

High Serum Asprosin Levels Are Associated with Presence of Metabolic Syndrome

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Research

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Abstract

Objective: Asprosin, a new adipocytokine, has been reported to be related with glucose release, dyslipidemia and insulin resistance (IR). However, the relationship of asprosin with metabolic syndrome (MetS) remains unknown. This study aims to investigate serum asprosin levels in MetS as well as its association with various metabolic parameters in humans.

Methods: The consecutive 131 patients with MetS and age-matched 162 healthy subjects were recruited for this study. Serum asprosin concentrations were determined by ELISA. Lipid profile, glucose, insulin, and inflammatory markers were also measured.

Results: Serum asprosin levels were higher in subjects with MetS 23.52 (16.70, 32.05) ng/ml compared to 16.70 (12.87, 22.38) ng/ml in the controls ($P < 0.01$), and showed an increasing trend with the increased numbers of metabolic components (P for trend < 0.01). In all studied subjects, serum asprosin levels were positive correlated with body mass index, waist circumference, percentage of body fat (%), triglyceride, fasting plasma glucose, 2h plasma glucose, fasting insulin, HOMA-IR, interleukin (IL) -6 and monocyte chemoattractant protein (MCP)-1, and negative correlated with HDL cholesterol ($P < 0.05$). In a multiple linear regression, asprosin was independently and positively correlated with triglyceride and HOMA-IR ($P < 0.05$). Binary logistic regression revealed that asprosin was independently and positively correlated with the occurrence of MetS and IR even after controlling for anthropometric variables, lipid profiles and inflammatory markers.

Conclusion: Asprosin may be a metabolic - related adipokine and related to insulin resistance and MetS.

Trial Registration: ChiCTR, ChiCTR1800018347. Registered 12 September 2018, <http://www.chictr.org.cn/showproj.aspx?proj=31050>.

1. Introduction

Metabolic syndrome (MetS) is a constellation of clinical features that include insulin-resistance (IR), obesity, dyslipidemia, hyperglycemia, and elevated blood pressure^[1]. A meta-analysis has shown that MetS is associated with an approximate doubling of cardiovascular disease risk and a 1.5-fold increased risk for all-cause mortality^[2]. Given the rapid increasing prevalence of MetS (20–30% of the general world population) and its possible harmful outcome^[3, 4], there is a severe need to further research to reveal its predictive factors and mechanisms. In recent years, some adipocyte-related cytokines have been shown to be biomarkers related to MetS such as adiponectin, zinc- α 2-glycoprotein and betatrophin^[5–7], and an increasing evidence shows that cytokines play an important role in the pathogenesis of MetS.

Asprosin, a 140-amino-acid C-terminal profibrillin, is a recently discovered fasting-induced adipokine, which was initially found in neonatal progeroid syndrome^[8]. It is highlighted that the elevated asprosin levels were observed in human and mouse with IR or obesity^[8]. A single injection of asprosin causes a

immediate spike in blood glucose and insulin in mice models and humans [7]. Conversely, genetic deficiency and specific antibody of asprosin results in improved insulin sensitivity and reduced appetite and body weight [8,9]. Recent studies found that serum asprosin levels are increased in T2DM, obesity and polycystic ovary syndrome, which are associated with fasting glucose and triglyceride (TG) [10–13]. However, the changes of asprosin abundances in MetS are still unknown. As MetS is strongly associated with lipid homeostasis, glucose and IR, we hypothesized that serum asprosin levels were altered in MetS. Therefore, we conducted a cross-sectional study to measure serum asprosin levels in MetS as well as its association with various metabolic parameters.

2. Materials And Methods

2.1 Research objects

A total of 293 subjects (including 131 MetS and age-matched 162 healthy controls) were recruited for this study from routine physical examinations at the First Affiliated Hospital of University of South China. MetS was defined according to the criteria set by a joint statement of the International Diabetes Federation Task Force on Epidemiology, and Prevention *et al* [1]. Subjects who fulfilled three of the following five criteria were defined as having MetS: 1) waist circumference ≥ 80 cm in women or 85 cm in men; 2) HDL cholesterol ≤ 1.3 mmol/l in women or ≤ 1.0 mmol/l in men; 3) triglycerides ≥ 1.7 mmol/l; 4) blood pressure $\geq 130/85$ mmHg or current use of antihypertensive drugs; 5) fasting plasma glucose ≥ 5.6 mmol/l (100 mg/dl) or previous diagnosis of type 2 diabetes or use of antidiabetic medication (insulin or oral agents). Age-matched healthy subjects without clinical evidence of major diseases were recruited as the controls. All participants completed a uniform questionnaire containing demographics, medical history, recent medication history and lifestyle factors (smoking and alcohol). MetS and healthy individuals had not been treated with any medicine including hypoglycemic, and lipid-lowering agents, as well as diet modification or exercise. Exclusion criteria included subjects with younger than 18 and older than 70 or suffering from any kind of infection, a history of cardiovascular disease, acute or chronic complications, heart, liver or kidney failure, pregnancy, or other known major disease. This study was approved by the Ethics Committee of the hospital, following the principles of the Helsinki Declaration, and all subjects were given written informed consent.

2.2 Anthropometric and biochemical evaluation

Measurement parameters including height, weight, waist circumference, and blood pressure (Systolic blood pressure [SBP] and diastolic blood pressure [DBP]) were measured using a standardized protocol. An analyzer of bioelectrical impedance was used to measure the percentage of body fat (Fat %). Body mass index (BMI) was defined as the individual's body weight divided by the square of the height.

Blood samples were obtained after fasting overnight for at least 10 hours. Total cholesterol, Triglyceride, Low-density lipoprotein cholesterol, High-density lipoprotein cholesterol, Fasting blood glucose, 2 h plasma glucose, HbA1c, and Fasting insulin were determined as published previously [14]. Homeostasis

model assessment of insulin resistance (HOMA-IR) index was calculated as [fasting plasma glucose (mmol/l) × fasting insulin (μU/ml) / 22.5].

2.3 Measurements of adipokines

Routine laboratory tests were performed in the accredited central laboratory of the Hospital according to standard protocols. Serum was obtained after centrifugation, aliquoted and then stored at - 80 °C for ELISA test. Serum asprosin levels were measured by the commercial Sandwich ELISA kits from abbexa Ltd (Cambridge, UK). Serum MCP-1 and IL-6 levels were measured by ELISA kits from R&D systems, Inc. (Minneapolis MN, USA). The average intra- and inter-assay coefficient of variations were 10% and 6% for Asprosin, 7.8% and 6.7% for MCP-1, 4.2% and 6.4% for IL-6, respectively.

2.4 Statistical analysis

The data were presented as mean ± SD or median with interquartile range (IQR). Normal distribution of the data was determined using the *Kolmogorov-Smirnov* test. Variables not normally distributed were logarithmically transformed to near normality before analysis. Comparisons of categorical and continuous variables were performed with the Chi-squared and one-way ANOVA tests, respectively. The correlations between variables were assessed using a *Pearson* correlation analysis by controlling for the covariates. The independent associations between asprosin and variables were determined using multiple linear regression. The adjusted odds ratio and 95% confidence interval for asprosin levels and MetS or IR were pre-sented by binary logistic regression. Receiver operator characteristic (ROC) curve was calculated to identify the abilities of asprosin to predict MetS and IR. The trends of asprosin levels associated with MetS were analyzed using the *Row mean score* test and *Cochran-Armitage* trend tests. $P < 0.05$ was taken to indicate statistically significant.

Because this was the first study to investigate serum asprosin levels in MetS, the post hoc power analysis of sample size was evaluated. Taking the serum asprosin levels in subjects with MetS and controls as the evaluation indicator. The data was logarithmically transformed to near normality before analysis. Control group [$\lg(\text{asprosin})$] = 1.226 ± 0.187 ng/ml, MetS group [$\lg(\text{asprosin})$] = 1.378 ± 0.212 ng/ml, population standard deviation (σ) = 0.212, power ($1-\beta$) set at 0.90 and $\alpha = 0.05$ (two side). Sample size was calculated by PASS 15.0.5 software. In total, 293 subjects were enrolled in our study. The sample size was considered to be adequate.

3. Results

3.1 Clinical and Biochemical Characteristics of the subjects

Anthropometric, biochemical and metabolic parameters of the 293 subjects were showed in Table 1. The age and gender were comparable between controls and MetS. As expected, MetS patients had higher BMI, Waist circumference, Fat%, SBP, DBP, TG, FIns, FPG, 2 h-PG, HOMA-IR, IL-6 and MCP-1, and lower HDL-C than the Controls ($P < 0.01$ or $P < 0.05$). However, there were no significant differences in recent smoking (%), TC and LDL-C between controls and MetS groups ($P > 0.05$).

Table 1
Clinical and biochemical characteristics of controls and MetS subjects.

Variables	Controls	MetS	P-Value
No. of subjects	162	131	-
Gender, M/F	78/84	65/66	0.802
Age(years)	49.47 ± 9.16	50.90 ± 9.45	0.236
Recent smoking (%)	18.5	26.7	0.093
BMI (kg/m ²)	23.28 ± 2.49	25.52 ± 3.02	< 0.001
Waist circumference (cm)	81.3 ± 7.5	87.2 ± 8.9	< 0.001
Fat (%)	27.61 ± 5.57	29.66 ± 3.68	< 0.001
SBP (mmHg)	123.7 ± 14.2	133.4 ± 13.3	< 0.001
DBP (mmHg)	74.9 ± 8.9	82.2 ± 9.4	< 0.001
TC (mmol/l)	4.73 ± 0.98	4.89 ± 1.05	0.181
TG (mmol/l) ^a	1.10 (0.82,1.39)	1.92 (1.58,2.49)	< 0.001
LDL cholesterol (mmol/l)	2.42 ± 0.90	2.60 ± 1.04	0.115
HDL cholesterol (mmol/l)	1.57 ± 0.36	1.18 ± 0.41	< 0.001
FPG (mmol/l) ^a	5.10 (4.74,5.41)	6.26 (5.26,8.89)	< 0.001
2 h-PG (mmol/l) ^a	5.82 (5.49,6.23)	7.71 (6.14,15.04)	< 0.001
FIns (mU/l) ^a	7.20 (6.06,8.79)	8.93 (7.78,10.62)	< 0.001
HOMA-IR ^a	1.60 (1.30,2.06)	2.53 (1.84,3.93)	< 0.001
IL-6 (ng/l)	12.62 ± 5.20	17.02 ± 6.32	< 0.001
MCP-1(ng/l)	118.95 ± 39.49	146.94 ± 52.41	< 0.001

Values were given as means ± SD or median with interquartile range.

^a, log transformed. *MetS*, Metabolic syndrome; *BMI*, Body mass index; *Fat*, Percentage of body fat; *SBP*, Systolic blood pressure; *DBP*, Diastolic blood pressure; *TC*, Total cholesterol; *TG*, Triglycerides, *LDL*, Low-density lipoprotein; *HDL*, High-density lipoprotein; *FPG*, Fasting plasma glucose; *2 h-PG*, 2 h plasma glucose; *FIns*, Fasting insulin; *HOMA-IR*, Homeostasis model assessment of insulin resistance; *IL-6*, Interleukin-6; *MCP-1*, Monocyte chemotactic protein-1.

3.2 Serum asprosin Levels

Serum asprosin levels were also assayed in all subjects. The distribution of serum asprosin was displayed in Fig. 1a for controls and Figure.1b for MetS subjects. Serum asprosin concentrations were located from 5.32 to 40.52 ng/ml for 95% controls and 8.42 to 79.41 ng/ml for 95% MetS subjects. Importantly, serum asprosin levels were significantly higher in MetS compared with controls [23.52 (16.70, 32.05) vs. 16.70 (12.87, 22.38) ng/ml, $P < 0.01$; Fig. 1c], and increased in a stepwise fashion as the number of MetS components increased (P for trend < 0.01 , Fig. 1e). Furthermore, subjects with abdominal obesity had significantly higher serum asprosin levels than those without ($P < 0.01$, Fig. 1d). However, there was no gender difference of serum asprosin between the control and MetS participants ($P > 0.05$), indicating that asprosin has no distinct sexual dimorphism.

3.3 Correlation of serum asprosin with clinical parameters in all subjects

Serum asprosin concentration were positively correlated with adiposity-related parameters (BMI, Waist circumference, Fat%, $P < 0.01$), an adverse lipid profile (increased TG and decreased HDL-C, $P < 0.01$), parameters of blood glucose (FPG and 2 h-PG, $P < 0.01$), insulin resistance indices (FIns and HOMA-IR, $P < 0.01$) and inflammatory markers (MCP-1 and IL-6, $P < 0.01$), Table 2. After further adjustment for age and BMI, all these correlations remained similar except for Waist circumference, Fat% and MCP-1. The multiple regression analyses of stepwise models to determine which variables were independently associated with serum asprosin concentrations. Only TG and HOMA-IR were independently related to serum asprosin ($P < 0.05$), with a multiple regression equation of $Y_{(\lg \text{ asprosin})} = 1.163 + 0.033 \times \text{TG} + 0.028 \times \text{HOMA-IR}$ (Table 2).

Table 2
Correlation of serum aprotin levels with clinical variables in all subjects

Variables	Simple		Multiple	
	<i>r</i>	<i>P</i> -value	$\beta \pm SE$	<i>P</i> -value
Age	0.003	0.961		
BMI	0.206	< 0.001		
Waist circumference #	0.171	0.003		
Fat (%)	0.113	0.054		
SBP	0.102	0.083		
DBP	0.111	0.058		
TC ^a	-0.022	0.710		
TG ^a	0.251	< 0.001	0.033 ± 0.010	0.001
HDL cholesterol	-0.194	0.001		
LDL cholesterol	0.012	0.842		
FPG ^a	0.302	< 0.001		
2 h-PG ^a #	0.366	< 0.001		
FIns ^a	0.314	< 0.001		
HOMA-IR ^a	0.316	< 0.001	0.028 ± 0.006	< 0.001
IL-6	0.183	0.002		
MCP-1	0.175	0.005		
^a log transformed before analysis.				
# WC did not enter into the multivariate regression due to its high intercorrelation with BMI (<i>r</i> = 0.746). 2 h blood glucose did not enter into the multivariate regression due to its high intercorrelation with fasting blood glucose (<i>r</i> = 0.931).				
In multiple linear regression analysis, values included for analysis were age, gender, BMI, TG, HDL-C, FPG, FIns, HOMA-IR, IL-6 and MCP-1.				
<i>BMI</i> , Body mass index; <i>Fat</i> , Percentage of body fat; <i>SBP</i> , Systolic blood pressure; <i>DBP</i> , Diastolic blood pressure; <i>TC</i> , Total cholesterol; <i>TG</i> , Triglycerides, <i>LDL</i> , Low-density lipoprotein; <i>HDL</i> , High-density lipoprotein; <i>FPG</i> , Fasting plasma glucose; <i>2 h-PG</i> , 2 h plasma glucose; <i>FIns</i> , Fasting insulin; <i>HOMA-IR</i> , Homeostasis model assessment of insulin resistance; <i>IL-6</i> , Interleukin-6; <i>MCP-1</i> , Monocyte chemotactic protein-1.				

3.4 The effect of serum asprosin on the incidence of MetS and IR

Serum asprosin concentrations were markedly related MetS and IR, even after adjustment for age, gender, BMI, lipid profile, inflammatory markers in an additive multivariate logistic regression model (Table 3). To further investigate the association of asprosin with MetS, asprosin levels were categorized using their quartile values (quartile 1: < 14.21 ng/ml; quartile 2: 14.21–19.26 ng/ml; quartile 3: 19.26–25.79 ng/ml, and quartile 4: > 25.79 ng/ml), and then logistic regression analysis was performed to calculate the odds of having MetS after controlling for the covariates. When asprosin levels were in quartile 4, the odds ratios of having MetS were 3.533 (vs. quartile 1, $P = 0.015$; Fig. 1f). Furthermore, a significant linear trend over increasing asprosin categories was seen for the presence of the MetS by the *Row Mean Scores* test and the *Cochran-Armitage* trend test (Table 4). Finally, The ROC curves analyses revealed that the area under the curve (AUC) for serum asprosin (gender and sex adjusted) to predict MetS was 0.712 (Fig. 2a), and to predict IR was 0.742 (Fig. 2b).

Table 3
Association of serum asprosin with MetS and IR in fully adjusted models

Model adjustment	MetS		IR	
	OR per 1 s.d increase (95% CI)	<i>P</i> -value	OR per 1 s.d increase (95% CI)	<i>P</i> -value
Model 1	2.076 (1.580, 2.728)	< 0.001	2.040 (1.574, 2.646)	< 0.001
Model 2	1.934 (1.467, 2.548)	< 0.001	1.952 (1.505, 2.531)	< 0.001
Model 3	1.726 (1.240, 2.402)	0.001	1.987 (1.506, 2.621)	< 0.001
Model 4	1.686 (1.206, 2.355)	0.002	1.961 (1.488, 2.584)	< 0.001
Model 5	1.544 (1.092, 2.183)	0.014		
Model 1 adjusted for age and gender;				
Model 2 further adjusted for BMI;				
Model 3 further adjusted for Lipid profiles (TC, TG, LDL-C, HDL-C);				
Model 4 further adjusted for inflammatory markers (IL-6 and MCP-1);				
Model 5 further adjusted for HOMA-IR.				
<i>IR</i> , insulin resistance; <i>BMI</i> , Body mass index; <i>TC</i> , Total cholesterol; <i>TG</i> , Triglycerides, <i>LDL-C</i> , Low-density lipoprotein cholesterol; <i>HDL-C</i> , High-density lipoprotein cholesterol; <i>IL-6</i> , Interleukin-6; <i>MCP-1</i> , Monocyte chemotactic protein-1. <i>HOMA-IR</i> , Homeostasis model assessment of insulin resistance.				

Table 4
Row mean scores and Cochran-Armitage trend test of the impact of asprosin on MetS

	MetS	
	χ^2	P-value
ROW Mean Scores Test	37.248	< 0.001
Cochran-Armitage Test	42.567	< 0.001

4. Discussion

This study was the first report to examine the serum concentration of asprosin in MetS subjects. The main findings were as follows: i) serum asprosin levels were markedly increased in MetS patients than in healthy controls, and showed an increasing trend with the increased numbers of metabolic components; ii) serum asprosin levels were positively correlated with BMI, WC, Fat%, FPG, 2 h-PG, FIns, HOMA-IR, TG, MCP-1 and IL-6, and negatively correlated with HDL cholesterol; iii) serum asprosin was independently and positively correlated with the occurrence of MetS and IR even after controlling for the covariates.

Asprosin has recently been identified as a white tissue-derived novel adipokine and its concentrations have been confirmed to be increased in adults with T2DM and PCOS [11–13]. However, the information remains unavailable regarding the role of asprosin in MetS. Here, we showed that serum asprosin levels were markedly elevated in MetS, which was similar to a recent study conducted in patients with T2DM [11, 12]. Nevertheless, the reason for the increase of asprosin secretion is unknown. Previous studies have demonstrated that plasma asprosin was pathologically elevated in mice and humans with IR, while asprosin-specific monoclonal antibody lowered plasma asprosin and improved insulin sensitivity in these mice [8, 9]. Hence, we speculate that asprosin may serve as a risk factor associated with the pathogenesis of MetS. However, the cross-sectional nature of the current study still can't rule out the possibility that the elevation of serum asprosin in MetS might be a compensatory up-regulation for counteracting the metabolic stress produced by adiposity, hyperglycemia, or hyperlipidemia. Therefore, a follow up study will be necessary.

IR is generally considered to be a root causative factor for developing MetS. Adipose tissue has the endocrine role to regulate energy balance and glucose homeostasis. Several adipose tissue-secreted cytokines can either enhance or impair insulin action [15]. Data for the current study clearly showed that asprosin was significantly positively correlated with the well-known indices of MetS in all individuals. Among these indices, HOMA-IR was independent related factors with serum asprosin levels. Previous studies have found that intraperitoneal injection of asprosin antibody can significantly reduce serum insulin levels and improve IR in obese mice [9]. Two recent clinical studies have also found that circulating asprosin concentrations are positively correlated with IR in patients with T2DM or PCOS [11, 13]. It is suggested that the correlation between asprosin and MetS may be part attributed to IR. In addition, the

ROC curve analysis showed that serum asprosin might be a useful marker for the prediction of MetS and IR in our study population. In one aspect, the value of AUC (0.712) and (0.742) was considered to be moderate significance, which may be due to the relatively small sample size and a non-normal distribution in the studied population. In another aspect, the serum asprosin may be not an ideal marker for predicting MetS and IR.

Dyslipidemia and hyperglycemia are the pathological state characterized of MetS and play crucial parts in the pathogenesis of the disease^[1]. Our data demonstrates that serum asprosin levels are significantly correlated with TG, HDL-C, FBG, and 2 h-PBG even after adjustment for age and BMI. Our multiple stepwise regression analysis has identified the TG as significant independent contributors to circulating asprosin levels. These findings raise the hypothesis that asprosin may provide a molecular association between glucose - lipid metabolism and MetS. Although it would be premature to conclude the causal effects of asprosin on these parameters, it would be of interest to explore whether therapeutically targeting asprosin may ameliorate metabolic disorder in MetS subjects.

Chronic low-grade inflammation is closely related to obesity and IR and can lead to the pathogenesis of some metabolic-related diseases. Previous study demonstrated that asprosin promotes hepatic glucose production by activating cAMP second - messenger system, which was also involved in the inflammatory response^[8]. Another in vitro experiment displayed that siRNA-mediated asprosin suppression improved NF- κ B phosphorylation and release of TNF- α and MCP-1 in the palmitic- treated pancreatic cells^[16]. However, a recent clinical research showed that serum asprosin had no significant association with the hs-CRP in diabetic patients, which function as an acute inflammatory marker of inflammation^[11]. These controversial results prompted us to further explore the association between asprosin and metabolic inflammation. Thus, we measured the inflammatory marker IL-6 and MCP-1 level in all subjects and found that even after adjusting for age and BMI factors, asprosin did find a significant correlation with IL-6. Further studies are still needed to clarify the precise function asprosin in metabolic inflammation.

This study also has certain limitations. Firstly, it is difficult to deduce the causal relationship between serum asprosin levels and MetS due to the cross-sectional study design. Hence, a larger sample of prospective studies needs to be confirmed. Secondly, the study is based on the Chinese population and therefore needs to be carefully promoted to other ethnic groups. Thirdly, related inflammation indicators such as hs-CRP were not tested. Fourth, our study only detects serum levels based on ELISA, and there may be some random measurement errors.

5. Conclusion

In summary, our data showed that elevated serum asprosin was associated with the occurrence of MetS. TG and HOMA-IR were independent factors affecting serum asprosin. We speculate that adipokine asprosin may serve as a risk factor which is involved in the pathogenesis of MetS and IR. Further mechanistic studies are required to confirm this speculation.

Declarations

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Availability of data and materials

The datasets used and/or analysed during the current study are available from the corresponding author on reasonable request.

Author Contributions

TH was responsible for study design and writing of the manuscript. JL analyzed the data and wrote this manuscript. YW and PB contributed to data collection and were responsible for ELISA detection. XQ, LR, JY and BY contributed to data collection. XX and JL were in charge of this study and paid a lot of time in revising this manuscript.

Conflict of Interest Statement

The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

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Figures

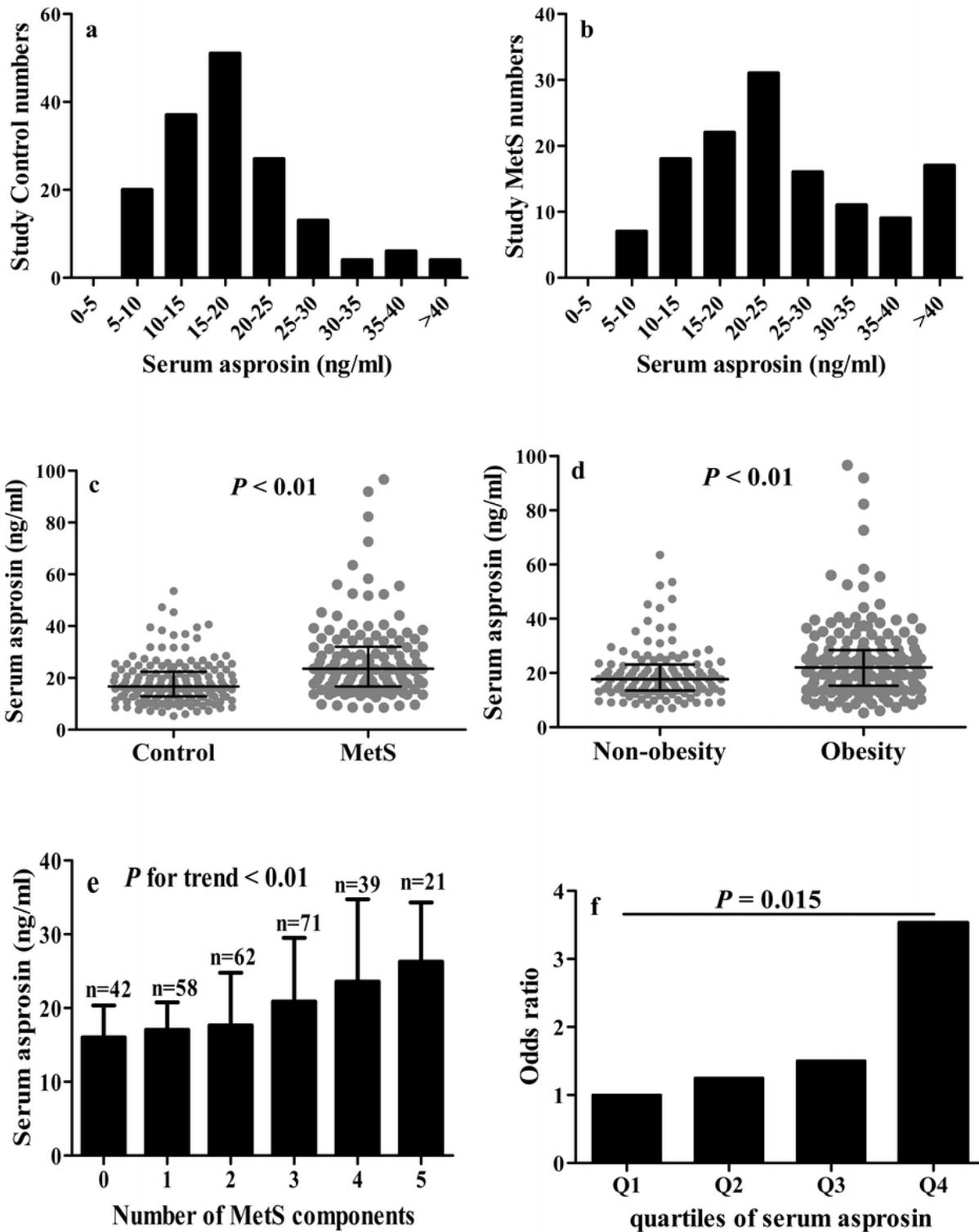
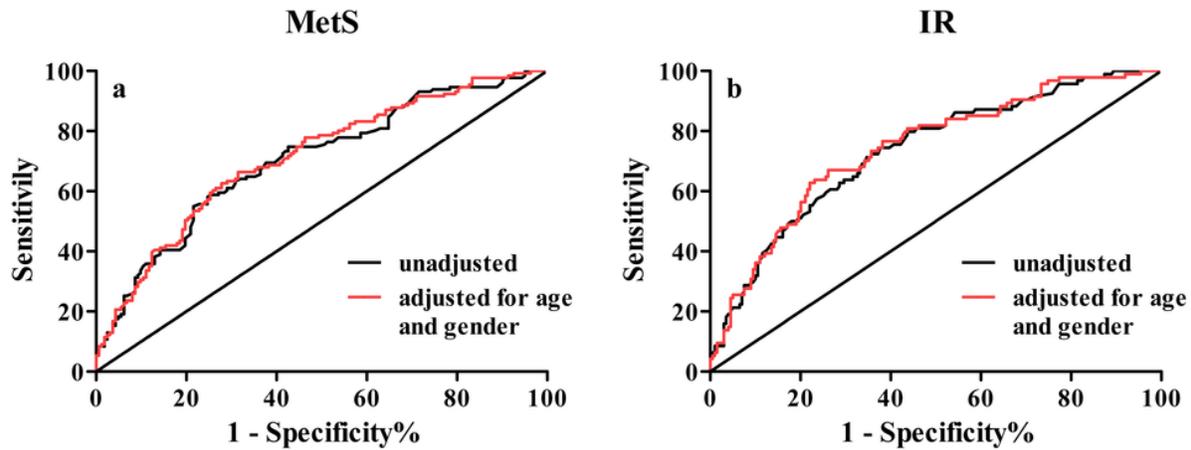


Figure 1

Serum asprosin levels in the study population. (a) distribution of serum asprosin levels in 162 control subjects; (b) distribution of serum asprosin levels in 131 MetS subjects; (c) serum asprosin levels in control and MetS subjects. Values were given as median with interquartile range and log transformed before analysis; (d) serum asprosin levels in all studied population according to waist circumference (Non-obesity: waist circumference < 85 cm in men or 80 cm in women and Obesity: waist circumference

≥ 85 cm in men or 80 cm in women. Values were given as median with interquartile range and log transformed before analysis; (e) serum asprosin levels increased progressively with increasing numbers of components of MetS. Values were given as median with interquartile range and log transformed before analysis; (f) odds ratio for having MetS according to the quartiles of serum asprosin levels (reference, the lowest quartile). Q1: < 14.21 ng/ml; Q2: 14.21-19.26 ng/ml; Q3: 19.26-25.79 ng/ml, and Q4: > 25.79 ng/ml. (Q: quartile).



Test variable	Variables	Area	cut-off points value	<i>P</i> -value	95% Confidence Interval	
					Lower limit	Upper limit
MetS	Asprosin	0.703	17.76	< 0.001	0.643	0.762
	Asprosin (age and gender adjusted)	0.712	18.08	< 0.001	0.653	0.771
IR	Asprosin	0.733	19.35	< 0.001	0.673	0.794
	Asprosin (age and gender adjusted)	0.742	20.14	< 0.001	0.682	0.802

Figure 2

ROC curve analyses were performed for the prediction of serum asprosin for MetS (a) and IR (b). MetS, Metabolic syndrome; IR, Insulin resistance.

Supplementary Files

This is a list of supplementary files associated with this preprint. Click to download.

- [rawdata.xls](#)