

Mesopo. Environ. j., Special Issue C :130-135, 2017 ISSN 2410-2598 proceeding of 1<sup>st</sup> National conference of science and Art University of Babylon

Mesopotemia Environmental journal journal homepage:www.bumej.com



# Assess the relationship between leptin levels with classes of obesity in adult of Babylon, Iraq

Amera Kamal Mohamed<sup>1</sup> Alaa Jawad Hassan<sup>2</sup>

1 Environmental research center / Babylon university, Iraq.

2Department of biology, College of Science / Babylon university, Iraq.

Corresponding Author: amira babylon @yahoo.com.

### To cite this article:

Mohamed.A.K, Hassan.A.J, Assess the relationship between leptin levels with classes of obesity in adult of Babylon, Iraq. *Mesop. environ. j.*, 2017, Spicial Issue A.;10-17.

This work is licensed under a <u>Creative Commons Attribution-Non Commercial-No Derivatives 4.0</u> International License.



### Abstract

The aim of this study was to evaluate the relationship between leptin levels and classes of obesity adults, who do not suffer from chronic diseases. This study was performed in department of nutrition at the Murjan teaching hospital / in Babylon province/Iraq, in January 2016. This study included 52 (100%) were patients with obesity ,female were 39(75.0%) and male were 13 (25.0%) for age range from (19-40 years) and mean BMI (32.32 for classes I,36.93 for classes II and 42.15 for class III). Correlation analysis showed that a significant positive correlate (y = 35.71+1.43\*BMI, r = 0.65, P = 0.03) between classes III of obesity depending on BMI and leptin levels ,while the results found there was no significant correlation between classes I and II and leptin levels.

Key words: Body mass index, classes, leptin, obesity.

### Introduction

Obesity is a multifactorial chronic disease involving environmental (social and cultural), genetic, physiologic, metabolic, behavioral and psychological components, which characterized by an increase in body weight that results in excessive fat accumulation, that it may cause a negative effect on health [21; 18].

In adults, overweight and obesity have been defined or classified through a number of different systems [14], including the National Institutes of Health (NIH) and the National Heart, Lung, and Blood Institutes (NHLBI)[16] that are based on a body mass index (BMI) classification system, which is the measured by ratio of weight in kilograms divided by the height in meters squared [16]. Where overweight is defined as a BMI between 25.0 kg/m2 and 29.9 kg/m2 and at risk of becoming obese, while obesity is defined as a BMI higher than 30.0 kg/m2, whereas those with a BMI of 18.5 kg/m2 to 24.9 kg/m2 are considered at low risk for morbidity, also the category of obesity is further divided into subcategories of class I (BMI 30.0–34.9 kg/m2), class II (BMI 35.0–39.9 kg/m2) and class III (BMI  $\geq$ 40 kg/m2) [20].

### Mesop. environ. j. 2016, Spicial Issue C.;130-135

(proceding of 1<sup>st</sup> National conference of science and Art –University of Babylon).

The waist circumference (concentration of fat in the abdominal region) measured in overweight and obese adults in order to assess the abdominal obesity also to measuring BMI where waist circumference (WC)  $\geq$ 40 in 102 cm for men and  $\geq$ 35 in 88 cm for women are considered elevated indicates increased risk of metabolic diseases and the disorder of energy balance [4]. Many European study reported that both body mass index and abdominal adiposity (waist circumference, waist-to-hip ratio) are strong predictors of mortality risk such as type II diabetes mellitus, hypertension, stroke, sleep apnea, osteoarthritis, gallstones and cancer [5,2008;22], and from these risk factors that causes overweight and obesity including; genetic factors, age, race, lack of physical activity, excessive eating or energy intake, medications, pregnancy, sleep deprivation, emotional and/or psychological factors such as depression (21; 9], but the principal cause of overweight and obesity is an imbalance between calories consumed and calories expended as a result of an increased intake of energy-dense foods that are high in fat, salt and sugars but low in vitamins, minerals and other micronutrients as well as a decrease in physical activity due to the increasing sedentary life styles such as amount of time spent watching television and technological developments [5].

Leptin composed of 167 amino acid peptide primarily produced in adipose tissue and circulates in proportion to whole body adiposity, but it is also synthesized in placenta, gastric fundic mucosa, and skeletal muscle and appears to be involved in both short- and long-term regulation of energy homeostasis. [3], furthermore leptin action occurs through receptors expressed centrally and peripherally, one of its main functions is to be an afferent signal to the central nervous system, on a negative feedback basis, this helps regulate the quantity of adipose tissue, body weight, and appetite [1]

The leptin acts on specific hypothalamic receptors and inhibits appetite by counteracting the effects of the orexigenic neuropeptide Y (NPY) [13], the circulating leptin levels promptly increased in response to increased calorie intake therefore many studies indicating that specific types of dietary such as sugars and fats are capable of inducing leptin resistance in experimental rodent models and that diet-induced leptin resistance is capable of increasing energy intake and elevating body weight gain under suitable dietary challenges, whereas the biological mechanisms on multiple levels may cause the dietary induction of leptin resistance, including alterations in the leptin blood-to-brain transport system, in peripheral glucose metabolism, and in central leptin receptor signaling pathways and leptin resistance can occur in the absence of elevated circulating leptin levels and body weight, translation it a potential cause and/or influencing factor to excess body weight gain and obesity [11].

### **Material and Methods**

The study was performed in department of nutrition at the Murjan teaching hospital / in Babylon province/Iraq in January 2016 .This study included 52 (100%) were patients with obesity ,female were 39(75.0%) and male were 13 (25.0%) for age range from (19-40 years). Data were collected through exploration of questionnaires for study participation included: diagnosed as having obesity through defined of WHO for obesity adult. Excluded were patients who suffer from any chronic diseases or imbalance in sex hormones and thyroid, kidney disease, liver, smoking and alcohol intake. Body mass index (BMI) was calculated using the formula BMI= weight (kg)/ height2 (m) 2 and classifying normal weight (BMI 18.5- 24.9)Kg/M2, obesity (BMI 30-39.9) Kg/M2 and morbid obesity (BMI > 40) Kg/M2 .The waist circumference was measured while the subject standing up , at the narrowest point of the torso width-wise, usually just above the belly button ,which is  $\leq 102$  cm in male and $\leq 88$  cm in female [4]. The leptin (LEP) hormones measured after 12 hours of fasting by enzyme linked immune sorbent assay is the complete kit from Ela science- China for the quantitative determination of leptin serum.

### **Statistical Analysis:**

The statistical analysis of this study was made by using SPSS program (Version 15.0) and the data are expressed as the Means and the correlation coefficient and Linear regression was performed with the BMI and leptin .Values were considered statistically significant if the associated P values were lower than 0.05.

### Results

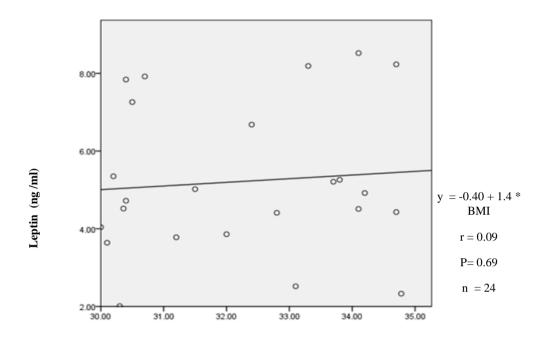
Results of statistical analysis showed there was the mean of age at classes I,II and III were (30.75, 28.29 and 29.72) years respectively ,where mean of BMI at classes I,II and III were (32.23,36.93 and 42.15) Kg/M2 respectively while mean of WC at classes I,II and III were (103.125, 114.882 and 126.272) cm respectively and mean of leptin at classes I,II and III were (5.22, 5.69 and 6.51) ng/ml respectively as showed in table (1). Correlation analysis showed there was positive correlation (y = 35.71+1.43\*BMI, r = 0.65, P = 0.03) between classes III of obesity depending on BMI and leptin levels ,while the results found there was no significant

Mesop. environ. j. 2016, Spicial Issue C.;130-135

(proceding of 1<sup>st</sup> National conference of science and Art –University of Babylon). correlation between classes I and II and leptin levels, as point to in Figure (1), (2) and (3).

Table (1) Distribution of mean Age, BMI, WC and leptin at the three classes of obesity.

Classes of obesity	No (%)	ean Age (years)	Mean BMI (Kg/M <sup>2</sup> )	lean WC(cm)	Iean LEP(ng/ml)
BMI class I(30.0 - 34.9 Kg/m2)	24(46.15%)	30.75	32.23	103.125	5.22
BMI class II (35.0 - 39.9 Kg/m2)	17(32.70%)	28.29	36.93	114.882	5.69
BMI Class III(≥40 Kg/m2)	11(21.15%)	29.72	42.15	126.272	6.51



BMI class I (Kg/M2)

Figure (1): The relationship between body mass indexes (BMI) class I  $\,$  (Kg/M2) and leptin hormone  $\,$  (ng/ml).

**Mesop. environ. j. 2016, Spicial Issue C.;130-135** (proceding of 1<sup>st</sup> National conference of science and Art –University of Babylon).

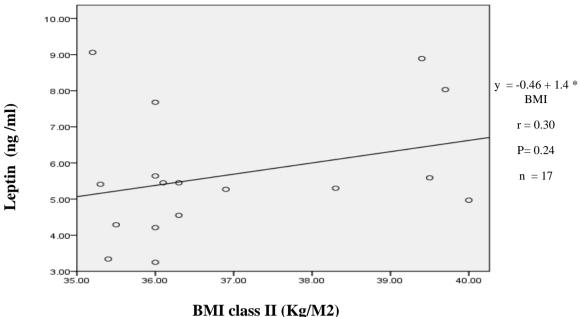


Figure (2): The relationship between body mass indexes(BMI) classes II (Kg/M2) and leptin hormone (ng/ml).

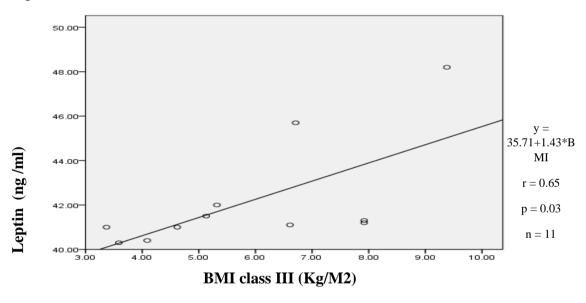


Figure (3): The relationship between body mass indexes(BMI) classes III (Kg/M2) and leptin hormone (ng/ml).

### Discussion

This study showed there was there was positive correlation between classes III of obesity depending on BMI and leptin levels, while the results found there was no significant correlation between classes I and II and leptin levels. This results is agreement with Concidine *et al.* (1996) [8]who founded that obese humans had (on average) leptin levels four times higher than non-obese individuals, also Adil, (2010) [2] reported that is a significant positive correlation between leptin and BMI categories obesity in Saudi female population represented by Makkah community and leptin levels were directly associated with BMI and WC, in addition, serum leptin levels are higher in the omani obese group

### ISSN 2410-2598

### Mesop. environ. j. 2016, Spicial Issue C.;130-135

### (proceding of 1<sup>st</sup> National conference of science and Art –University of Babylon).

and correlate positively with body fatness and obesity and females have higher serum leptin concentrations than males [15], and this agrees with reported range in female Kuwaitis [12] may be that the larger amounts of body fat mass, predominantly subcutaneous fat in women, and Cassabiell *et al* .(1998)[7] suggest that effect of sex steroid hormones which increase leptin production, therefore the level of serum leptin correlates with the fat content of the body [6, 23] where in this study the female represent the majority of obese groups and all of them have abnormal waist circumference.

Other studies have shown that in most obese individuals, leptin levels are either normal or higher than in normal individuals [17], and these results indicate that in obese individuals, there may be a leptin resistance [10], this resistance is believed to play a role in the development of obesity due to the fact that the excess amount of leptin cannot perform its role in controlling food intake, due to the presence of leptin receptor resistance [17], on the other hand leptin resistance may occur directly as a result of obesity, but these may also be a lack of sensitivity to circulating leptin, which could contribute to the a etiology of obesity [15]. This may be due to differences in fat distribution between the obese, in addition , it has been shown that subcutaneous fat produces more leptin than visceral fat, and obese have more visceral fat and less subcutaneous fat, also high intensity and long term exercise probably by increasing the muscle mass, skeletal muscle glucose transport protein by increasing or decreasing the synthesis of fatty acids, insulin sensitivity and glucose uptake by skeletal muscle activity increases [19].

### Acknowledgements

We express our gratitude to the all patients for their cooperation with us and specialized people who give us scientific information on obesity

### References

[1]Abizaid, A. and Horvath, TL. (2008). Brain circuits regulating energy homeostasis. Regul Pept 149:3–10, 2008.

[2]Adil, O. B. Relationship of Leptin hormones with body mass index and waist circumference in Saudi female population of the makkah community. The open obesity Journal, 2, 95-100, 2010.

[3]Ahima, RS. and Flier, JS. Leptin. Annu Rev Physiol; 62: 413- 437, 2000.

[4]Anderson, E.J.; Lustig, M.E.; Boyle, K.E.; Woodlief, T.L.; Kane, D.A.; Lin, C.T.; Price, J.W., 3rd; Kang, L.; Rabinovitch, P.S.; Szeto, H.H.; Houmard, JA.; Cortright, RN.; Wasserman, DH. and Neufer, PD. Mitochondrial H2O2 emission and cellular redox state link excess fat intake to insulin resistance in both rodents and humans. J. Clin. Invest; 119, 573–581, 2009.

[4]Andersson, L.B. Genes and obesity. Ann Med. (28): 5-7, 1996.

[5]Berthoud, H.R. and Morrison, C. The brain, appetite, and obesity. Annu Rev Psychol.; 59:55-92, 2008.

[6]Casimiro-Lopes, G, de Oliveira, AV.; Portella, ES.; Lisboa, PC.; Donangelo, CM.; de Moura, EG.; Koury, JC. Plasma leptin, plasma zinc, and plasma copper are associated in elite female and male judo athletes. Biol Trace Elem Res; 127: 109-115, 2009.

[7]Cassabiell, X.; Pineiro, V. and Peino, R. Gender differences in both spontaneous and stimulated leptin secretion by human omental adipose tissue in vitro: dexamethasone and estradiol stimulate leptin release in women, but not in men. J Clin Endocrinol Metab; 83:2149-2155, 1998.

[8]Concidine, R. V.; Sinha, M.K.; Heiman, M.; Kriauciunas, A.; Stephens, T.; Nyce, M.; Ohannesian, J. P.; Marco, C. C.; McKee, J. and Bauer, L. Serum immunoreactive-leptin concentrations in normal-weight and obese humans. N Engl J Med; 334:292-295, 1996.

**[9]Harrington, D. and Elliot, S.** Weighing the importance of neighbourhood: A multilevel exploration of the determinants of overweight and obesity. Social Science and Medicine; 68: 593-600, 2009.

**Mesop. environ. j. 2016, Spicial Issue C.;130-135** (proceding of 1<sup>st</sup> National conference of science and Art –University of Babylon).

[10]Igel, M.; Becker, W.; Herberg, L.and Joost, H.J. Hyperleptinemia, leptin resistance and polymorphic leptin receptor in the new Zealand obese mouse. Endocrinology; 138 (10): 4234-4, 1997.

[11]Joseph, R.; Vasselli, Philip, J.; Scarpace, R.; Harris, B. S. and William A. B. Dietary Components in the Development of Leptin Resistance. American Society for Nutrition. Adv. Nutr. 4: 164–175, 2013.

[12]Kamal, A.S.; Al-Shoumer, B.; Vasanthy, A.K.; Hanan, A.; Makhlouf, M.and Al-Zaid. M. Leptin levels in Arabs with primaryhyperthyroidism. Ann of Saudi Med; 20:113-118, 2000.

[13]Koch, A.; Weiskirchen, R.; Zimmermann, HW.; Sanson, E.; Trautwein, C.; Tacke, F. (2010). Relevance of serum leptin and leptin-receptor concentrations in critically Illpatients.MediatorsInflamm.Koda, S.; Date, Y.; Murakami, N.; Shimbara, T.; Hanada, T.; Toshinai, K.; Niijima, A.; Furuya, M.; Inomata, N.; Osuye, K.and Nakazato, M.(2005). The role of the vagal nerve in peripheral PYY3-36- induced feeding reduction in rats. Endocrinology; 146(5): 2369-2375, 2010.

**[14] Kuczmarski, R.J. and Flegal, K.M.** Criteria for definition of overweight in transition: background and recommendations for the United States. AmJClinNutr;72:1074-1081, 2000.

[15]Masoud, A.Y and Adel AA. Correlation between serum leptin levels, body mass index and obesity in Omanis. Sultan Qaboos Med J; 6: 28-31, 2006.

[16]National Heart, Lung, and Blood Institute. Obesity Education Initiative Expert Panel Clinical Guidelines on the Identification, Evaluation, and Treatment of Overweight and Obesity in Adults: The Evidence Report. National Institutes of Health Publication; 98-4083, 1998.

[17]Oksanen, L.; Kaprio, J.; Mustajoki, P.and Kontula K. A common pentanucleotide polymorphism of the 3'-untranslated part of the leptin receptor gene generates a putative stem-loop motif in the mRNA and is associated with serum insulin levels in obese individuals. Int J Obes. 22:634–640, 1998.

[18]Reeder, B.A. and Katzmarzyk, P.T. Prevention of weight gain and obesity in adults: asystematic review. Ottawa, ON: Canadian Task Force on Preventive Health Care, 2006.

[19]Soraya, K.; Reza, N.; Mehrzad, M. and Mehdi M. Leptin and insulin resistance in young adult obese females: effect of eight weeks resistance training. Research in Endocrinology, Article, 665365, 8, 2014.

[20]Weisberg, SP.; McCann, D.; Desai, M.; Rosenbaum, M.; Leibel, RL and Ferrante, A.W. Jr. Obesity is associated with macrophage accumulation in adipose tissue. J Clin Invest; 112: 1796–1808, 2003.

[21]**WHO**, Preventing and Managing the Global Epidemic.World Health Organization: Geneva, Switzerland, Technical Report Series 894 Obesity, 2000.

[22]WHO, (2010b). Global Infobase (accessed November 23, 2010).

[23]Williams, KW.; Scott, MM.and Elmquist, JK. From observation to experimentation: leptin action in the mediobasal hypothalamus. Am J Clin Nutr; 89: 985-90, 2009.