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# Relationships of Leptin and Anthropometry, Physical Work Capacity, Metabolic Syndrome in Chinese Postmenopausal Women

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**ABSTRACT**

Postmenopausal women gain abdominal and visceral fat during the menopausal period. Leptin is an adipocyte-secreted hormone that is involved in metabolic disturbance disease. However, few studies have investigated the associations between leptin and metabolic syndrome (MS) in Chinese postmenopausal women. The purpose of this study was to examine the relationships of serum leptin and anthropometry, physical work capacity and MS in sixty Chinese postmenopausal women. Factor analysis extracted five factors characterized by leptin and obesity, muscle mass and physical work capacity, blood pressure and atherosclerosis, cardiovascular risk marker, and cholesterol, which accounted for 78.38% of the total variance. Leptin correlated positively with waist-to-hip ratio, body mass index and systolic blood pressure, and correlated negatively with physical work capacity of 75% heart rate max ($R^2 = 0.805$, $p < 0.001$). Leptin levels in MS subjects with central obesity were higher than in both non-MS subjects ($p < 0.01$) and MS subjects without central obesity ($p < 0.05$). Subjects with a high leptin level had higher risks of the development of MS with central obesity than non-MS subjects (OR = 16.00, 95% confidence interval (CI): 3.15-51.26, $p < 0.01$), and MS subjects without central obesity (OR = 16.00, 95% CI: 1.69-81.53, $p < 0.05$). In conclusion, serum leptin can be predicted by waist-to-hip ratio, BMI, physical work capacity, and systolic blood pressure. In addition, a high leptin level increases the risk of MS with central obesity.

***Key words:*** *Leptin, Anthropometry, Metabolic syndrome, Postmenopausal*

Leptin is an adipocyte-secreted hormone that acts on the central nervous system to play a key role in energy homoeostasis[7)]. Leptin is initially considered for treatment for obesity because of its function of reducing food intake and body weight[24)]. However, obese individuals often have increased leptin levels, interpreted as leptin resistance, and leptin administration shows very limited effects[25)].

Although leptin levels are positively correlated with the percentage of body fat[10)], other factors such as gender, age, and sex hormones may also be relevant. Gender difference in leptin levels is evident in women having 2-3 fold higher leptin levels than men due to the distinction of endocrine involvement[18)]. The influence of aging on leptin levels remains controversial, as previous studies have shown that leptin levels are reduced[14)], left unchanged[10)], or increased during aging[4)]. For women, going through the menopausal transition is accompanied by marked sex hormonal alterations, and gains in body fat mass and abdominal visceral fat deposition[33)]. Leptin levels increase from the premenopause stage to the postmenopause stage with increases in follicle-stimulating hormone concentrations in non-obese women, though this pattern is not observed in obese women, who have consistently higher leptin levels at each stage[34)].

In postmenopausal women, the increase in visceral adiposity is positively correlated with leptin levels[20)]. Moreover, an elevated leptin level is predictive of insulin resistance and may relate to metabolic syndrome (MS)[9)]. MS is a cluster of metabolic disturbances, including central obesity, insulin resistance, impaired glucose tolerance, hypertension, and dyslipidemia. MS represents a leading risk factor for cardiovascular disease and

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a significant cause of morbidity and mortality[26]. The prevalence of MS is 22.6% in women, but increases with age, being 40-50% in postmenopausal women[23]. Thus prevention of MS in postmenopausal women has become a major public health issue.

Leptin may be a useful biomarker for predicting MS, but the association between serum leptin levels and MS in Chinese postmenopausal women has never been reported. Additionally, determination of circulating leptin is not applied in clinical practice in China. Previous studies have suggested that the serum leptin level can be predicted by anthropometric and blood physiological measurements in obese patients[1]. Only a few studies have examined the association of leptin levels and physical work capacity[27].

The purpose of this study was to investigate the relationships of serum leptin and anthropometry, physiological measurements, physical work capacity, and MS in Chinese postmenopausal women.

## MATERIALS AND METHODS

### *Subjects*

Sixty Chinese postmenopausal women aged 66 ± 4 years (range, 60-72 years) volunteered to participate in this study. The study was approved by the institutional review board at Shenyang Sport University and written informed consent was obtained. Data including demographic variables, health status, and medical histories were collected using a face-to-face interview method by a trained researcher.

### *Anthropometric measurements*

Measurements were performed in a research laboratory. After 10 min of rest, systolic blood pressure (SBP) and diastolic blood pressure (DBP) were taken from the right arm with subjects in a sitting position. Weight (nearest 0.1kg) was measured with subjects in their underwear using a calibrated electronic scale. Height (nearest 0.1 cm) was measured using a height board. Body mass index (BMI) was calculated as weight/height$^2$ (kg/m$^2$). Waist circumference was measured as the minimum value between the iliac crest and the lateral costal margin. Hip circumference was measured at the level of the maximum posterior protrusion of the buttocks. Waist-to-hip ratio was calculated as waist/hip (cm/cm). Body composition was determined with a body composition analyzer (Zeus 9.9 Body Composition Analyzer, Jawon Medical, Korea).

### *Physiological measurements*

A blood sample was drawn in the morning after a 12 hr overnight fast. Fasting blood glucose (FBG) was directly measured with an enzymatic method to screen for diabetes mellitus (LifeScan SureStep® Blood Glucose Monitoring System, LifeScan, USA). Each blood sample was centrifuged at 3000 rpm for 15 min and the serum sample was immediately stored at -70°C. Serum lipids including triglycerides, total cholesterol, high-density lipoprotein cholesterol (HDL cholesterol), and low-density lipoprotein cholesterol (LDL cholesterol) were determined using standard methods in a hospital laboratory. Serum leptin was determined using radioimmunoassay (Linco Research, St Charles, MO, USA) with an intra-assay coefficient of variation 4.7% and an inter-assay coefficient of variation of 5%. The limit for sensitivity was 0.1 ng/ml.

Brachial-ankle pulse wave velocity, an atherosclerosis indicator, was measured in the laboratory by a VP-1000 device (Omron BP-203RPE III Non-invasive Vascular Screening Device, Colin Corporation, Japan). The details of methodology have been described elsewhere[36].

### *Physical work capacity*

The physical work capacity of 75% heart rate max (PWC75%HRmax) was determined on a bicycle ergometer (Monark 839E, Stockholm, Sweden). After driving the ergometer at 0 W as a warming up, each subject pedaled at 50 rpm with an increment of 1 W every 3 sec to achieve 75% maximal age-predicted heart rate. Maximal heart rate was calculated by 220 minus age. Heart rate was monitored by telemetry (Polar S-610 Heart Rate Telemetry, Polar Electro Oy, Finland).

### *Definition of metabolic syndrome*

Using modified National Cholesterol Education Program Adult Treatment Panel III (NCEP ATP III) criteria[13], MS was defined as the presence of three or more of the following items: (1) Central obesity: Waist circumference ≥ 80 cm (for Asian women); (2) FBG: ≥ 5.60 mM or previously diagnosed type 2 diabetes; (3) Hypertension: SBP/DBP ≥ 130/85 mmHg or on antihypertensive medication; (4) Triglycerides: ≥ 1.70 mM or treatment for this abnormality; (5) HDL cholesterol: <1.29 mM or treatment for this abnormality.

### *Statistical analysis*

A One-sample Kolmogorov-Smirnov test was used to examine whether variables were normally distributed. Factor analysis using a principal axis factoring method with an analyzing correlation matrix and a promax rotation was performed to extract factors when eigenvalues exceeded 1.0. Backward linear regression analysis was fitted to predict serum leptin concentration. Mean differences of serum leptin among the three categories of MS with central obesity, MS without central obesity, and non-MS were tested by GLM Univariate Analysis, and post-hoc tests were done

using the Bonferroni method. Serum leptin levels were classified into the two same sample size categories: the high level (> 8.6 ng/ml), and the low level (≤ 8.6 ng/ml). Chi square test was performed for comparing frequencies of MS and serum leptin levels. Binary logistic regression analysis was used to evaluate the relationships among the serum leptin levels, the three categories of MS with central obesity, MS without central obesity, and the non-MS. All data were analyzed using SPSS 16.0 (Chicago, IL, USA).

## RESULTS

### *Characteristics of the subjects*

Table 1 summarizes the anthropometry, physiological measurements, physical work capacity and serum leptin for the subjects.

### *Relationships of serum leptin and the multiple indicators*

Table 2 presents the results of factor analysis. Five factors were extracted, accounting for 78.38% of the total variance. The first factor represented leptin and obesity, which showed positive loadings of leptin, body fat mass, percentage of body fat, waist circumference, waist-to-hip ratio and BMI. The second factor was characterized by muscle mass and physical work capacity, with positive loadings of BMI, fat free mass, skeletal muscle mass and PWC75%HRmax. These two factors correlated significantly ($r$ = 0.474, p < 0.01). Blood pressure and atherosclerosis indicators typified the third factor with positive loadings of SBP, RbaPWV, LbaPWV and fasting blood glucose. The fourth factor was defined as cardiovascular risk marker (triglycerides-to-HDL cholesterol ratio[6]), which included positive loading of fasting blood glucose, triglycerides and negative loading of HDL cholesterol. The fifth factor was named cholesterol, with positive loading of LDL cholesterol and total cholesterol (Table 2).

Table 3 shows the results of backward linear regression analysis. Waist-to-hip ratio, BMI, PWC75%HRmax and SBP explained 80.5% of the total variation of serum leptin (p < 0.001); Serum leptin concentrations correlated positively with waist-to-hip ratio, BMI, and SBP, and correlated negatively with PWC75%HRmax (all p < 0.05).

**Table 1.** Characteristics of the sixty Chinese postmenopausal women

| Parameters | Mean ± SD |
|---|---|
| Age (years) | 66 ± 4 |
| BMI (kg/m$^2$) | 24.6 ± 2.7 |
| Waist circumference (cm) | 91.4 ± 3.4 |
| Waist-to-hip ratio (cm/cm) | 0.94 ± 0.04 |
| Body fat mass (kg) | 20.1 ± 4.6 |
| Fat free mass (kg) | 38.5 ± 3.6 |
| Skeletal muscle mass (kg) | 19.9 ± 2.5 |
| Percentage of body fat (%) | 34.0 ± 4.1 |
| SBP (mmHg) | 135.0 ± 24.1 |
| DBP (mmHg) | 74.0 ± 8.4 |
| PWC75%HRmax (W) | 61.4 ± 21.1 |
| Total cholesterol (mM) | 5.2 ± 0.9 |
| HDL cholesterol (mM) | 1.2 ± 0.2 |
| LDL cholesterol (mM) | 2.9 ± 0.9 |
| Triglycerides (mM) | 1.7 ± 1.2 |
| Fasting blood glucose (mM) | 5.3 ± 0.8 |
| Leptin (ng/ml) | 9.1 ± 2.8 |
| RbaPWV (m/s) | 16.1 ± 2.9 |
| LbaPWV (m/s) | 16.0 ± 2.9 |

Notes. PWC75%HRmax: Physical work capacity of 75% heart rate max;
RbaPWV: Right brachial-ankle pulse wave velocity;
LbaPWV: Left brachial-ankle pulse wave velocity.

**Table 2.** The results of factor analysis using the data of serum leptin and the multiple variables

| Parameters | Factors | | | | |
|---|---|---|---|---|---|
| | 1st | 2nd | 3rd | 4th | 5th |
| Total variance explained (%) | **31.64** | **19.30** | **10.82** | **10.01** | **6.61** |
| Serum leptin | **0.79** | 0.19 | 0.25 | 0.05 | -0.11 |
| Body fat mass | **0.95** | 0.71 | 0.05 | 0.07 | -0.17 |
| Percentage of body fat | **0.96** | 0.41 | 0.13 | 0.13 | -0.09 |
| Waist circumference | **0.89** | 0.37 | 0.17 | 0.13 | -0.19 |
| Waist-to-hip ratio | **0.95** | 0.42 | 0.29 | 0.19 | -0.26 |
| BMI | **0.87** | **0.80** | 0.07 | 0.14 | -0.21 |
| Fat free mass | 0.46 | **0.98** | -0.12 | 0.02 | -0.18 |
| Skeletal muscle mass | 0.46 | **0.97** | -0.12 | 0.03 | -0.18 |
| PWC75%HRmax | 0.20 | **0.54** | -0.16 | 0.17 | -0.15 |
| DBP | 0.05 | 0.28 | 0.40 | 0.17 | 0.17 |
| SBP | 0.21 | 0.00 | **0.87** | 0.23 | -0.17 |
| RbaPWV | 0.17 | -0.30 | **0.90** | -0.04 | -0.33 |
| LbaPWV | 0.15 | -0.26 | **0.88** | 0.03 | -0.37 |
| Fasting blood glucose | 0.16 | -0.04 | **0.63** | **0.51** | -0.08 |
| Triglycerides | -0.09 | -0.08 | 0.18 | **0.90** | -0.22 |
| HDL cholesterol | -0.22 | -0.27 | -0.01 | **-0.70** | 0.11 |
| LDL cholesterol | -0.12 | -0.14 | -0.22 | -0.14 | **0.98** |
| Total cholesterol | -0.35 | -0.33 | -0.28 | -0.07 | **0.87** |

Notes. PWC75%HRmax: Physical work capacity of 75% heart rate max; Heart rate max = 220-age; RbaPWV: Right brachial-ankle pulse wave velocity; LbaPWV: Left brachial-ankle pulse wave velocity.
Factor analysis using the principal axis factoring method with an analyzing correlation matrix and a promax rotation was performed to extract factors when eingenevalues exceed 1.0. Factor loading of an absolute magnitude of greater than 0.5 was used for interpretation. Five factors account for 78.38% of the total variance.

**Table 3.** The results of regression analysis for serum leptin concentrations with multiple indicators

| Predictors | $\beta$-coefficient ± S.E. | Standardized $\beta$-coefficient | t | p |
|---|---|---|---|---|
| Waist-to-hip ratio | 89.54 ± 16.68 | 0.69 | 5.37 | 0.000 |
| PWC75%HRmax | -0.12 ± 0.02 | -0.52 | -7.50 | 0.000 |
| BMI | 0.45 ± 0.23 | 0.26 | 2.00 | 0.039 |
| SBP | 0.03 ± 0.01 | 0.15 | 2.05 | 0.046 |
| $R^2$ | 0.805 | Adjusted $R^2$ | | 0.787 |

Notes. PWC75%HRmax: Physical work capacity of 75% heart rate max;
Heart rate max = 220-age; F = 44.50, p = 0.000.

***Relationships of serum leptin and MS***

Figure 1 shows the result of the GLM Univariate Analysis. Serum leptin levels were different among the categories of non-MS, MS without central obesity, and MS with central obesity ($F$ = 6.75, p = 0.002). Serum leptin levels in the subjects of MS with central obesity were significantly higher than in both the subjects of non-MS ($p < 0.01$) and the subjects of MS without central obesity ($p < 0.05$).

Subjects with a high leptin level (> 8.6 ng/ml) had a higher prevalence of MS than those with a low level (≤ 8.6 ng/ml) (60% (18/30) versus 20% (6/30), $\chi^2$ =10.00, $p < 0.05$). Binary logistic regression analysis estimated an odds ratio (*OR* = 6.00, 95% confidence interval (CI): 1.89-19.03, $p < 0.01$), which demonstrated that the risk of subjects with a high leptin level of the development of MS was 6 times greater than that of those with a low leptin level. Subjects with a high leptin level had higher risks of the development of MS with central obesity than those of the non-MS (OR = 16.00, 95% CI : 3.15-51.26, $p < 0.01$), and the MS without central obesity (OR = 16.00, 95% CI: 1.69-81.53, $p < 0.05$).

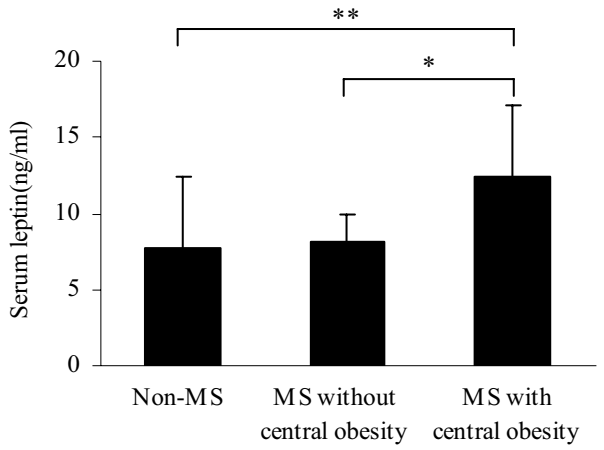


**Fig. 1.** Serum leptin concentrations in the categories of non-MS, MS without central obesity, and MS with central obesity

Notes. MS: metabolic syndrome; Central obesity: Waist circumference ≥ 80 cm for Asian women. Data are shown as Mean + SD; Differences of mean were tested by GLM Univariate Analysis (F = 6.75, $p < 0.01$); **: $p < 0.01$, significant difference between the MS with central obesity and the non-MS; *: $p < 0.05$, significant difference between the MS with central obesity and the MS without central obesity.

## DISCUSSION

To the best of our knowledge, this is the first study to investigate the relationships of serum leptin and anthropometry, physical work capacity, and MS simultaneously. The study showed that serum leptin concentrations could be predicted by waist-to-hip ratio, BMI, PWC75%HRmax and SBP in Chinese postmenopausal women. Additionally, a high serum leptin level was shown to increase the risk of the development of MS with central obesity.

Obesity parameters have strongly positive correlations with serum leptin levels. Our results showed that serum leptin and obesity parameters (percentage of body fat, body fat mass, waist-to-hip ratio, waist circumference, and BMI) loaded the first key factor which contributed 31.64% of total variance. Among these obesity parameters, waist-to-hip ratio and BMI were selected by backward linear regression model to predict serum leptin concentration. In particular, waist-to-hip ratio, a marker of abdominal obesity, was the most important predictor that showed the largest magnitude of standardized coefficient. These results are consistent with previous research studies which have demonstrated that circulating leptin levels are highly correlated with central located body fat in postmenopausal women[21,29].

Physical work capacity is correlated with circulating leptin levels. Our results indicated that PWC75%HRmax was the second most important determinant of serum leptin concentration and had a significantly negative correlation. Physical work capacity is proportional to exercise efficiency[2]. Exercise, with associated changes in energy expenditure, fuel flux and systemic hormone concentrations, may also contribute to leptin regulation[19]. To our knowledge, this is the first study that uses physical work capacity to predict serum leptin. This notion is supported by evidence that exercise decreases circulating leptin concentration, independent of body composition[12,35]. Moreover, exercise resulted in a decrease of plasma leptin concentration and an increase of maximal oxygen consumption in obese premenopausal women[17,27]. It has also been demonstrated that short term exercise resulted in a decrease of leptin concentration[15,28].

Blood pressure is correlated with circulating leptin levels. Our results demonstrated that SBP was a determinant of serum leptin concentration and had a significantly positive correlation with serum leptin. This result is supported by previous studies which showed independently positive correlations between circulating leptin levels and blood pressure[16,30]. Although the mechanism of leptin on blood pressure in humans has not yet been clarified, chronic hyperleptinemia increases blood pressure and it has been inferred that acute depressor effects are impaired and/or additional sympathetic nervous system-independent pressor effects appear, such as oxidative stress, NO deficiency enhanced renal $Na^+$ reabsorption and overproduction of endothelin[5].

Serum leptin levels are related with MS,

particularly a high leptin level increases the risk of the MS with central obesity. In this study, the risks of subjects with a high leptin level of the development of MS were 6 times higher than of those with a low leptin level. Importantly, subjects with a high leptin level had higher risks of the development of MS with central obesity than that of the non-MS, and the MS without central obesity. Our results demonstrate that central obesity is linked to hyperleptinemia and MS. This view is consistent with several previous reports that have shown that high leptin levels are associated with MS but that this association is significantly mediated through the effects of central obesity[3,11]. Sieminska et al[31] have suggested that leptin is correlated with MS components but leptin appears to play a role only in postmenopausal women. A recent study in 153 Korean postmenopausal women has demonstrated that waist-to-hip ratio and the number of MS components has a positive correlation with serum leptin level[22]. On the contrary, Chiu et al[8] have indicated that leptin levels are independently associated with diabetes, hypercholesterolemia and MS after adjustment for BMI in Taiwanese women. These discrepancies may be due to the ethnic group, age, menopause period, and sample size. Further research is needed to elucidate the associations among circulating leptin levels, obesity, and MS in different ethnic women with a wide range of age.

Central obesity in postmenopausal women increases serum leptin levels, and contributes to insulin resistance[9,32]. Evidence suggests that hyperleptinaemia and insulin resistance contribute to hypertension, impaired glucose metabolism, and pro-antherogenic state in obesity and MS[26]. On the basis of previous evidence and our results, we conclude that leptin is an important adipokine link in the process of MS. In other words, the linking in obesity-related metabolic disorders leads to the development of MS in postmenopausal women.

This study has several limitations. First, for the cross-sectional design, the effect of high leptin level on the MS can not be observed. Second, sample size was small and age range was narrow. Therefore, our results should be interpreted with caution.

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