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Full Length Research Paper

Visceral adiposity in Saudi females and its relationship to diet and serum adipocytokine levels

Eman Mokbel Alissa[1]* and Hanan Ahmad AlKadi[2]

[1]Faculty of Medicine, Department of Clinical Biochemistry, King AbdulAziz University, Kingdom of Saudi Arabia.
[2]Faculty of Medicine, Department of Physiology, King AbdulAziz University, Kingdom of Saudi Arabia.

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To investigate the relationship between visceral fat, habitual dietary intake and circulating levels of three adipokines in Saudi females. A total of 127 apparently healthy female students were randomly recruited from Medicine College at KAU, Saudi Arabia. Anthropometric measurements were performed and a questionnaire was used to assess demographic variables, general health, medication use and dietary intake. Biochemical parameters were measured in fasting blood samples. Subjects were categorized into 2 subgroups according to presence or absence of visceral obesity, which was defined by a waist circumference (WC) value above 75th percentile. Significantly lower serum adiponectin ($p<0.01$) in addition to significantly higher serum lipid parameters, glucose, insulin ($p<0.0001$), leptin and dietary protein levels ($p<0.05$) were found among subjects with WC≥79 cm than those with WC<79 cm. Multiple regression analysis identified serum adiponectin (β= -0.71, P=0.013), leptin (β= 0.335, P=0.05) and daily intake level of adjusted protein (β= 2.817, P=0.011) as independent predictors for WC. Hypoadiponectemia, hyperleptinemia and high dietary protein level adjusted for total caloric intake are suggested to be associated with visceral fat among a group of Saudi females independent of body weight.

Key words: Visceral obesity, leptin, adiponectin, resistin, dietary intake, Saudi female.

INTRODUCTION

Obesity is a major risk factor for chronic diseases and plays a central role in the insulin resistance (IR) and metabolic syndrome (MetS); the latter comprises hyperinsulinemia, hypertension, hyperlipidemia, impaired glucose tolerance, and is associated with an increased risk of cardiovascular disease (CVD) (WHO, 2000). Hence obesity that often has its origins in childhood may precede the hyperinsulinemic state, and its increasing prevalence globally is likely to result in a substantial public health burden in the near future (Sinaiko et al., 2001).

In recent years it has become clear that the distribution of adipose tissue is an important determinant for the increased risk of CVD in obese people (Yusuf et al., 2005; Schneider et al., 2010). As an overall index of obesity, body mass index (BMI) cannot distinguish fat mass from muscle mass, nor can it reflect the relative fat distribution (visceral versus subcutaneous); hence, it may not be the most appropriate measure for predicting risk of diseases (Green, 2009). Waist circumference (WC) has been found to be a better anthropometric predictor of metabolic risk factors than BMI (Valsamakis et al., 2004). Skinfold thickness is also used as a measure of adiposity, but studies comparing its predictive ability to other measures of adiposity have produced inconsistent findings (Jones et al., 1992; Birmingham et al., 1993). An additional measure of adiposity is bioelectrical impedance analysis of the total body fat. However, whether it maintains stronger associations with CVD risk factors compared with simpler measures, such as BMI or WC, remains unknown (Nakanishi et al.,2000; Tseng, 2003).

Although largely influenced by genetic and environmental factors (Wellen and Hotamisligil, 2003),

*Corresponding author. E-mail: em_alissa@yahoo.com. Tel: (966) 2 6400000 Ext. 23432. Fax: (966) 2 66 434 99.

obesity is a very strong correlate of cardio-metabolic risk, especially in the presence of visceral obesity (Goodman et al., 2005). Whilst there is no consensus cutoff for WC, visceral obesity has been defined as a WC above the 75th percentile for age and gender in the population studied (Moreno et al., 2002; de Ferranti et al., 2004; Katzmarzyk et al., 2004).

The adipocyte-derived hormones resistin, adiponectin, and leptin may play a role in the development of hypertension, MetS, and IR, but the data in humans are equivocal, and nearly all studies have been done in adults (Steppan et al., 2001; Mukherjee et al., 2005). Although the increase of adipose tissue, particularly visceral fat mass, is considered a risk factor of IR, its secretory products (adipokines) are acquiring a recognized role as determinants of IR (Fruhbeck et al., 2001).

Several dietary factors have been linked to the aetiology of diabetes mellitus (DM), CVD and MetS. Thus dietary modification presents a powerful means by which to both prevent and manage MetS. Although the intake of various macronutrients such as carbohydrates, dietary fiber, and fat have been related to individual components of MetS (Aleixandre and Miguel, 2008; Skilton et al., 2008; Chen et al., 2009), the role of mixed diet in contributing to MetS is not well understood.

Previous evidence from intervention and observational studies on the dietary determinants of visceral adiposity is limited and conflicting (Halkjaer et al., 2006; Romaguera et al., 2009). However, little is known of the relationship between dietary factors and circulating adipokines concentrations in youth. Thus, the purpose of our study was to investigate the relationship between visceral fat, habitual dietary intake and circulating levels of three adipokines in young adult Saudi females.

METHODS

Study design and subjects

We have used a cross-sectional study design in which apparently healthy female medical students were randomly recruited from the college of Medicine at King Abdul Aziz University (KAU) in Jeddah, Saudi Arabia. Informed consent was obtained from all subjects and the study was approved by the Ethics committee of KAUH. A power calculation estimated that 120 subjects would be necessary for our study. One hundred and forty subjects were recruited. Of these 127 subjects satisfied the study criteria and were also willing to provide the required blood samples.

Exclusion criteria were inflammatory diseases, hepatic and renal diseases, impaired glucose tolerance or diabetes, and obesity secondary to genetic or metabolic disorders.

Anthropometric and blood pressure measurements

Anthropometric measurements were performed by a trained staff following standard operating procedures with height being measured without shoes to the nearest 0.1 cm using a rigid stadiometer and weight in light indoor clothing measured to the nearest 0.1 kg. WC was measured at the mid-point between the lower costal margin and the level of the anterior superior iliac crest to the nearest 0.1 cm. Hip circumference (HC) was measured as the maximal circumference over the buttocks. Blood pressure was measured twice with the participant in the sitting position and resting for 5 min.

Questionnaire and dietary assessment tool

Self-completed questionnaires were used to assess demographic variables, general health, medication use and history of any chronic illness, in the participant as well as in the family, including DM, hypertension, and CVD. A previously validated food frequency questionnaire (FFQ) was used to investigate the usual foods and beverages intake of the subjects in the past year (Alissa et al., 2005a). The FFQ included 93 different food items. The nutrient database used was based primarily on the UK food composition tables (McCance et al., 1991) supplemented by the food composition tables for use in East Asia (1972) and the US handbook of food composition (Watt and Merrill, 1963) and all were used for computing the nutrients. Measured dietary components were total caloric intake and micronutrients including fiber and cholesterol.

Biochemical measurements

Overnight fasting blood specimens were collected by venipuncture for measurement of serum lipids and plasma glucose. Blood specimens were centrifuged and plasma was stored at –80°C until laboratory assay.

Fasting plasma glucose, total cholesterol (TC), and triglycerides (TG) were assessed enzymatically using commercially available kits (Crescent Diagnostics, KSA). High-density lipoprotein (HDL) cholesterol was measured after dextran-magnesium precipitation. The concentration of low-density lipoprotein (LDL) cholesterol was calculated by using the Friedewald equation for participants with a TG level lower than 4.5 mmol/L. The intra-assay and inter-assay coefficients of variation for TC were 9.4 and 8.4% respectively, for HDL-C were 5.4 and 11.4% respectively, and for TG were 5.6 and 13.0% respectively.

The insulin resistance index was assessed by the homoeostasis model assessment score (HOMA-IR), calculated as the product of the fasting serum glucose in mmol/L and fasting insulin level in mU/L, divided by 22.5 (Matthews et al., 1985).

Serum adipokines levels

Serum leptin, adiponectin and resistin concentrations were evaluated in one run using a commercially available ELISA kits (ALPCO Diagnostics, Salem, NH, USA) with a detection limit of ≤0.5 ng/ml (leptin) and of ≤100 pg/ml (in adiponectin and resistin) according to the supplier's package insert. Determination of plasma insulin was carried out by a sandwich chemiluminescence immunoassay method using commercial kits (DiaSorin, Italy).

Statistical analysis

Continuous variables are presented as mean ± standard errors of mean for normally distributed variables and as median (inter-quartile ranges) for non-normally distributed variables. t-tests and Man- Whitney U-test were used for comparison of continuous variables that had normal and non-normal distribution respectively. The relationships between variables were assessed by Pearson correlation coefficient, or by Spearman's correlation for variables

Table 1. Demographic, anthropometric and biochemical characteristics in the study participants (n= 127) categorized according to the 75th percentile of waist circumference.

Variables	WC< 79 cm; n=95 (75%)	WC≥ 79 cm; n= 32 (25%)	*P*
Age (years)	20.28 ± 0.15	20.59 ± 0.34	NS
Weight (kg)	58.19 ± 1.13	87.83 ± 3.13	<0.0001
Height (cm)	158.0 ± 0.68	160.1 ± 1.05	NS
BMI (kg/m^2)	23.26 ± 0.41	34.29 ± 1.19	<0.0001
WC (cm)	66.42 ± 0.64	88.38 ± 2.13	<0.0001
HC (cm)	96.61 ± 0.79	116.4 ± 2.34	<0.0001
WHR	0.69 ± 0.0	0.76 ± 0.01	<0.0001
SBP (mmHg)	105.4 ± 1.23	112.6 ± 1.98	<0.01
DBP (mmHg)	70.96 ± 0.61	73.75 ± 1.32	NS
TC (mmol/L)	4.77 ± 0.08	5.48 ± 0.09	<0.0001
TG (mmol/L)	1.52 (1.25 – 1.64)	1.76 (1.61 – 1.84)	<0.0001
LDL-C (mmol/L)	3.42 ± 0.10	3.85 ± 0.13	<0.05
HDL-C (mmol/L)	1.21 (0.31 – 1.68)	1.52 (0.69 – 1.83)	NS
FBG (mmol/L)	4.80 ± 0.06	5.42 ± 0.11	<0.0001
Fasting insulin (µU/ml)	8.08 ± 0.40	16.65 ± 1.85	<0.0001
HOMA	1.74 ± 0.10	4.06 ± 0.44	<0.0001
Adiponectin (ng/ml)	9.75 (8.64 - 11.15)	9.08 (7.97 – 9.81)	<0.05
Leptin (ng/ml)	13.73 (11.68 – 16.55)	17.19 (13.49 – 20.15)	<0.05
Resistin (ng/ml)	13.49 ± 0.26	13.79 ± 0.27	NS

Normally distributed data presented as mean ± SEM. Continuous data were compared by unpaired t tests. BMI: body mass index, DBP: diastolic blood pressure, FBG: fasting blood glucose, HC: hip circumference, HDL-C: high density lipoprotein cholesterol, HOMA: homoeostasis model assessment, LDL-C: low density lipoprotein cholesterol, NS: not significant, TC: total cholesterol, TG: triglycerides, SBP: systolic blood pressure, SEM: standard error of mean, WC: waist circumference, WHR: waist hip ratio.

not normally distributed. Stepwise multiple regression analysis was used to determine a subset of independent predictors for WC. SPSS version 15.0 (SPSS, Inc. Chicago, IL) was used for statistical analysis; the significance level was set at $P < 0.05$.

RESULTS

The study population was categorized into 2 subgroups according to the presence or absence of visceral obesity, which was defined by a WC value above the 75th percentile.

Table 1 lists the demographic and anthropometric characteristics of individuals in the study. The study groups were of similar age ($p>0.05$). On average, weight, BMI, HC, WHR and SBP values were all statistically higher in individuals with WC≥79 cm than those with WC<79cm ($p<0.01$).

Biochemical parameters of study participants are summarized in Table 1. As would be expected, fasting lipid parameters, fasting glucose and insulin levels and HOMA-IR were significantly higher among subjects with WC≥79cm than those with WC<79cm ($p<0.0001$). Differences were also observed with respect to serum adiponectin ($p<0.01$) and serum leptin ($p<0.05$) but not serum resistin ($p>0.05$).

Concerning reported food intake, Table 2 presents the dietary characteristics of individuals in the study. Dietary intake was hypercaloric in both groups; the mean intake of macronutrients was within the acceptable range, except for total fat, saturated fatty acids (SFA), monounsaturated fatty acids (MUFA) and cholesterol, which exceeded the recommended limit. Conversely, dietary fiber and polyunsaturated fatty acids (PUFA) intakes were less than the recommended limit. Only the daily intake of protein either as crude intake, adjusted for energy level, or in the form of % of total caloric intake showed significant differences between both groups ($p<0.05$).

Table 3 shows analysis of associations between visceral obesity and various parameters for the entire population. Fasting serum adiponectin levels were inversely correlated with WC (Figure 1) and fasting serum leptin levels were positively correlated with WC (Figure 2). Resistin concentrations were not associated with WC in univariate analyses. WC was shown to be correlated with daily intake of proteins, SFA and cholesterol. These associations were no longer significant when assessed by multivariate analysis except for dietary protein.

Finally, multiple regression analysis of data, identified serum adiponectin ($\beta= -0.71$, $P=0.013$), serum leptin ($\beta= 0.335$, $P=0.05$) and daily intake level of adjusted protein ($\beta= 2.817$, $P=0.011$) as independent predictors for WC in

Table 2. Daily intake levels of dietary nutrients of the study participants (n= 127) categorized according to the 75th percentile of waist circumference.

Variables	RNI	WC<79 cm; n=95 (75%)	WC≥79 cm; n= 32 (25%)	*P*
Energy (kcal)	1940 (15-18year)	2182.6	2378.0	NS
	1900 (19-59 year)	(1600.2-2741.1)	(1923.5-2807.0)	
Unadjusted protein (g)		80.78 ± 2.72	91.99 ± 4.23	<0.05
Energy-adjusted protein (g)		82.78 ± 0.10	83.19 ± 0.16	<0.05
% of energy	15%	14.68 ± 0.22	15.64 ± 0.35	<0.05
Unadjusted carbohydrates (g)		269.65 ± 7.65	286.54 ± 15.4	NS
Energy-adjusted carbohydrates (g)		260.68 ± 0.10	260.67 ± 0.19	NS
% of energy	55%	49.59 ± 0.65	48.36 ± 1.18	NS
Unadjusted total fat(g)		88.47 ± 3.44	93.33 ± 4.21	NS
Energy-adjusted total fat (g)		84.87 ± 0.10	84.75 ± 0.19	NS
% of energy	30%	35.73 ± 0.64	36.00 ± 1.08	NS
Cholesterol (mg)	200 mg	273.85 ± 13.76	280.92 ± 23.64	NS
Unadjusted SFA (g)		33.02 ± 1.37	36.61 ± 2.06	NS
Energy-adjusted SFA (g)		32.86 ± 0.10	32.97 ± 0.19	NS
% of energy	10%	14.79 ± 0.33	15.56 ± 0.61	NS
Unadjusted MUFA (g)		31.29 ± 1.34	33.09 ± 1.73	NS
Energy-adjusted MUFA (g)		29.54 ± 0.10	29.44 ± 0.20	NS
% of energy	10%	13.97 ± 0.31	14.22 ± 0.60	NS
Unadjusted PUFA (g)		14.57 ± 0.62	14.70 ± 0.60	NS
Energy-adjusted PUFA (g)		13.60 ± 0.11	13.36 ± 0.14	NS
% of energy	10%	6.61 ± 0.17	6.38 ± 0.22	NS
Fiber	18 gm	15.70 ± 0.77	14.97 ± 0.94	NS

Normally distributed data presented as mean ± SEM and non-normally distributed data presented as median (inter-quartile range). Continuous data were compared by unpaired t tests or Mann-Whitney tests for non-normally distributed data. MUFA: monounsaturated fatty acids, NS: not significant, PUFA: polyunsaturated fatty acids, RNI: recommended nutritional intake, SEM: standard error of mean, SFA: saturated fatty acids, WC: waist circumference.

Table 3. Correlation coefficients between visceral obesity and various parameters for the study participants (n= 127). Pearson's or Spearman's correlation coefficients were calculated for normally distributed data and non-normally distributed data respectively.

Variables	r	*P*
Age (years)	0.200	<0.05
Weight (kb)	0.896	<0.0001
Height (cm)	0.203	<0.05
BMI (kg/m^2)	0.892	<0.0001
HC (cm)	0.907	<0.0001
WHR	0.717	<0.0001
SBP (mmHg)	0.316	<0.01
DBP (mmHg)	0.283	<0.01
TC (mmol/L)	0.452	<0.0001
TG (mmol/L)	0.325	<0.0001
LDL-C (mmol/L)	0.237	<0.0001
HDL-C (mmol/L)	0.145	NS
FBG (mmol/L)	0.419	<0.0001
Fasting insulin (µU/ml)	0.542	<0.0001
HOMA	0.564	<0.0001
Adiponectin (ng/ml)	-0.267	<0.01
Leptin (ng/ml)	0.296	<0.01

Table 3. Contd.

Resisitin (ng/ml)	0.066	NS
Energy (kcal)	0.142	NS
Unadjusted protein (g)	0.264	<0.01
Energy-adjusted protein (g)	0.291	<0.01
% of energy from protein	0.281	<0.01
Unadjusted carbohydrates (g)	0.067	NS
Energy-adjusted carbohydrates (g)	- 0.112	NS
% of energy form carbohydrates	- 0.160	NS
Unadjusted total fat(g)	0.121	NS
Energy-adjusted total fat (g)	0.125	NS
% of energy from total fat	0.074	NS
Cholesterol (mg)	0.176	<0.05
Unadjusted SFA (g)	0.177	<0.05
Energy-adjusted SFA (g)	0.036	NS
% of energy from SFA	0.017	NS
Unadjusted MUFA (g)	0.122	NS
Energy-adjusted MUFA (g)	- 0.120	NS
% of energy from MUFA	- 0.042	NS
Unadjusted PUFA (g)	0.143	NS
Energy-adjusted PUFA (g)	- 0.065	NS
% of energy from PUFA	- 0.052	NS
Fiber	- 0.018	NS

BMI: body mass index, DBP: diastolic blood pressure, FBG: fasting blood glucose, HC: hip circumference, HDL-C: high density lipoprotein cholesterol, HOMA: homoeostasis model assessment, LDL-C: low density lipoprotein cholesterol, MUFA: monounsaturated fatty acids, NS: not significant, PUFA: polyunsaturated fatty acids, SBP: systolic blood pressure, SFA: saturated fatty acids, TC: total cholesterol, TG: triglycerides, WC: waist circumference, WHR: waist hip ratio.

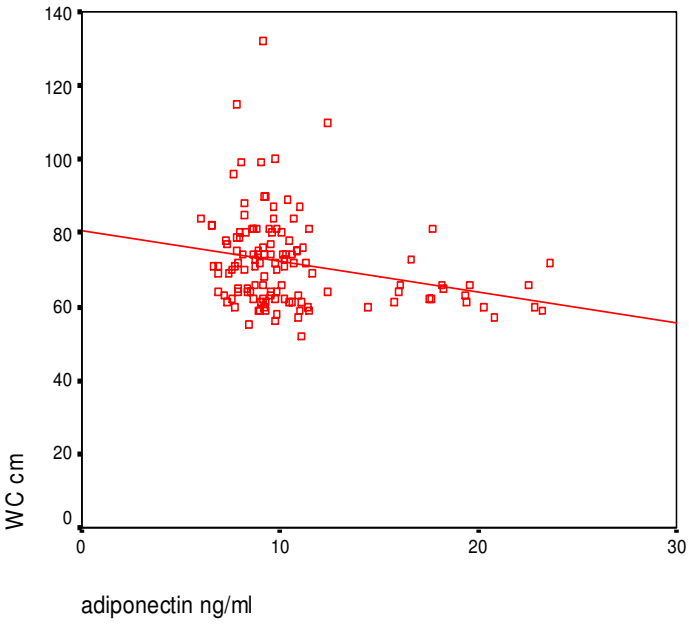

Figure 1. Scatter plot demonstrating the correlation between waist circumference and serum adiponectin concentration among the whole population (r = -0.279, p <0.01).

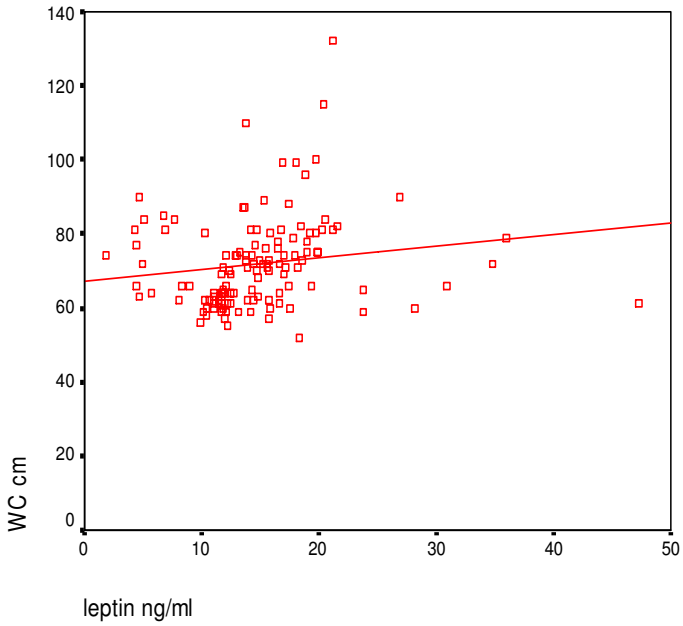

Figure 2. Scatter plot demonstrating the correlation between waist circumference and serum leptin concentration among the whole population (r = 0.296, p <0.01).

Table 4. Multiple regression analysis between visceral obesity and independent predictors in the study participants (n= 127).

Independent predictors	β	*P*	95% CI for β (lower limit-upper limit)	
Adiponectin	- 0.710	0.013	-1.27	-0.15
Leptin	0.335	0.05	0.0	0.67
Energy adjusted protein	2.817	0.011	0.66	4.97

95% CI: 95% confidence interval.
Total $R^2 = (0.384)^2 = 14.8$ %.

Table 5. Correlation coefficients between the three adipokines with dietary factors in the study participants (n= 127). Pearson's or Spearman's correlation coefficients were calculated for normally distributed data and non-normally distributed data respectively.

Adipokines	Dietary factors	r	*P*
Adiponectin	Energy adjusted protein	-0.264	<0.01
	% of energy from protein	-0.251	<0.01
Leptin	Unadjusted SFA	0.203	<0.05
Resistin	% of energy from total fat	0.215	<0.05
	% of energy from SFA	0.178	<0.05

SFA: saturated fatty acids.

the study population (Table 4).

Table 5 displays correlations of the three adipokines with dietary factors irrespective of visceral fat. Overall, statistically significant associations among the total cohort were only observed: between serum adiponectin and mean proteins either adjusted for energy level or as % of total energy intake, between serum leptin and crude intake of SFA, between serum resistin and % energy from both of total fat and SFA.

DISCUSSION

Obesity is a multi-factorial condition that may influence sensitivity to insulin (Solga et al., 2004). Differences in lifestyle behaviors, in particular, dietary habits may play a role in determining sensitivity to insulin. There are major gaps in knowledge linking the intake of some macro-nutrients with the prevention and treatment of chronic diseases like MetS, DM.

The purpose of our study was to examine the inter-relationship between habitual dietary intake, circulating adipocytokine levels and visceral fat in young adult Saudi females.

As mentioned earlier, there is no consensus cutoff for WC; however, visceral obesity has been defined as a WC above the 75th percentile for age and gender in the population studied (Moreno et al., 2002; de Ferranti et al., 2004; Katzmarzyk et al., 2004). As expected, clinical and biochemical parameters were less favorable in subjects with visceral obesity. Concordantly, WC has been positively associated with anthropometric indices, blood pressure elevation, fasting glucose and lipid abnormalities in our study subjects. Population-based studies concerning the correlation between anthropometric indices and risk factors for chronic disease in adults have documented that visceral fat confers increased risk of metabolic complications such as dyslipidemia, high fasting glucose, and high blood pressure (Yusuf et al., 2003; Katzmarzyk et al., 2004; Valsamakis et al., 2004).

As far as dietary habits are concerned, our entire population of young adult females reported a trend to higher consumption of total energy intake, total fat, and saturated fat, a pattern consistent with a Western diet, as reported previously (Alissa et al., 2005b). A Western diet is high in saturated fat, simple sugars, and cholesterol, and is associated with several metabolic abnormalities; including IR and inflammation, the initiating factors to the development of MetS (Bullo et al., 2007). Over the last few decades, tremendous urbanization and economic development has led KSA into a stage of the nutrition transition defined by the adoption of western lifestyle and a high prevalence of non-communicable diseases which has greatly increased concerns for public health (Reddy, 2002).

WC association with dietary levels of SFA, cholesterol and protein (expressed either as unadjusted, adjusted for caloric intake or as a percentage of total energy intake) are in accordance with the unequivocal role of high fat diet as the main environmental trigger for visceral obesity (Togashi et al., 2010). Novel finding of this study include the predictive role of hypoadiponectinemia,

hyperleptinemia and high dietary protein level adjusted for total caloric intake on WC. Both leptin and adiponectin have been independently shown to be associated with WC, albeit in opposite directions (Moran and Phillip, 2003; Shargorodsky et al., 2009). However, a clear functional link between these two hormones has yet to be extensively described, although suggested.

Adiponectin, leptin, and resistin are adipokines that have been suggested to play a role in regulating energy homeostasis (Wozniak et al., 2009). Hormonal and nutritional factors have been proposed to influence their concentrations in humans (Pittas et al., 2004).

It has been reported that serum leptin, which has an important role in the regulation of appetite and energy balance, is increased in obese subjects and correlates with the percentage of body fat (Considine et al., 1996). In this study in female young adults, serum leptin levels were found to be associated with weight ($r=0.290$, $p<0.001$) and BMI values ($r=0.300$, $p<0.001$). A suspected role for leptin has been proposed in the aetiology of IR and DM (Cohen et al., 1996, Segal et al., 1996) whereas reduced adiponectin concentrations are suggestive to be associated with high risk of obesity, MetS, DM, and CVD (Weyer et al., 2001; Kumada et al., 2003; Kadowaki et al., 2006). Our findings revealed that individuals with high WC values had lower serum adiponectin and higher serum leptin levels than those of normal WC.

The inverse association between waist and adiponectin are already established and are thought to remain constant over adulthood (Lindsay et al., 2002). In agreement with this fact, serum adiponection levels were found to be inversely associated with weight ($r= -0.200$, $p<0.05$) and BMI values ($r= -0.196$, $p<0.05$) among the study subjects.

Unlike most other adipocyte-derived hormones, adiponectin, which has anti-inflammatory and cardio-protective properties, exerts prominent effects on lipid profiles and might play a protective role in the development of IR and DM (Spranger et al., 2003). Our results indicate such a potential role as we found a positive association between fasting serum levels of adiponectin and HDL-C ($r= 0.217$, $p<0.05$) and a negative association with LDL-C ($r= -0.188$, $p<0.05$).

Further, serum concentrations of leptin have been associated positively with IR, whereas adiponectin is associated negatively (Yannakoulia et al., 2003; Cambuli et al., 2008). Given the potential role of insulin in the regulation of circulating leptin concentrations (Pratley et al., 2000), it would be plausible to explain the association between insulin and leptin ($r=0.311$, $p<0.00001$), in particular, in our population. However, the relationship between serum resistin and IR, DM and visceral obesity remains controversial (Steppan et al., 2001; Utzschneider et al., 2005; Burnett et al., 2006). Although our study showed weak association ($r=0.187$, $p<0.05$) between serum resistin and IR (as estimated by HOMA), the role of insulin in the regulation of circulating resistin is still subject to controversy (Janke et al., 2002; McTernan et al., 2002).

Given the role fat mass and distribution as well as total energy and macronutrient intake play in both obesity and IR, the identification of dietary factors that are associated with circulating adiponectin, resistin, and leptin levels could be of clinical importance. There are few studies on dietary determinants of blood pressure, IR, and the three adipokines concentrations in normal and overweight individuals, and their data are inconsistent (Vessby et al., 2001). Despite documented beneficial effects of high protein diet on weight loss and blood lipids (Ouellet et al., 2007), it has been shown recently to increase insulin resistance and diabetes risk (Weickert et al., 2011). The positive effects of dietary protein against the development of insulin resistance in experimental animals have been attributed to fish protein (Lavigne et al., 2001). However, relationship between these adipokines with dietary protein level remains to be clarified.

Cross-over design studies have shown that diets higher in SFA reduce insulin sensitivity more so than do diets higher in other types of FA (Vessby et al., 2001). Interestingly, serum leptin concentration was found to be correlated with intake level of SFA in the whole population of female young adults. Although a Western dietary pattern (Fung et al., 2001) and high-fat diet (Cooling et al., 1998) have been positively associated with leptin concentrations, negative associations have been shown in other studies in women (Havel et al., 1999). The above conflicting data may reflect variations in the study design and experimental conditions, but more importantly they may reflect the lack of adjustment for potential confounding factors, i.e. gender, body mass.

In contrast to leptin, limited data are available regarding the relationships between dietary factors and serum adiponectin levels, and none regarding serum resistin in humans.

Fasting serum adiponectin level was found to be adversely correlated with dietary protein level (adjusted for caloric intake or as a percentage of total energy intake) in our population of healthy young adult females. These associations are in accordance with the proposed role of this hormone as mediators of visceral obesity and IR (Lihn et al., 2005).

Resistin is a protein involved in glucose homeostasis, lipid metabolism (Rajala et al., 2003). The weak association between serum resistin and total fat and SFA in our study is intriguing and deserves some remarks. However, the evidence for serum resistin role as a metabolic or lipid marker is less consistent with some studies reporting a positive association (Azuma et al., 2003) and others finding no relation (Reilly et al., 2005). Further interventional studies controlling for potential confounding factors and focusing on the role of these hormones in mediating the effect of visceral obesity on IR are warranted.

There are several study limitations. This study uses a

cross-sectional design, and hence cannot explain causality but only association between variables. Furthermore, although we adjusted for potential confounders, the existence of residual confounding is always a limitation in observational studies. Also, FFQ like any other dietary assessment instrument are subjected to reporting errors such as under-reporting of energy intake. This could be due to the tendency of subjects to conceal their true intake in case of obesity. Finally, the present findings may only be applicable to Saudi young adult overweight/obese females.

In conclusion, the present study suggests that hypoadiponectemia, hyperleptinemia and high dietary protein level adjusted for total caloric intake are associated with visceral fat among a group of young adult Saudi females independent of body weight, a finding consistent with our hypothesis. Although no associations were observed with resistin in the cohort as a whole, we did detect a relationship with insulin sensitivity. Simple dietary and physical activity modifications are believed to have positive effects on metabolic and clinical parameters in overweight/obese young adults. Our findings provide an initial step towards examining the relationship between dietary factors and adipokines profile with MetS as the main outcome in future youth studies.

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