.1)	16.0 -17.4	0.0000	27.0 (10.9-56.0)	26.9 -27.5	21.4 (10.1-30.1)	20.8 -22.0	0.0000
AC (mm)	33.0 (17.5-39.0)	32.0 -34.0	27.5 (24.0-32.0)	26.7 -29.4	0.0000	32.5 (27.0-53.0)	32.0 -33.5	27.0 (23.0-32.0)	26.9 -27.5	0.0000
TSF (mm)	25.6 (12.2-36.0)	22.9 -28.1	19.4 (6.0-27.0)	17.4 -20.5	0.0000	27.1 (15.8-58.0)	25.2 -27.9	22.0 (14.7-31.7)	21.2 -23.2	0.0000
MAMC (mm)	25.8 (18.2-32.3)	24.8 -26.4	21.9 (17.7-25.4)	21.3 -23.2	0.0000	24.4 (16.2-39.0)	23.8 -24.8	20.1 (16.9-24.7)	19.5 -20.4	0.0000
Waist (cm)	98.5 (65.0-122.0)	93.0 -103.0	80.5 (64.0-89.0)	74.7 -83.7	0.0000	90.0 (71.0-127.0)	86.4 -91.0	71.0 (60.0-83.0)	70.0 -72.1	0.0000
Hip (cm)	106.5 (89.5-129.0)	103.4 -110.0	94.0 (87.0-102.0)	91.0 -95.4	0.0000	108.0 (86.5-151.5)	106.0- 110.0	92.0 (82.0-101.1)	91.4 -93.1	0.0000
W/H ratio	0.93 (0.67-1.01)	0.91 -0.94	0.87 (0.72-0.93)	0.82 -0.88	0.0000	0.83 (0.70-0.99)	0.82 -0.84	0.77 (0.65-0.88)	0.76 -0.78	0.0000
Systolic BP (mmHg)	131.0 (96.0-170.0)	129.8 -140.0	124.0 (109.0-184.0)	117.2 -128.7	0.0107	123.0 (100.0-190.0)	120.0 -130.0	111.5 (88.0-184.0)	110.0 -118.2	0.0000
Diastolic BP (mmHg)	90.0 (60.0-120.0)	81.8 -97.0	78.5 (68.0-104.0)	75.7 -87.0	0.0023	80.0 (57.0-130.0)	80.0 -84.0	75.0 (58.0-109.0)	70.0 -77.2	0.0000

BMI = body mass index SST = Subscapular skinfold thickness TSF = Tricep skinfold thickness
AC = arm circumference MAMC = mid arm muscle circumference W/H = waist/hip ratio
[a]Mann-Whitney U-Wilcoxon Rank Sum W test (Two-tailed)

Table 3
Medians, ranges and 95% confidence interval (CI) of antioxidant enzymes in overweight and control subjects between male and female.

Parameter	Male				p-value	Female				p-value[a]
	Overweight (N=48)		Control (N=26)			Overweight (N=167)		Control (N=80)		
	Median (range)	95%CI	Median (range)	95%CI		Median (range)	95%CI	Median (range)	95%CI	
SOD (U/gHb)	1,613 (234-6,624)	1,171-2,776	2,750 (1,316-23,881)	2,241-3,472	0.0564	1,571 (235-16,312)	1,320-1,886	2,528 (1,111-11,395)	2,245-3,033	0.0000
GPX (U/gHb)	25.4 (11.1-62.1)	21.6-30.8	29.8 (9.3-68.8)	19.5-49.1	0.509	28.7 (4.2-232.1)	25.7-30.4	35.6 (9.1-179.4)	30.4-38.7	0.0052
CAT (x10^{4}IU/gHb)	16.2 (6.5-36.4)	14.4-19.8	19.7 (9.2-33.1)	16.2-21.7	0.1985	17.8 (7.0-47.7)	16.8-18.7	19.1 (5.5-58.2)	17.9-21.4	0.0135

SOD = superoxide dismutase GPX = glutathione peroxidase CAT = catalase
[a]Mann-Whitney U-Wilcoxon Rank Sum W test (Two-tailed)

females (Table 2). The prevalence of hypertension based on systolic blood pressure of ≥ 160 mmHg and diastolic blood pressure of ≥ 95 mmHg, were 8.3% and 37.5% for males and 5.4% and 18.6% for females, respectively (Table 4). A higher prevalence of SOD ≤ 2,866 U/gHb, GPX ≤ 15.96 U/gHb (Winterbourn *et al*, 1975; Bentler, 1984; Ongajyooth *et al*, 1987) in females was found when compared with males. High percentage of lower catalase (CAT ≤ 19.2x10^4 IU/gHb) (Winterbourn *et al*, 1975; Bentler, 1984; Ongajyooth *et al*, 1987) was found in both sexes (64.5% in males and 64.5% in females) (Table 4).

Tables 5 and 6 show the correlation coefficients between various parameters in male and female overweight subjects (BMI ≥ 25.0 kg/m^2) respectively. There was a significantly positive correlation between systolic and diastolic blood pressure in both overweight and obese subjects, as well as arm circumference (AC), tricep skinfold (TSF)and waist/hip ratio in the overweight.

In obese subjects (BMI ≥ 30.0 kg/m^2), there were significantly positive relationships between systolic and diastolic blood pressure, systolic or diastolic blood pressure, and age and waist. A significant positive correlation was found between systolic blood pressure and waist/hip ratio, as well as weight, BMI, AC, TSF, hip and SOD. SOD could be related to weight, BMI, as well as GPX and CAT whereas the opposite result was observed for age and SOD (Table 5).

DISCUSSION

The distribution of obese subjects in this study according to the grading of the 1995 WHO group was 38.6, 56.3 and 5.1% for grades I, II and III, respectively, with a predomination of grade II (BMI=30.0-39.99 kg/m^2). This result may indicate the need for counseling and controlling the transformation from grade II to grade III. Over 80% of obese subjects had a waist/hip ratio higher than the cut-off point (Table 4). Although overweight can be estimated from anthropometric measurements (BMI), waist/hip ratio has been shown to be closely correlated with more direct measurements of visceral fat in women, especially in Asian women (Bentler, 1984). It has been shown that Vietnamese women appear to have a low waist/hip ratio with also a low BMI, and this has been noted in other Asian women (Masuda *et al*, 1993) where lean women in China

Table 4
Number and percentage of individuals with overweight, hypertension and abnormal plasma antioxidant enzymes.

Parameter	Male		Female		Total	
Grading of overweight by BMI (kg/m^2)	N/Total	%	N/Total	%	N/Total	%
Grade I (BMI = 25.0-29.99)	20/48	41.7	63/167	37.7	83/215	38.6
Grade II (BMI = 30.0-39.99)	28/48	58.3	93/167	55.7	121/215	56.3
Grade III (BMI ≥ 40.0)	-	0.0	11/167	6.6	11/215	5.1
Waist/Hip ratio					181/215	84.2
Male ≥ 0.90	36/48	75				
Female ≥ 0.77			145/167	86.8		
Systolic BP ≥ 160 mmHg	4/48	8.3	9/167	5.4	13/215	6.1
Diastolic BP ≥ 95 mmHg	18/48	37.5	31/167	18.6	49/215	22.8
Plasma antioxidant enzymes						
SOD ≤ 2,866 (U/gHb)	21/31	67.7	88/119	73.9	109/150	72.7
GPX ≤ 15.96 (U/gHb)	1/30	3.3	17/118	14.4	18/148	12.2
Catalase ≤ 19.2x10^4 IU/gHb	20/31	64.5	75/118	63.0	95/149	63.8

Table 5
Correlation coefficients of age, anthropometric variables and antioxidant enzymes in both overweight males and females (BMI ≥ 25.0 kg/m^2).

Parameter	Systolic	Diastolic	SOD	GPX	Catalase
Age	0.1977[b]	0.1127	-0.0460	0.0627	-0.0826
Weight	0.1135	0.1977[b]	0.1916[a]	-0.0735	-0.027
Height	0.0923	0.1012	0.1280	-0.0513	0.0099
BMI	0.0634	0.1560[a]	0.1523	-0.0533	-0.0338
SST	0.1742[a]	0.2027[b]	0.2581[b]	0.0302	0.0715
AC	0.1486[a]	0.2369[b]	0.1996[a]	-0.1235	-0.0893
TSF	0.1827[b]	0.1966[b]	0.1963[a]	-0.0779	0.0077
MAMC	0.1076	0.1724[a]	0.0757	-0.1062	-0.1238
Waist	0.2754[b]	0.2810[b]	0.1432	-0.0208	-0.0554
Hip	0.1429[a]	0.2061[b]	0.2012[a]	-0.0588	-0.0979
Waist/Hip ratio	0.2237[b]	0.1619[a]	-0.0139	0.0084	-0.0187
Systolic	1.0000	0.7500	0.0629	-0.0588	0.0988
Diastolic	0.7500	1.0000	0.1158	-0.0659	-0.0274

Significant difference: [a]$p < 0.05$, [b]$p < 0.01$

tended to have a higher waist/hip ratio of 0.8 while mean BMI was relatively low. Therefore, the identification of obesity should use both indicators, BMI and waist/hip ratio, because the relatively high waist/hip ratio means that with increasing weight gain they could reach unacceptable levels of abdominal obesity leading to increase risk of diabetes mellitus and heart disease (Folsom *et al*, 1994).

A high prevalence of hypertension in males might lead to an increased risk of heart disease; advice and support for behavioral modification, including eating a healthy diet and stopping smoking, which is a major health risk in males, should be recommended, especially in obese subjects.

There was significantly lower SOD, GPX and CAT activity in overweight and obese subjects than in control subjects. In overweight and obese subjects, 72.7% had low red blood cell SOD and 63.8% had low red blood cell CAT (Table 4). The overweight and obese had a higher risk, including artheriosclerotic heart disease, angina pectoris, stroke, hypertension, non-insulin dependent diabetes mellitus, obstructive pulmonary disease, gout, nephrolithiasis and sudden cardiac death (Berensen *et al*,

Table 6
Correlation coefficients of age, anthropometric variables and antioxidant enzymes in both obese males and females (BMI ≥ 30.0 kg/m^2).

Parameter	Systolic	Dyastolic	SOD	GPX	Catalase
Age	0.2140[a]	0.1974[a]	-0.1743[a]	-0.0095	-0.1311
Weight	0.0886	0.1184	0.2100[a]	0.0940	0.1038
Height	0.0832	0.0622	0.0656	0.0002	0.0618
BMI	-0.0185	0.0523	0.2292[a]	0.1532	0.1050
SST	0.1782[a]	0.1751[a]	0.1735	-0.0866	0.0487
AC	0.0634	0.0761	0.2714[a]	0.1320	0.0997
TSF	0.1643	0.1159	0.3021[a]	0.1413	0.2005
MAMC	0.0026	0.0290	0.1359	0.1003	0.0220
Waist	0.2780[b]	0.2405[a]	0.1459	0.0511	0.0655
Hip	0.1001	0.1386	0.2203[a]	0.0969	0.0896
Waist/Hip ratio	0.2224[a]	0.1498	-0.0406	-0.0456	-0.0406
Systolic	1.0000	0.7688[b]	0.1117	0.0553	0.1698
Diastolic	0.7688[b]	1.0000	0.1125	0.0160	0.1015

Significant difference: [a]$p < 0.05$, [b]$p < 0.01$

1998). Increased oxidative stress in obesity may be exacerbated by decreased availability of antioxidants. Decreased antioxidant levels have been found in obese adults (Moor de Burgos *et al*, 1992). The depletion of antioxidant enzymes might increase the risk of heart disease, cancer, age-associated degenerative diseases and genetic damage, which might cause by DNA damage (Mandal, 1991; Reddy *et al*, 1991; Rongliang *et al*, 1991). Epidemiological and experimental studies show that antioxidants and antioxidant enzyme may protect against free radical-mediated damage (van Antwerpen, 1995). Depletion of total antioxidants and antioxidant enzymes among the obese may also elevate levels of lipid peroxides (Olinescu *et al*, 1992). SOD is believed to play a major role in the metabolism of ROS (reactive oxygen species). It is the first enzyme involved in the destruction of superoxide anion radicals. Then it converts superoxide into hydrogen peroxide, which is metabolized by CAT and GPX in synergy with glutathione reductase (GSH). Animal cells contain two intracellular forms of SOD: the cytoplasmic or copper-zinc form (Cu-Zn SOD) and mitochondrial or manganese form (Mn-SOD) (Fridovich, 1985). This enzyme is the first line of defense against superoxide anion radicals and can be induced rapidly in some conditions such as exposure to an oxidative stress of cells or organs (Coursin *et al*, 1985). Like SOD activity, CAT activity was significantly lower in our overweight subjects, compared with the control subjects.

From a kinetic point of view, CAT and GPX are both able to destroy hydrogen peroxide, but GPX has a much higher affinity for hydrogen peroxide than does CAT, suggesting that hydrogen peroxide is mainly degraded by GPX under normal conditions. It may be that GPX activity first increases under lipoperoxidation via an adaptive response similar to that of SOD, and then that GPX and CAT activity decreases as a result of its consumption. Although there was no correlation between BMI with antioxidant enzyme in the overweight (BMI ≥ 25.0 kg/m^2), antioxidant enzyme status especially GPX and CAT was decreased with anthropometric parameters.

Reddy *et al* (1997) suggested that free radical concentration and DNA damage should take account of BMI in comparison of individuals along with other factors such as smoking and alcoholism. Intake of dietary fat might be another factor to enhance increasing free radical generation (Reddy *et al*, 1997). A higher metabolic rate among lean persons may promote an increase in oxidative damage (Chun *et al*, 1992; Shah *et al*, 1998). The marked reducing of antioxidant enzyme might indicate the elevation of free radical generation in overweight and obese subjects. It was of interest in this study that the weight regulation or metabolic control in the overweight and obese people require particular attention in terms of public health intervention.

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