|| ISSN(online): 2589-8698 || ISSN(print): 2589-868X ||
International Journal of Medical and Biomedical Studies
Available Online at www.ijmbs.info
NLM (National Library of Medicine ID: 101738825)
Index Copernicus Value 2019: 79.34
Original Research Article
Volume 5, Issue 4; April: 2021; Page No. 150-153

# THE RELATIONSHIP BETWEEN LEPTIN AND ANTHROPOMETRIC PARAMETERS IN TYPE-2 DIABETIC PATIENTS

**Dr. Sajidali S. Saiyad[1], Dr Alkesh Vara[2]***
[1]Assistant Professor, Department of Physiology, Medical College, People's College of Medical Sciences and RC, Bhopal
[2]Associate Professor, Department of Physiology, Nootan Medical College & Research Center, Visnagar

**Article Info:** Received 01 February 2021; Accepted 25 March 2021
**DOI:** https://doi.org/10.32553/ijmbs.v5i4.1979
**Corresponding author:** Dr Alkesh Vara
**Conflict of interest:** No conflict of interest.

**Abstract**
**Aim:** The Relationship between Leptin & Antithropometric Parameters in Type-2 Diabetic Patients.
**Methods:** In the present study, adult's (male & female) subjects were selected from the Diabetes Center at the medical college, India. Ethical approval was obtained from the local institution's review committee and consent was obtained from all participants. For the purposes of this study, subje
cts were classified into categories of abnormal glucose homeostasis if they had a single abnormal FPG. Anthropometric parameters of the subjects i.e. Blood pressure was measured using a mercury sphygmomanometer to the right arm while the individuals were in a sitting position. Blood samples were collected after a 12-hour fast for the determination of glucose, insulin, total cholesterol, HDL, triglycerides and leptin.
**Results**: A total of 140 diabetic patients suffering from metabolic syndrome were enrolled in the study. There were 90 male and 50 female patients of 60-70 years age group. The anthropometric parameters in both male and female subjects suffering from metabolic syndrome were studied. In the diabetes group, FPG and LDL were significantly higher than in the control. Total cholesterol was also significantly higher in women with diabetes.
**Conclusion:** In the above study, it was observed that serum leptin levels were significantly associated in females as compared to males with the metabolic syndrome. It is directly related with the serum insulin and lipid profile levels. The significant correlation of leptin to selected anthropometric measurements of obesity is confirmed in non-diabetic Subjects.
**Keywords:** Leptin, Antithropometric Parameters, Diabetic Patients, Biochemical Parameters, Cholesterol, HDL & LDL

## Introduction:

Leptin was discovered in nineteen century and it is a 16-kDa product of the obese gene which is mainly produced by white adipose tissue. It belongs to long chain class I cytokines with four helix bundle conformation. Leptin is an important factor related to the regulation of food intake and it plays a vital role in the pathophysiology of the obesity as well.[1] Leptin reaches the hypothalamus through the blood–brain barrier and acts to reduce food intake and increase metabolism. It is observed that leptin acts through the increase in the effect of Cholecystokinin (CCK) which is released after meal intake from the small intestine. It is done through the receptor mediated activation.[2]

It also acts on the hypothalamus to signal when the body has sufficient energy stores, thus inhibiting appetite and regulating the energy metabolism. The actions of leptin occur over both short and long time frames. In the short term, plasma leptin serves as a satiety signal, and over longer periods, daily mean plasma leptin concentration communicates long-term energy status to the brain.[3]

Leptin is important for body weight regulation. In mice, due to nonfunctional obese gene and mutations of this gene on chromosome seven, that encodes the leptin protein and results in obesity and type 2 diabetes. Leptin secreted by white adipose tissue is proportional to the volume of body of adipose tissue; therefore, adiposity greatly influences leptin levels.[4] In addition, other factors, including rapid or excessive food intake, sleep, body temperature, gender, circadian rhythm, and other hormones such as insulin, growth hormone, glucocorticoids, testosterone, and thyroid hormone, impact leptin secretion and expression.[5]

Although the predictive and clinical utility of metabolic syndrome has been debated in some circles, it is generally accepted that metabolic syndrome serves as a construct to identify individuals who have an increased and long-term risk of atherosclerotic cardiovascular disease (ASCVD) with or without type 2 DM.[6]

The aim of our study was to assess the relationship between serum leptin levels and antithropometric parameters in type 2 diabetic patients.

## Methods

In the present study, adult's (male & female) subjects were selected from the Diabetes Center at the medical college, India. Ethical approval was obtained from the local institution's review committee and consent was obtained

from all participants. For the purposes of this study, subjects were classified into categories of abnormal glucose homeostasis if they had a single abnormal FPG. 140 patients had type-2 diabetes (90 males and 50 females). **Since diabetes mellitus is a metabolic** syndrome, so for the selection process of these patients who is suffered from metabolic syndrome, regular examinations of their blood biochemistry were studied. For the confirmation of metabolic syndrome, blood parameters i.e. GBL, Hb1Ac, Lipid profile were checked in respect to normal patients. All patients with diabetes were treated with a low-carbohydrate diet, with or without oral antidiabetetic agents, mainly glibenclamide and Metformin.[7]

***Anthropometric parameters***

Anthropometric parameters of the subjects i.e. Blood pressure was measured using a mercury sphygmomanometer to the right arm while the individuals were in a sitting position. The participant's weight and height was measured without shoes. BMI was calculated as weight (kg) divided by the square of height (m2). The waist circumference measurement was taken at the end of expiration and in between the midpoint of the last rib and superior iliac crest.[8]

***Estimation of Different Biochemical Parameters***

Blood samples were collected after a 12-hour fast for the determination of glucose, insulin, total cholesterol, HDL, triglycerides and leptin. All samples were stored at -70°C prior to analysis by using routine laboratory methods, except for insulin and leptin assay. Insulin was analyzed by a solid phase enzyme amplified sensitivity immunoassay (Medgenix INS-ELISA). Leptin concentrations were measured by radio-immunoassay (Linco Research).[9]

***Statistical Analysis***

Data were analyzed using the Statistical Package for the Social Sciences (SPSS) version 10 (SPSS, Evanston, IL, USA) for Windows. Biochemical parameters not normally distributed were analyzed after being logarithmically transformed. Students' unpaired t-test and one-way analysis of variance (ANOVA) were used to compare the results of the different groups. Data were expressed as mean (SD) or median (range); statistical significance was set at $p < 0.05$.

**Results**

A total of 140 diabetic patients suffering from metabolic syndrome were enrolled in the study. There were 90 male and 50 female patients of 60-70 years age group. The anthropometric parameters in both male and female subjects suffering from metabolic syndrome were studied. There was significant difference in BMI and waist circumference of the female subjects as compared to the male subjects. The data were presented in Table 1.

**Table 1: Anthropometric parameters in metabolic syndrome patients**

| Parameters | Metabolic Syndrome (male) | Metabolic Syndrome (female) |
|---|---|---|
| **Age (yrs)** | 60.6±2.5 | 62.8±4.11 |
| **Height (cms)** | 151.0±5.22 | 155.4±4.55 |
| **Weight (kgs)** | 72.3±8.34 | 79.6±9.23 |
| **BMI (kg/m$^2$)** | 33.5±4.47 | 39.4±5.28 |
| **Waist circumference (cms)** | 91.2±9.11 | 98.4±8.58 |
| **Systolic blood pressure (mm/Hg)** | 152.4±2.33 | 158±3.85 |
| **Diastolic blood pressure (mm/Hg)** | 118.2±6.44 | 120±4.29 |

Table 2 & 3 highlights the clinical and metabolic parameters in the male & females. In the diabetes group, FPG and LDL were significantly higher than in the control. Total cholesterol was also significantly higher in women with diabetes.

**Table 2: Clinical characteristics and metabolic parameters of male subjects with type-2 diabetes and non diabetes**

| Blood Parameters | Non Diabetes | Type-2 Diabetes |
|---|---|---|
| Fasting Glucose (mmol/L) | 4.7 | 13.7 |
| Cholesterol (mmol/L) | 5.5 | 6.7 |
| HDL (mmol/L) | 0.91 | 0.6 |
| Triglyceride (mmol/L) | 1.5 | 2.9 |
| LDL (mmol/L) | 3.2 | 4.5 |
| Insulin μmol/L | 12.9 | 16.6 |
| Leptin ng/mL | 3.3 | 12.7 |

**Table 3: Clinical characteristics and metabolic parameters of female subjects with type-2 diabetes and normal control**

| Blood Parameters | Non diabetes | Type-2 diabetes |
|---|---|---|
| Fasting Glucose (mmol/L) | 4.9 | 14.3 |
| Cholesterol (mmol/L) | 5.1 | 6.8 |
| HDL (mmol/L) | 1.3 | 1.4 |
| Triglyceride (mmol/L) | 1.4 | 2.5 |
| LDL (mmol/L) | 3.7 | 4.8 |
| Insulin (μmol/L) | 13.3 | 14.3 |
| Leptin ng/mL | 4.2 | 13.5 |

## Discussion

It was observed that regardless of obesity status of the study subjects, the decrease in serum leptin levels reduces the cardiovascular risk factors and hence, the rate of metabolic syndrome.[11] During 8-year follow-up done in Olivetti Heart Study (OHS), it was suggested that increase in leptin levels were strongly associated with increase in development of hypertension and impaired fasting glucose which points to rise in cases of metabolic syndrome.[12] In a prospective population based study in 2005, it was observed that glucose intolerance and insulin resistance are specific factors related to the development of metabolic syndrome.[13]

Data were presented according to gender, since it is already an established fact that leptin levels are significantly higher in women than in men. There are several possible explanations for the difference. One is that females have more adipose tissue than males, but a growing literature indicates that estrogen, especially at higher levels, will stimulate the production of leptin, whereas androgens will suppress the levels of leptin.[14] In this cross-sectional study, we found positive correlations between leptin and hip circumference in pre-diabetic patients. This is consistent with a previous study, which also showed that this group is at high risk of developing CHD.[15] The positive association with hip circumfer- ence remained significant after adjusting for gender and BMI, which confirmed the results in previous studies for healthy men and women.[16,17] With impaired glucose tolerance, small changes in circulating insulin also alter insulin levels.[18]

Serum leptin concentration may contribute to the risk of CVD by altering lipid metabolism and contributing to hypertension via the activation of the sympathetic nervous system and increasing renal sodium re-absorption.[19,20] However, in our study there was no correlation between leptin and blood pressure. In another study, lep- tin signaling directly promoted atherosclerosis and may therefore represent a therapeutic target for the prevention of atherosclerosis.[21] Lichnovska and her colleagues in their recent report mentioned the significant role of serum leptin in the progression of insulin resistance, but this was not confirmed in the present study.[22] The difference can be due to the fact that the earlier study considered elderly and hyperlipemic patients, while the present study focused on pre-diabetic, diabetic and non-diabetic adults. Our results, however, confirm the results of Adamia *et al.*, which reveal no correlation between leptin and insulin resistance.[23]

The association of leptin to waist circumference (a surrogate for upper body obesity) and hip circumference confirms findings in Mexican American populations.[24,25] After adjustment for BMI, serum leptin concentrations in pre-diabetic men were independent of waist circumference, but in women they were associated with hip circumference. Hip circumference is a proxy measure of peripheral fat in serum leptin concentrations, aside from the fact that women have a significantly larger volume of subcutaneous fat than do men. In the control subjects, leptin concentrations were directly and significantly related to subcutaneous fat, with a strong inverse relationship to waist-hip ratio. When the results were considered according to gender, it was found that 33% of the male with waist circumference >102 cm, had diabetes. In female subjects, 27% of those with a high waist circumference ≥88 cm had diabetes, compared to only around 13% of those with a normal waist circumference.[8] Leptin is related to waist and hip circumference and is directly proportional to body fat. However, in those with diabetes, this relationship is lost, reflecting the effect of other factors, including hyperinsulinaemia and the activation of sub-clinical inflammation.

## Conclusion

In the above study, it was observed that serum leptin levels were significantly associated in females as compared to males with the metabolic syndrome. It is directly related with the serum insulin and lipid profile levels.

The significant correlation of leptin to selected anthropometric measurements of obesity is confirmed in non-diabetic Subjects. In those with diabetes, this relationship is lost, reflecting the effect of other

factors, including hyperinsulinaemia and the activation of sub-clinical inflammation.

## References


1. Huising MO, Kruiswijk CP, Flik G. Phylogeny and evolution of class-I helical cytokines. J Endocrinol. 2006 Apr;189(1):1-25.
2. Tartaglia LA, Dembski M, Weng X, Deng N, Culpepper J, Devos R, et al. Identification and expression cloning of a leptin receptor, OB-R. Cell. 1995 Dec 29;83(7):1263-71.
3. Chehab FF, Mounzih K, Lu R, Lim ME. Early onset of reproductive function in normal female mice treated with leptin. Science. 1997 Jan 3;275(5296):88-90.
4. Myers Jr MG, Leibel RL, Seeley RJ, Schwartz MW. Obesity and leptin resistance: distinguishing cause from effect. Trends Endocrinol Metab. 2010 Nov 1;21(11):643-51.
5. Zhang Y, Proenca R, Maffei M, Barone M, Leopold L, Friedman JM. Positional cloning of the mouse obese gene and its human homologue. Nature. 1994 Dec;372(6505):425-32.
6. Grundy SM, Cleeman JI, Daniels SR, Donato KA, Eckel RH, Franklin BA, et al. Diagnosis and management of the metabolic syndrome: an American Heart Association/National Heart, Lung, and Blood Institute scientific statement. Circulation. 2005 Oct 25;112(17):2735-52.
7. American Diabetes Association: Diagnosis and classification of diabetes mellitus. Diabetes Care 2004, 27(Suppl 1):S5-S10.
8. Al-Daghri N, Al-Rubean K, Bartlett WA, Al-Attas O, Jones AF, Kumar S: Serum leptin is elevated in Saudi Arabian patients with metabolic syndrome and coronary artery disease. Diabet Med 2003, 20(10):832-837.
9. Matthews DR, Rudenski AS, Naylor BA, Treacher DF, Turner RC: Homeostasis model assessment: insulin resistance and beta- cell function from fasting plasma glucose and insulin concen- trations in man. Diabetologia 1985, 28(7):412-419.
10. Yun JE, Kimm H, Jo J, Jee SH. Serum leptin is associated with metabolic syndrome in obese and nonobese Korean populations. Metabolism. 2010 Mar 1;59(3):424-9.
11. Galletti F, Barbato A, Versiero M, Iacone R, Russo O, Barba G, et al. Circulating leptin levels predict the development of metabolic syndrome in middle aged men: an 8-year follow-upstudy. J Hypertension. 2007 Aug 1;25(8):1671-7.
12. Franks PW, Brage S, Luan JA, Ekelund U, Rahman M, Farooqi IS, et al. Leptin predicts a worsening of the features of the metabolic syndrome independently of obesity. Obes Res. 2005 Aug;13(8):1476-84.
13. Castracane VD, Henson MC: When did leptin become a repro- ductive hormone? Semin Reprod Med 2002, 20(2):89-92.
14. Alexander CM, Landsman PB, Teutsch SM: Diabetes mellitus, impaired fasting glucose, atherosclerotic risk factors, and prevalence of coronary heart disease. Am J Cardiol 2000, 86(9):897-902.
15. Zhong N, Wu XP, Xu ZR, Wang AH, Luo XH, Cao XZ, Xie H, Shan PF, Liao EY: Relationship of serum leptin with age, body weight, body mass index, and bone mineral density in healthy mainland Chinese women. Clin Chim Acta 2005, 351(1–2):161-168.
16. Maugeri D, Bonanno MR, Speciale S, Santangelo A, Lentini A, Russo MS, Calanna A, Malaguarnera M, Motta M, Testai' M, Panebianco P: The leptin, a new hormone of adipose tissue: clinical findings and perspectives in geriatrics. Arch Gerontol Geriatr 2002, 34(1):47-54.
17. Leonhardt W, Horn R, Brabant G, Breidert M, Temelkova-Kurkt- schiev T, Fucker K, Hanefeld M: Relation of free and specifically bound leptin to insulin secretion in patients with impaired glucose tolerance (IGT). Exp Clin Endocrinol Diabetes 1997, 107(1):46-52.
18. Asakawa H, Tokunaga K, Kawakami F: Relationship of leptin level with metabolic disorders and hypertension in Japanese type 2 diabetes mellitus patients. J Diabetes Complications 2001, 15(2):57-62.
19. Couillard C, Mauriege P, Prud'homme D, Nadeau A, Tremblay A, Bouchard C, Despres JP: Plasma leptin concentrations: gender differences and associations with metabolic risk factors for cardiovascular disease. Diabetologia 1997, 40(10):1178-1184.
20. Reilly MP, Iqbal N, Schutta M, Wolfe ML, Scally M, Localio AR, Rader DJ, Kimmel SE: Plasma leptin levels are associated with coro- nary atherosclerosis in type 2 diabetes. J Clin Endocrinol Metab 2004, 89(8):3872-3878.
21. Lichnovska R, Gwozdziewiczova A, Hrebicek J: Gender differences in factors influencing insulin resistance in elderly hyperli-pemic non-diabetic subjects. Cardiovasc Diabetol 2002, 1:4.
22. Adamia N, Virsaladze D, Charkviani N, Skhirtladze M, Khutsishvili M: Effect of metformin therapy on plasma adiponectin and lep- tin levels in obese and insulin resistant postmenopausal females with type 2 diabetes. Georgian Med News 2007:52-55.
23. Haffner SM, Gingerich RL, Miettinen H, Stern MP: Leptin concen- trations in relation to overall adiposity and regional body fat distribution in Mexican Americans. Int J Obes Relat Metab Disord 1996, 20(10):904-908.
24. Kafulafula G, Moodley J: Leptin levels in the obese African par-turient. J Obstet Gynaecol 2001, 21(3):228-231.
25. Haffner SM, Stern MP, Hazuda HP, Mitchell BD, Patterson JK: Cardi-ovascular risk factors in confirmed prediabetic individuals. Does the clock for coronary heart disease start ticking before the onset of clinical diabetes? JAMA 1990, 263(21):2893-2898.