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Serum Selenium and Glutathione Peroxidase in Patients with Obesity and Metabolic Syndrome

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Abstract: There are limited data on the relationship between antioxidant status and features of the Metabolic Syndrome. We have determined the serum selenium and glutathione peroxidase concentrations in Caucasian patients with obesity and metabolic syndrome. Patients (n = 237) were recruited from Cardiovascular risk management clinics at the Royal Surrey County Hospital, Guildford. Individuals who were non-obese, without a history of coronary disease and who were not on any prescribed medication (n = 135) were recruited from staff of the university and hospital. All data were adjusted for age and gender using analysis of covariance (ANCOVA). Overall, clinic patients had a significantly higher dietary intake of total fat, protein and selenium compared with the healthy individuals. Patients also had a significantly higher serum selenium (1.08±0.23 umol/L, p<0.05) and lower serum GPx (0.31±0.01 U/mL, p<0.001) concentrations compared to the healthy individuals (1.03±0.2 and 0.36±0. 1 respectively). Within the patient group, obese subjects had significantly higher serum concentrations of selenium (1.04±0.24 umol/L, p<0.05) and lower serum GPx (0.28±0.09 U/mL, p<0.001) compared with non-obese patients (1.10±0.23 and 0.33±0.10 respectively). Moreover, within this group, serum selenium concentrations decreased significantly with accumulating features of metabolic syndrome (p<0.05). The lower levels of serum GPx in obesity and lower concentrations of serum selenium associated with accumulating features of the metabolic syndrome may be related to the presence of an atherosclerosis prone state with an increased consumption of antioxidants by free radical interaction.

Key words: Selenium, GPx, obesity and metabolic syndrome

Introduction

Selenium (Se) is an essential trace element (Thomson and Robinson, 1980) and is a key component of several functional selenoproteins required for normal health of which glutathione peroxidase (GPx) is the best known. In some studies an increased risk of cardiovascular disease has been reported in populations with low serum selenium (<0.57 µmol/L). In a Finnish study, serum selenium levels were inversely related to cardiovascular death and myocardial infarction (Salonen *et al*., 1982). In a Dutch case-control study, lower toenail selenium levels were associated with an increased risk of acute nonfatal myocardial infarction (Kok *et al*., 1989), a finding consistent with a another European multicenter case-control study that showed that lower toenail selenium levels were associated with acute myocardial infarction (Kardinaal *et al*., 1997), although in this case the association was restricted to current smokers. Although the evidence from other case-control and prospective studies for an association between selenium status and cardiovascular disease remains controversial (Rayman, 2000; Salonen *et al*., 1995; Neve, 1996; Alissa *et al*., 2003).

Metabolic syndrome as defined by International Diabetic Federation criteria, affects up to 39% (men 39.9±1.7%, women 38.1±1.2%) of the adult population (Ford *et al*.,

2003) and is probably the most-prevalent risk factor for cardiovascular disease in populations with normal or moderately increased serum cholesterol. It has been reported that adults with the metabolic syndrome have suboptimal concentration of several antioxidants, such as selenium (Ford, 2005a). The prevalence of obesity has tripled between 1981 and 2001, with one in five adults being classified as obese and nearly two-thirds of men and over half of women being overweight or obese [Body Mass Index (BMI)>25 kg/m^2] in the UK. (James, 2004; James *et al*., 2004).

It has been reported that obese patients have significantly lower serum selenium concentrations compared to non-obese patients (Gjorup and Andersen, 1988). Furthermore (Ozata *et al*., 2002), found lower GPx concentrations in obese versus non-obese patients. In this present study we intended to investigate measures of selenium and GPx status in subjects with metabolic syndrome and obesity compared to healthy subjects.

Materials and Methods

Subjects: Patients (n = 237) were recruited from the Cardiovascular risk management clinic that divided by gender at the Royal Surrey County Hospital, Guildford, U.K. The size of the sample population was sufficient for an 80% chance of detecting a difference between groups at a level of $p<0.05$ based on previous study (Ford *et al*., 2003). Of the patients, 82 were obese (BMI = 30 kg/m^2) and 161 had metabolic syndrome as defined by IDF, 2005 criteria (Ford, 2005b).

Healthy individuals (n = 135) were recruited from staff of the university and hospital who were not obese. They did not have any known medical disorder, nor were taking any prescribed medication. Each subject gave informed written consent to participate in the study, which was approved by the South-West Surrey Research Ethics Committee and The University of Surrey Advisory Committee on Ethics.

Anthropometric and other measurements: All subjects were measured for height, waist and hip circumference (in centimeters) and weight in kilograms using a stand-on Bio Impedance Analyzer (BIA) (Tanita-305 body fat analyzer, Tanita Corp., Tokyo, Japan). The latter was also used to estimate percent body fat. Blood pressure measurements were made using an automated device (DINAMAP compact monitor, model TS, Critikon, Tampa, Florida, USA, FA 33634). Dietary intake over the previous 12 months was assessed using a Food Frequency Questionnaire (FFQ) as previously detailed (Ghayour-Mobarhan *et al*., 2005a).

Routine biochemical and Serum selenium analyses: All chemicals were obtained from Sigma (Sigma Chemical Co, Dorset, UK) unless stated otherwise.

A full, fasted lipid profile, comprising total cholesterol, triglycerides and HDL cholesterol, was determined for each patient. LDL cholesterol was calculated using the Friedwald equation, except for patients with triglycerides >4.0 mmol/L (Friedewald *et al*., 1972). Serum lipid and blood glucose were measured by enzymatic methods and high sensitivity CRP concentrations were determined by PEG enhanced immuno-turbidimetry using a Bayer Advia 1650 analyser (Bayer, Newbury, UK). Serum selenium was determined by electrothermal atomic absorption spectrometry with Zeeman background correction using a palladium chloride chemical modifier (Ghayour-Mobarhan *et al*., 2005b). Typical between batch precision (CVs) was 3.7%.

Glutathione peroxidase assay: Serum glutathione peroxidase (GPx) was measured using a modification of the method of Paglia and Valentine (Paglia and Valentine, 1967). Briefly 10 µL of serum, standard (0.1-0.3 U/mL purified glutathione peroxidase) or water (blank) was added in quadruplicate to a 96-well plate. 290 µL of 0.1 M phosphate buffer containing; 5mmol/L EDTA, 200µmol/L sodium azide, 1U/mL glutathione reductase, 0.86mmol/L NADPH, 2 mmol/L reduced glutathione and 7.8 µmol/L *t*-butyl hydroperoxide; was added to each well. The reagents were mixed and the absorbance at 340 nm measured continuously for 5 minutes in an iEMS MF plate reader. The between assay CV was typically 5.5%.

Statistical analysis: Statistical analysis was undertaken using Minitab (release 13, Minitab Inc, 2000, USA). Between-group comparisons of dietary and biochemical parameters were assessed by ANOVA. Post-hoc, Dunnett tests were used after using one-way analysis of variance. Non-normally distributed data such as serum hs-CRP and triglycerides were log transformed before using one-way analysis of variance. Categorical data were compared using Fisher's exact, or Chi square tests. Values were expressed as mean±SD, as they are normally distributed. Analysis of covariance (ANCOVA) was used to assess differences after adjustment for important confounding factors such as age and gender. A p value of <0.05 was considered significant.

Results

The demographic and biochemical data for the patients and healthy subjects are summarised in Table 1. There was a high frequency of obesity 34.5%(men 35.2%, women 33.6%), metabolic syndrome 67% (men, 65.4%, women 71.5%) and positive smoking habit (18%) in the patient group. These findings are typical of a cardiovascular risk management clinic population. Serum fasting blood glucose, triglycerides and total and LDL cholesterol were significantly higher in the patient group, whether with or without obesity, or metabolic syndrome, compared to the healthy subjects. Indices of adiposity including waist circumference, waist/hip ratio, BMI and percentage

Table 1: Clinical and biochemical characterisation of patients and healthy subjects (Males and females)

		---------- Clinic Patients ----------				
	Total	---------- obesity ----------		------ Metabolic syndrome ------		Healthy
Group	patient	yes	no	yes	no	subjects
Number	237	82	155	161	76	135
Age (years)	55.1±13.3[3]	54.4±11.3[3]	55.8±14.2	57.3±12.4[3]	50.5±14.3	48.8±14.6
Male/Female	142/95	50/32	92/63	108/68	34/27	67/68
Current smokers (%)	18	26*	14	17	20	17
Systolic BP (mmHg)	146.7±20.4[3]	149.8±19.3[3]	145±20.8	151.3±19.2[3***]	136.8±19.4	125.2±16
Diastolic BP (mmHg)	82.1±0.7[3]	83.8±13[3]	81.1±11.2	84.4±12[3***]	77±9.8	74.3±9.1
%Body fat	32.4±8.2[3]	37.8±7.7[3***]	29.6±6.9	34.9±7.4[3***]	27.1+7.1	26.5±7.3
BMI	29±5.2	34.5±4.1	26.1±2.3	30.5±4.8	26±3.9	24.2±2.9
WHR	0.92±0.08	0.96±0.08	0.90±0.07	0.94±0.07	0.88±0.08	0.85±0.08
Triglycerides (mmol/L)	2.6 (1.7-4.5)[3]	3.5 (1.9-5.8)[3]	2.3 (1.5-3.5)	2.95 (1.9-5)[3***]	1.9 (1.1-2.8)	1.06 (0.8-1.3)
HDL-C (mmol/L)	1.2±0[3]	1.1±0[3***]	1.3±0.4	1.2±0[3***]	1.3±0.4	1.7±0
FBG (mmol/L)	5.9±1.6[3]	6.4±1.9[3***]	5.6±1.3	6.2±1.7[3***]	5.2±0.9	4.9±0.4
Hs-CRP (mg/L)	1.25(0.4-3.3)[3]	2.8 (1.0-5.8)[3***]	0.84 (0.3-2.0)	1.7 (0.6-3.7)[3***]	0.6 (0.2-2.6)	0.57 (0.1-1.4)
Serum Selenium (µmol/L)	1.08±0.23[1]	1.04±0.24*[1]	1.10±0.23	1.08±0.23	1.08±0.23	1.03±0.03
Serum GPx (U/mL)	0.31±0.01[3]	0.28±0.09[3***]	0.33±0.10	0.31±0.11[2]	0.31±0.08	0.36±0.1

Values expressed as Mean±SD or median and interquartile range. Comparisons between the healthy subjects and Clinic patients and between different sub-categories of the Clinic patients (obese and non-obese; metabolic syndrome and non-metabolic syndrome) were analysed using one-way analysis of variance, Post-hoc, Dunnett's tests were used after using one-way analysis of variance. Non-normally distributed data such as serum hs-CRP and triglycerides log transformed before using one-way analysis of variance. 1 = p<0.05, 2 = p<0.01 and 3 = p<0.001 compared to healthy subjects. * = p<0.05, ** = p<0.01 and *** = p<0.001 comparisons between different sub-categories of the Clinic patients (obese v. non-obese; metabolic syndrome and non-metabolic syndrome).BP = Blood pressure; HDL-C = High density lipoprotein cholesterol; FBG = Fasting blood glucose; Hs - CRP = High sensitivity C-reactive protein; Gpx = Glutathione peroxidase. WHR = waist hip ratio

Table 2: Clinical and biochemical characterisation of patients and healthy subjects (Females)

		---------- Clinic Patients ----------				
		---------- Obesity ----------		------- Metabolic syndrome -------		Healthy
Group	Total	Yes	No	Yes	No	subjects
Number	95	32	63	68	27	68
Age (years)	57.9±13.7[3]	56.5±10.9[3]	58.7±15	60.4±11.4	51.6±17	48.7±14
Smoking (%)	18	27	15	18	21	19
Systolic BP (mmHg)	149.4±23.3[3]	154.5±21.7[3]	146.9±23.9[3]	154.6±20.6[3]	136.4±2.[3]	126±18[3]
Diastolic BP (mmHg)	79.7±12.7	81.3±15.6	78.8±11.0	81.7±13.1[3]	74.7±10.4[3]	73.72±9.9
%Body fat	38.0±7.2	43.4±6.7[3]	35.2±5.4[3]	40.4±0.7[3]	31.9±7.5[3]	31.1±6.5[3]
BMI	29.8±6.3[3]	30.5±5[3]	25.4±2.5[3]	30.8±6.0[3]	25±5.2[3]	23.7±2.8[3]
WHR	0.87±0.08[3]	0.91±0.09[3]	0.85±0.0±7[3]	0.91±0.07	0.81±0.07	0.80±0.06
Triglyceride (mmol/L)	2.0 (1.4-3.3)	3.4 (1.8-4.4)	1.9 (1.1-2.9)	2.9 (1.7-4.2)	1.1 (0.9-1.9)	0.98 (0.7-1.2)
HDL-C (mmol/L)	1.40±0.40[3]	1.25±0.3214[3]	1.48±0.427[3]	1.31±0.03843[3]	1.62±380[3]	1.83±0.47[3]
FBG (mmol/L)	5.8±1.4[3]	6.2±1.5[3]	5.5±1.3[3]	6.1±1.5[3]	5±0.4[3]	4.9±0.4[3]
Hs-CRP (mg/dL)	1.5 (0.49-4.47)	3.70(2.02-7.45)	0.8 (0.4-2.2)	2 (0.7-4.22)	0.61(0.21-3.10)	0.94 (0.19-2.13)
Serum Se (µmol/L)	1.07±0.25	1.04±0.26	1.09±0.24	1.06±0.24	1.11±0. 25	1.01±0..23
Serum GPx (U/mL)	0.30±0.10	0.27±0.07	0.32±0.10	0.30±0.10	0.30±0.07	0.35±0.12

Values expressed as Mean±SD or median and interquartile range. Comparisons between the healthy subjects and Clinic patients and between different sub-categories of the Clinic patients (obese and. non-obese; metabolic syndrome and non-metabolic syndrome) were analysed using one-way analysis of variance. Post-hoc, Dunnett tests were used after using one-way analysis of variance. Non-normally distributed data such as serum hs-CRP and triglycerides log transformed before using one-way analysis of variance. 1 = p<0.05, 2 = p<0.01 and 3 = p<0.001 compared to healthy subjects. * = p<0.05, ** = p<0.01 and *** = p<0.001 comparisons between different sub-categories of the Clinic patients (obese v. non-obese; metabolic syndrome and non-metabolic syndrome). BP = Blood pressure; HDL-C = High density lipoprotein cholesterol; FBG = Fasting blood glucose; Hs-CRP = High sensitivity C-reactive protein; Gpx = Glutathione peroxidase. WHR = waist hip ratio

body fat were also higher in the patients compared to the healthy subjects. Patients were on average 6.5 years older than healthy subjects.

Association between dietary selenium and their serum levels: The patients had a significantly higher dietary intake of total fat, protein and selenium compared with controls (p<0.05), but dietary intake did not differ between subgroups of patients with and without obesity or metabolic syndrome (p>0.05). A weak but significant association between dietary selenium intake and its serum levels were

Table 3: Clinical and biochemical characterisation of patients and healthy subjects (Males)

		---------- Clinic Patients ----------				
		---------- Obesity ----------		------ Metabolic syndrome ------		Healthy
Group	Total	Yes	No	Yes	No	subjects
Number	142	50	92	93	44	67
Age (years)	53.3±12.7	52.4±11.4	53.8±13.4	55.1±12.6	49.9±12.3	49±15.3
Current smokers (%)	16	25	15	16	19	16
Systolic BP (mmHg)	144.8±18.0	146.8±17.2	143.7±18.4	148.9±17.8	13.0±15.8	124.4±13.6
Diastolic BP (mmHg)	83.6±11.0	85.4±10.9	82.7±11	86.5±10.8	78.3±9.4	75.7±8.2
%Body fat	28.7±6.5	34.2±6	25.7±4.6	31.0±6	24.4±25.2	21.9±4.7
BMI	29.1±3.9	33.2±2.8	26.7±1.9	30.3±3.7	26.5±2.8	24.8±2.8
WHR	0.95±0.06	0.99±0.06	0.93±0.05	0.97±0.07	0.92±0.07	0.90±0.07
Triglycerides (mmol/L)	1.2 (0.9-1.4)	3.6 (2.2-6.4) [3]	2.6 (1.7-4.6)	2.9 (2.1-5.6)	2.4 (4.3-4.9)	1.1 (0.9-1.4)
HDL-C (mmol/L)	1.1±0.8	1±0.2	1.2±0.3	1.4±0.3	1.2±0.3	1.5±0.3
FBG (mmol/L)	6.0±1.7	6.5±2.1	5.6±1.3	6.3±1.8	5.4±1	5.0±0.4
Hs-CRP (mg/L)	1.1 (0.4-3.2)	2.0 (0.7-4.7)	0.8 (0.2-1.5)	1.7(0.5-3.4)	0.6 (0.2-1.9)	0.4 (0.2-0.9)
Serum Selenium (µmol/L)	1.08±0.22	1.05±0.22	1.10±0.22	1.09±0.23	1.06±0.22	1.05±0.23
Serum GPx (U/mL)	0.32±0.10	0.29±0.10	0.34±0.10	0.32±0.11	0.31±0.09	0.36±0.12

Values expressed as Mean±SD or median and interquartile range. Comparisons between the healthy subjects and Clinic patients and between different sub-categories of the Clinic patients (obese v. non-obese; metabolic syndrome and non-metabolic syndrome) were analysed using one-way analysis of variance, Post-hoc, Dunnett tests were used after using one-way analysis of variance. Non-normally distributed data, such as serum hs-CRP and triglycerides log transformed before using one-way analysis of variance, 1 = p<0.05, 2 = p<0.01 and 3 = p<0.001 compared to healthy subjects. * = p<0.05, ** = p<0.01 and *** = p<0.001 comparisons between different sub-categories of the Clinic patients (obese v. non-obese; metabolic syndrome and non-metabolic syndrome). BP = Blood pressure; HDL-C = High density lipoprotein cholesterol; FBG= Fasting blood glucose; Hs-CRP = High sensitivity C-reactive protein; Gpx = Glutathione peroxidase. WHR = waist hip ratio

Table 4: Daily dietary intake of patients and healthy subjects (Male and females)

		---------- Clinic Patients ----------				
		---------- Obesity ----------		------- Metabolic syndrome -------		Healthy
Group	Total	Yes	No	Yes	No	subjects
Number	238	82	156	142	96	135
Dietary Total Fat (g)	76.7±20.6	79±24.4	75.6±18.7	78.6±21.2	72±18.4	72.6±24.1
Dietary Protein (g)	88.1±25.2[1]	91.4±24.5[1]	85.5±25.5	91±25.8[1]	81.1.±22.5	80.9±21
Carbohydrate (g)	283.6±113.5	284±119.4	283.4±111.1	287±115.6	273.3±108.7	278.1±103.0
Dietary Selenium (µg)	106.1±60[1]	103.4±49.2	107.5±64.7	111.4±65[2]	93.3±43.9	84.8±40.1
Saturated Fat (g)	29.3±8.1	30.2±9	28.9±7.5	29.8±8	28±8.5	29.2±12.1
Cholesterol (mg)	412.7±101.9	439.5±133.7[1]*	400.0±80.5	424.4±110.7[1]*	384±69.4	391.9±94.9
Monounsaturated Fat (g)	27.7±7.9	28.3±9.2	27.4±7.3	28.3±8.2[1]	26.1±7.1	25.6±8.2
Polyunsaturated Fat (g)	10.9±3.4	10.9±3.7	11±3.3	11.3±3.7	10.1±2.6	10.2±3.3
Vitamin E (mg)	6.5±2.4	6.2±2.6	6.6±2.3	6.4±2.5	6.5±2.2	6.1±1.9
Vitamin C (mg)	76.7±13.2	66.7±12.2	81.4±13.6	79.9±14	69.2±10.9	53.5±67.56
Carotene (mg)	2268±1671	2258±1704	2272±1662	2231±1803	2356±1306	2079±936
Zinc (mg)	14.13±2.01[2]	14.04±2.24[2]*	14.17±1.90	14.15±2.01	14.08±2.02	13.69±2.30
Copper (mg)	1.78±0.81	1.84±0.87	1.76±0.78	1.84±0.83	1.64±0.74	1.70±0.65

Values expressed as Mean±SD. Comparisons between the healthy subjects and Clinic patients and between different sub-categories of the Clinic patients (obese and. non-obese; metabolic syndrome and non-metabolic syndrome) were analysed using one-way analysis of variance. Post-hoc, Dunnetttests were used after using one-way analysis of variance. 1 = p<0.05 and 2 = p<0.01 compared to healthy subjects. * = p<0.05 comparisons between different sub-categories of the Clinic patients (obese and. non-obese; metabolic syndrome and non-metabolic syndrome)

observed in the patients (r = 0.15, p = 0.04) and controls (r = 0.19, p = 0.02). The dietary macro- and micronutrient intake of the patients and healthy subjects is shown in Table 4.

Serum selenium and GPx concentrations: Overall, the patients had a significantly higher serum selenium level (p<0.05) and lower serum GPx (p<0.001) compared with controls (Table 1). Within the patient group, those who were obese had significantly lower serum selenium (p<0.05) and GPx (p<0.001) compared with those who were non-obese (Table 1). Serum selenium and GPx did not differ significantly in the patient subgroups with and without metabolic syndrome (p>0.05, Table 1).

Within the patient group as a whole, serum selenium concentrations decreased significantly with accumulating features of the metabolic syndrome (from 1.20±0.09 to 0.93±0.05, p<0.05). This remained the

case for the female patient subgroup (from 1.31±0.10 to 0.87±0.04 umol/L, p<0.01), but did not reach statistical significance for the male patient subgroup (p>0.05). Within the patient group, dietary intake of protein and zinc rose with accumulating features of the metabolic syndrome (p<0.05).

Discussion

We have found that patients attending a cardiovascular risk management clinic had significantly higher serum selenium levels and lower serum GPx concentrations compared to healthy subjects. On analysis of a dietary questionnaire, the patients appeared to have a significantly higher dietary intake of selenium compared to the healthy individuals. Although a large proportion of the patient group were on lipid lowering therapy, we have previously shown that statin therapy does not have any significant effect on serum selenium and GPx (Ghayour-Mobarhan *et al*., 2005c). The higher serum selenium concentrations in the clinic patients may be related to their higher dietary intake.

Consistent with a previous report (Gjorup *et al*., 1988), we found that obese patients had significantly lower serum selenium concentrations compared to non-obese patients. Also, as reported by Ozata *et al*., 2002 we found lower GPx concentrations in obese versus non-obese patients. Although we found no significant differences in serum selenium levels between patients with and without metabolic syndrome, which is consistent with other reports (Ford *et al*., 2003), we did find decreasing serum selenium concentrations were associated with increasing features of metabolic syndrome and this was independent of dietary selenium intake.

The limitation of this present study is its cross-sectional design; hence the associations that were revealed by the statistical analysis may not indicate causality. Estimates of dietary intake by FFQs may be associated with a number of intrinsic errors; however we have used a well validated questionnaire in samples of patients and healthy subjects of adequate sample size. Although some of the statistically significant differences observed may have arisen by chance, they remained after adjustment of possible confounding variables and were consistent with previous reports.

In conclusion, the lower levels of serum GPx in obesity and lower levels of serum selenium associated with accumulation features of metabolic syndrome may be related to the presence of an atherosclerosis prone state and consequential consumption of antioxidants by free radical interaction.

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