



CODEN [USA]: IAJPBB

ISSN: 2349-7750

**INDO AMERICAN JOURNAL OF  
PHARMACEUTICAL SCIENCES**<http://doi.org/10.5281/zenodo.1461937>Available online at: <http://www.iajps.com>

Research Article

**ASSOCIATION OF ROUTINE LIFESTYLE WITH THE LEVELS  
OF PLASMA OBESTATIN IN NORMAL, OBESE,  
NORMOTENSIVE AND HYPERTENSIVE OBESE PATIENTS**<sup>1</sup>Dr. Saad Saleem Dogar, <sup>2</sup>Dr. Sonia Fida, <sup>3</sup>Dr. Ayesha Umar<sup>1</sup>Nishtar University Hospital Multan<sup>2</sup>MIKD Multan<sup>3</sup>VAWC Multan**Abstract:**

**Objective:** Due to the disparity between thermal intake and disbursement, a diseased condition called obesity results. Fatness is specified by immoderate dissipation of body fat. Due to this and a load of life-related comorbidities, the qualities of life and life expectancy is affected severely.

To analyze the role of levels of plasma obestatin in obesity, which is also correlated with daily life routine is the main objective of this study.

**Methods and Patients:** According to the rules and regulations of the hospital's ethical committee, this study was organized. We collected information from 50 fat patients. These patients were also going through the heart and cholesterol disorders. The patients were analyzed at Service Hospital, Lahore (January 2017 to July 2017).

**Results:** In normal and fat person, mean fasting obestatin level was  $(0.450 \pm 0.468)$  and  $(0.959 \pm 0.889)$  respectively. With the value of  $p$  as 0.000, the difference between both groups related to mean fasting obestatin levels was statistically important.

**Conclusion:** In obesity, the important role is played by obestatin. There is a direct relation of obestatin with blood glucose level.

**Keywords:** Obestatin, Obesity, Disease and Patients.

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Please cite this article in press Saad Saleem Dogar et al., Association of Routine Lifestyle with the Levels of Plasma Obestatin In Normal, Obese, Normotensive and Hypertensive Obese Patients., Indo Am. J. P. Sci, 2018; 05(10).

**INTRODUCTION:**

Due to the disparity between thermal intake and disbursement, a diseased condition called obesity results. Fatness is specified by immoderate dissipation of body fat. Due to this and load of related comorbidities, the qualities of life and life expectancy are affected severely. Recent data from World Health Organization related but Obesity is found among the 11% of the world population (more than half a billion people) whereas 35% of the world population is overweighted [1]. Moreover, all over the world, the extensiveness of obesity is progressing continuously. So, it is urgent and mandatory to find effective treatments and demonstrating the pathomechanism. During the past decades, much research has demonstrated that there is a strong complementary connection between neurotransmitter systems [2]. These systems are controlling appetite and feeding behaviour, cognitive function, stress and reward behaviour. Homeostatic drive normally regulates the intake of food to re-attain the energy balance. Whereas in a certain situation, there is an intake of highly pleasant tasting, energy-dense foods due to greedy or reward-based regulation [3]. Obestatin is a 23-acid metabolic peptide. It is a derivative of preproghrelin gene. In 2005, this gene was first isolated from the stomach of rat [4]. However, the expression of obestatin is also found in another GI organ (include pancreas, liver), adipose tissue, skeletal muscle, lungs, thyroids and mammary glands and testes. This presence indicates that obestatin has a multi-functional role. This role can be both centrally and peripherally [5].

It was actually explained as with anorexigenic effect it is a direct antagonist of ghrelin. The consumption of food in a time and dose-dependent manner, attain body weight and intentional motility is decreased by both central and peripheral injection. This is done through the G-protein coupled receptor 39 (GPR 39). GPR 39 is a member of GHSR family but several studies, it was quickly refuted as an obestatin receptor [6]. According to information, it is revealed

that in an autocrine /paracrine manner, obestatin may act through the GPR 39 receptor peripherally, namely as a mitogenic factor in myoblasts. In the adipose tissue and GI system, GPR 39 [7] could arbitrate the metabolic effects of obestatin. Furthermore, with Ghrelin, the positive correlation of obestatin has been shown. This indicates that in obesity and insulin resistance, the levels of both obestatin and Ghrelin may be changed. When stimulated with tumour necrosis factor- $\alpha$ , obesity has been reported to decrease vascular cell adhesion molecule-1 expression in endothelial cells. It is also used to increase oxidized low-density lipoprotein binding to macrophages. Therefore, in the blood pressure regulation., It may also have a potential function [8].

**METHODS AND PATIENTS:**

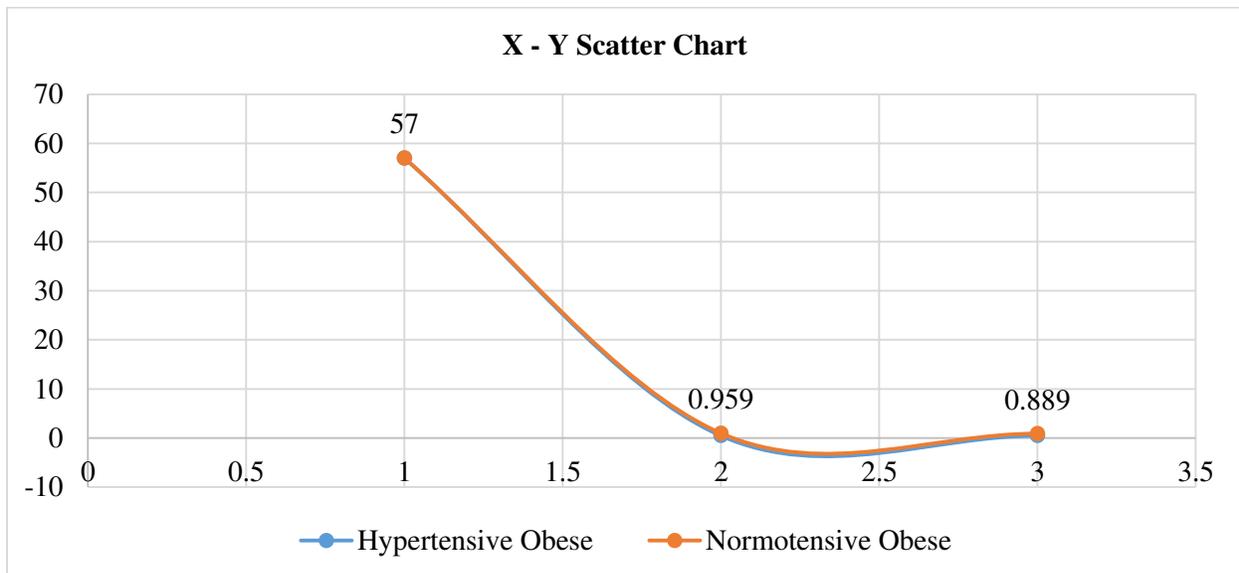
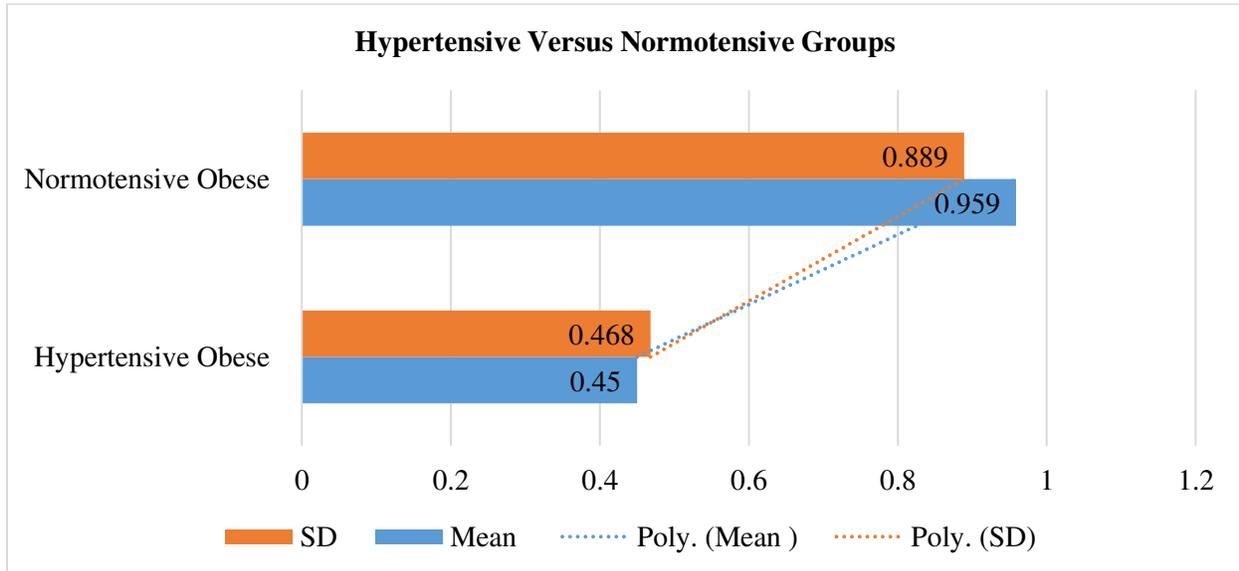
According to the rules and regulations of the hospital's ethical committee, this study was organized. We collected information from 50 fat patients. These patients were also through the heart and cholesterol disorders. The patients were analyzed at Service Hospital, Lahore (January 2017 to July 2017). We also asked the demographic factors to the students. For the patients, we calculated Body mass index (BMI) and waist circumference (WC) and controls as anthropometrical tests. Whereas, by using the spectrophotometric technique, fasting serum glucose (FSG) was measured. Using the enzyme-linked immune sorbent assay (ELISA), each serum sample was analyzed. The analysis was done for obestatin hormone and fasting insulin. In making a comparison of the two-tailed P value of two groups, the SPSS analysis test was used with a significance set at ( $p < 0.05$ ). If the value of two-tailed p was less than 0.05, then the results were believed to be of statistical importance.

**RESULTS:**

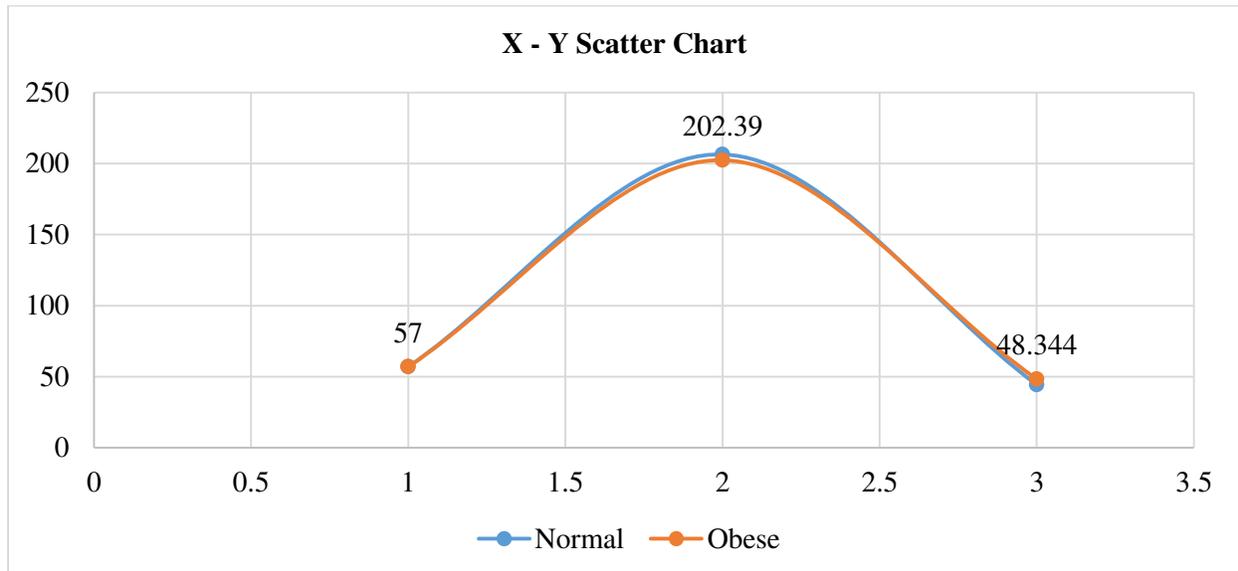
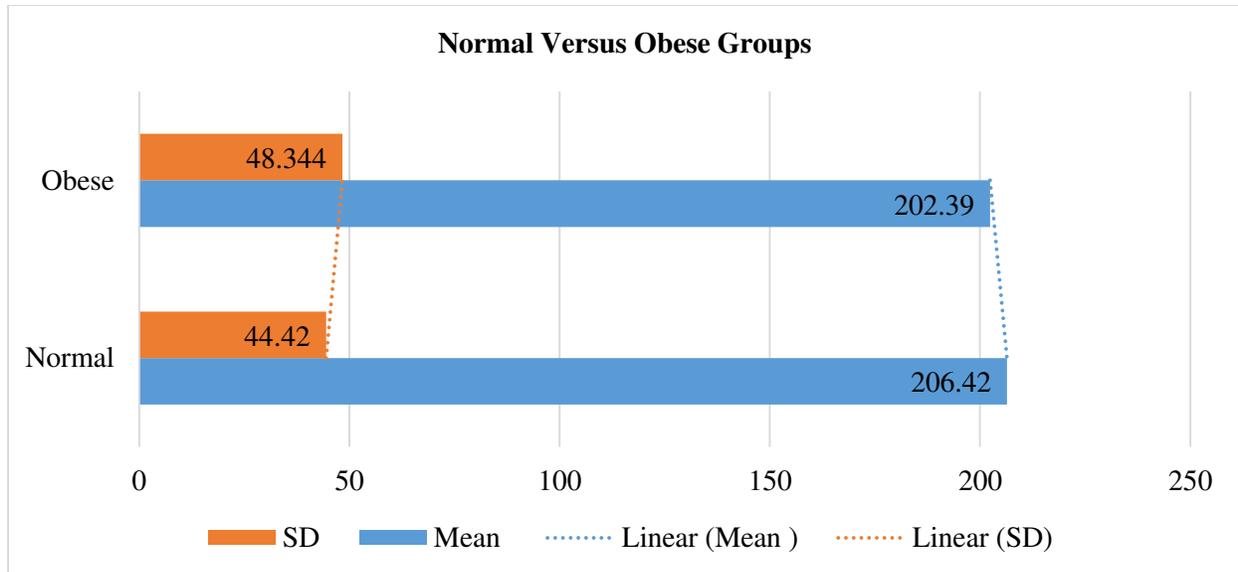
Mean fasting blood cholesterol level was ( $206.42 \pm 44.420$ ) and ( $202.39 \pm 48.344$ ) respectively in normal and obese and the difference was not statistically significant with p-value 0.644.

**Table – I:** Mean fasting obestatin levels Comparison between Normotensive and Hypertensive Obese Patients

Group	Number	Mean	SD	P-Value
Hypertensive Obese	57	0.45	0.468	0.0000
Normotensive Obese	57	0.959	0.889	

**Table – II:** Mean fasting blood cholesterol levels comparison between Obese and Normal Patients

Group	Number	Mean	SD	P-Value
Normal	57	206.42	44.42	0.6440
Obese	57	202.39	48.344	



In hypertension and normotensive obese, means fasting obestatin level was  $(0.450 \pm 0.468)$  and  $(0.959 \pm 0.889)$  respectively. With the value of P as 0.000, the difference between both groups related to mean fasting obestatin levels was statistically important.

#### DISCUSSION:

The integration and control of neuro-circuits of metabolism, thirst, thermoregulation, and sleep overlapping in the hypothalamus is in the hands of hormones and neuropeptides. Central actions of obestatin were also indicated accordingly beside the peripheral effects of obestatin. To note first, when supervised ICV, in fed and fasted male rats, the thirst

is reserved of angiotensin 2 can also be neutralized by pretreatment with obestatin. Moreover, it was also noticed that the consequence of the thirst inhibition which is called dehydration anorexia is the anorexigenic effect of this peptide [9, 10]. In an adult hippocampus, the neurogenesis includes the proliferation, migration and differentiation of progenitor cells. Different conditions which include

hypoxia, addictive drugs, sustained exposure to stress among others weaken the above-mentioned processes. Whereas, proliferation and survival of the hippocampal neurons are promoted by certain hormones and growth factors [11].

All over the world, obesity has become a major problem about public health. At least one-third of the Arabs are fat. This ratio is continuously increasing regardless of increased interest in fitness. Development of insulin resistance, glucose intolerance and diabetes mellitus-2 are due to excess storage of fat. The excessive spreading of obesity is a serious health issue. There is a strong relation of obesity disease. But the diseased procedure relating the hypertension and obesity have not been determined fully in obesity and metabolic syndrome, the positive role of obestatin and ghrelin have been studied. For the obesity and hypertension, alternation in the concentrations of these hormones and in the ghrelin/obestatin ratio may be dangerous factors. There was an important reduction in the obestatin level. This reduction indicates that in an opposing manner, the secretion of the ghrelin and obestatin is regulated by the nutritional status. These findings demonstrate that endogenous Ghrelin actions could be regulated by obestatin. It is also shown that jejunal activity may also be suppressed [12]. In BP regulation and insulin sensitivity, the role of obestatin is not obvious. But for the ghrelin/obestatin ratio, systolic PB has been demonstrated to be an independent predictor. Furthermore, in insulin resistance, concentrations of fasting plasma of obestatin are positively related with whole-body insulin sensitivity [13]. Fat people had lower fasting plasma obestatin concentrations than controls in the present study.

### CONCLUSION:

In obesity, the important role is played by obestatin. There is a direct relation of obestatin with blood glucose level. Moreover, between the observation and both BP and HOMA-IR, there found a clear association. This relation indicates that in BP regulation, obestatin might play a role.

### REFERENCES:

- Berthold, HK, Giannakidou, E, Krone, W. Influence of ghrelin gene polymorphisms on hypertension and atherosclerotic disease. *Hypertens Res* 2010; 33: 155–160.
- Gurriarán-Rodríguez, U, Al-Massadi, O, Roca-Rivada, A. Obestatin as a regulator of adipocyte metabolism and abiogenesis. *J Cell Mol Med* 2011; 15: 1927–1940.
- Schulteis G, Yackey M, Risbrough V, Koob GF. Anxiogenic-like effects of spontaneous and naloxone-precipitated opiate withdrawal in the elevated plus-maze. *Pharmacol Biochem Behav.* 1998;60(3):727-31.
- Li T, Hou Y, Cao W, Yan CX, Chen T, Li SB. Naloxone-precipitated withdrawal enhances ERK phosphorylation in prefrontal association cortex and acumens nucleus of morphine-dependent mice. *Neurosci Lett.* 2010;468(3):348-52.
- Vicennati V, Genghini S, and Iasio DR. Circulating obestatin levels and the ghrelin/obestatin ratio in obese women. *European Journal of Endocrinology* 2007; 157: 295–301.
- Taskin MI, Bulbul E, Adali E, Hismiogulları AA, and Inceboz U. Circulating levels of obestatin and co-peptin in obese and non-obese women with polycystic ovary syndrome. *Eur J Obstet Gynecol Reprod Biol.* 2015 Jun; 189:19-23.
- Yildiz G, Yucel A, Nayon V et al. Evaluation of effects of diet on serum obestatin levels in overweight patients with polycystic ovary syndrome. *Journal of obesity and metabolic research.*2014;1(4):230-237.
- Dixit, VD, Yang, H, Cooper-Jenkins, A. Reduction of T cell-derived ghrelin enhances pro-inflammatory cytokine expression: implications for age-associated increases in inflammation. *Blood* 2009; 113: 5202–5205.
- Kellokoski, E, Kunnari, A, Jokela, M. Ghrelin and obestatin modulate early atherogenic processes on cells: enhancement of monocyte adhesion and oxidized low-density lipoprotein binding. *Metabolism* 2009; 58: 1572–1580.
- Matthews, DR, Hosker, JP, Rudenski, AS. Homeostasis model assessment: insulin resistance and beta-cell function from fasting plasma glucose and insulin concentrations in man. *Diabetologia* 1985; 28: 412–419.
- Kadoglou, NP, Sailer, N, Moutzouglou, A. Visfatin (nampt) and ghrelin as novel markers of carotid atherosclerosis in patients with type 2 diabetes. *Exp Clin Endocrinol Diabetes* 2010; 118: 75–80.
- Li, ZF, Guo, ZF, Yang, SG. Circulating ghrelin and ghrelin to obestatin ratio are low in patients with untreated mild-to-moderate hypertension. *Regul Pept* 2010; 165: 206–209.
- Rhéaume, C, Leblanc, MÈ, Poirier, P. Adiposity assessment: explaining the association between obesity, hypertension and stroke. *Expert Rev Cardiovasc Ther* 2011; 9: 1557–1564.