

Correlation of gender and age with serum adipokines level in north indian adults

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ABSTRACT

Objective: There are two forms of adipose tissue, brown adipose tissue (BAT) and white adipose tissue (WAT). Distribution of adipose tissue differs in different gender and different age group. Adipose tissue expresses various secretory proteins, including leptin and adiponectin, collectively called adipokines. The purpose of this study to elucidated the correlation of serum leptin, serum adiponectin levels with age and gender in north Indian population. **Materials and Methods:** 100 subjects (55 male, 45 female) aged between 18-25 yrs were included in the study. Blood sample were taken after overnight fasting for biochemical assay. Serums were separated by centrifugation method. Serum leptin and serum adiponectin were analyzed by using RayBio ELISA Kit. **Result:** The serum leptin (pg) was significantly ($p=0.001$) higher among female (428.28 ± 83.06) subjects compared with males (365.42 ± 102.56). Similarly, serum adiponectin (pg) level was also significantly ($p=0.02$) higher among females (5313.15 ± 1151.12) than males (4794.72 ± 1078.38). The age was mildly and negatively correlated with Serum adiponectin ($r = -0.317$, $p=0.001$) and had no correlation with serum leptin levels. **Conclusion:** Serum adipokines levels in young north Indian adults are influenced by gender and age of subjects, suggests that female subject, relative to the males, are at increased risk for obesity-related metabolic co-morbidities.

Key words: Serum leptin, Serum adiponectin, Gender

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INTRODUCTION

Obesity is a complex, multifactorial disorder characterized by an excess of adipose tissue or body fat. In obesity there is excessive adipose tissue mass.¹ Obesity arises when energy intake exceeds energy expenditure. There is convincing experimental evidence showing that the balance between energy intake (food consumption) and energy expenditure is tightly regulated. A homeostatic network maintain energy store through a complex interplay between the feeding regulatory centers in the central nervous system (CNS), particularly in the hypothalamus and the peripheral fat stores.²

Adipose tissue is one of the largest tissues in human body and total amount deposited will have a detrimental

impact on regular body function. The quantity of body fat could be significant source of hundreds of biologically active molecules the adipokines including more than 50 cytokines, chemokines, hormone like growth factor and other mediators like leptin, adiponectin, visfatin, apelin, vaspin, hepcidine, chemerin, omentin including tumor necrosis factor alpha (TNF α), monocyte chemoattractant protein-1 (MCP-1) and plasminogen activator protein (PAI).

Leptin is a 16 kDa protein hormone that plays a key role in regulating energy intake and energy expenditure, including appetite and metabolism. Leptin acts primarily on the hypothalamus. leptin activates POMC neurons, activated POMC neuron release α -MSH into the synapses, which activates the projected neurons via binding to MCRs and lead to anorexigenic responses and increased energy expenditure.

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Simultaneously, leptin inhibits NPY/AgRP neurons, offsetting the antagonistic effect of AgRP on MCRs.³ NPY/AgRP neurons stimulates orexigenic responses and inhibits POMC neuron via direct synaptic connection.⁴

Adiponectin has insulin-sensitizing actions in the liver, and lowers blood glucose levels in diabetic animals by improving insulin-mediated suppression of gluconeogenesis. In liver and skeletal muscle, adiponectin also improves glucose utilization and stimulates fatty acid oxidation via a pathway that involves AMP kinase (AMPK) and acetyl-CoA carboxylase. In the present study, we elucidated the correlation of serum leptin, serum adiponectin levels with age and gender in north Indian population.

MATERIALS AND METHODS

This was a cross sectional study enrolled with hundred subjects (55 male, 45 female) aged (Table 1) between 18 to 25 years (Table 2). This study was approved by ethical committee of institute.

Inclusion criteria

Age between 18 to 25 years irrespective of sex, apparently healthy subjects, Place of birth north India.

Exclusion criteria

Any chronic illness eg- Tuberculosis, COPD; Any apparent cardiac illness, Any apparent metabolic disorder eg- Diabetes Mellitus, Hypertension, Any congenital disorder.

Biochemical analysis

Blood samples were taken after overnight fasting and serum was separated, after coagulation, by centrifugation method. Serum leptin and serum adiponectin were estimated by using RayBio ELISA Kit.

Statistical analysis

The results were presented in mean \pm SD and percentages. The p -value <0.05 was considered significant. All the analysis was carried out by using SPSS 16.0 version (Chicago, Inc., USA).

RESULTS

The serum leptin (pg) levels (Table 3) were significantly ($p=0.001$) higher among female (428.28 ± 83.06) subjects compared with males (365.42 ± 102.56). Similarly, serum adiponectin (pg) levels (Table 4) were also significantly ($p=0.02$) higher among females (5313.15 ± 1151.12) than males (4794.72 ± 1078.38). The age was mildly and negatively correlated with Serum adiponectin ($r= -0.317$, $p=0.001$) and had no correlation with serum leptin levels (Table 4).

Table 1: Gender distribution of the subjects

Gender	No. (n=100)	Percentage
Male	55	55.0
Female	45	45.0

Table 2: Age distribution of the subjects

Age in years	No. (n=100)	Percentage
<20	17	17.0
20-22	62	62.0
>22	21	21.0
Mean \pm SD (Min.-Max.)	21.35 \pm 1.50 (18-24)	

Min.: Minimum, Max.: Maximum

Table 3: Comparison of biochemical parameters with gender

Biochemical parameters	Mean \pm SD		p-value ¹
	Male (n=55)	Female (n=45)	
Serum leptin (pg)	365.42 \pm 102.56	428.28 \pm 83.06	0.001*
Serum adiponectin (pg)	4794.72 \pm 1078.38	5313.15 \pm 1151.12	0.02*

¹Unpaired t-test, *Significant $p<0.05$

Table 4: Correlation coefficient (r) between age and among the biochemical parameters

Biochemical parameters	Age
Serum leptin (pg)	$r=0.092$ $P=0.363$
Serum adiponectin (pg)	$r=-0.317^{**}$ $P=0.001$

*Correlation is significant at the 0.05 level (2-tailed). **Correlation is significant at the 0.01 level (2-tailed)

DISCUSSION

Data was analyzed according to gender, and we observed that serum leptin levels were significantly higher in females (428.28 ± 83.06) subjects as compared with male (365.42 ± 102.56) subjects. There are several possible explanation for difference. One is that females have more adipose tissue than males, but some literature indicate that estrogen level are higher in female which stimulate adipose tissue and increase the production of leptin, whereas higher androgen level in male suppress the level of leptin production.⁵ Finding of Daghari et al⁶ in 2007 corroborate with our results.

In the present study, we also studied the correlation of serum adiponectin levels with different genders and observed that serum adiponectin levels are significantly ($p=0.02$) higher in females (5313.15 ± 1151.12) as compared with males (4794.72 ± 1078.38). Same results also obtained by Böttner A. et al,⁷ 2004 and Chamukuttan Snehalatha et al,⁸ 2008.

In the present study age does not show any correlation with serum leptin levels. Zhong N *et al*,⁹ 2005 found high serum leptin levels in postmenopausal women than premenopausal women, but after adjusting for BMI no association of serum leptin with age was found. Our study also showed significant negative correlation with age and serum adiponectin levels. This finding was contradicted by Obata Y. *et al*,¹⁰ 2013 as they observed that serum adiponectin levels are significantly and positively associated with age in healthy subjects, but finding of our study was supported by Sunita J Ramanand *et al*,¹¹ 2014 as they found that adiponectin levels decreases as age advances.

CONCLUSION

Serum adipokines levels in young north Indian adults are influenced by gender and age of subjects, suggests that female subject, relative to the males, are at increased risk for obesity-related metabolic co-morbidities. Owing to small sample size of this study we were not definitely established our results. Hence, further studies required in future with large sample size.

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Authors Contribution:

Dr. Narsingh Verma – Contributed to the original idea, designed the study; **Dr. Sunita Tiwari**–Conceived hypothesis, designed study, preparing of manuscript and reviewing the manuscript; **Dr. Neena Srivastava** - Contributed to the study design, data analysis. **Dr. Shivani Pandey** – Contributed to patient enrolment and biochemical analysis; **Dr. Pravesh kumar, Dr. Naveen Bharti Porwal and Dr. Pradeep Kumar** – Contributed to patient enrolment, data analysis, preparing of manuscript and reviewing the manuscript. **Dr. Rahul** - Contributed to patient enrolment, data analysis, patient blood collection.

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