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Correlation of Ghrelin and C-Peptide Hormones and Some Other Biochemical Factors With Women Obesity

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Abstract

The study was conducted in National Center for Obesity Treatment in Mosul city, from December 2020 to March 2021, and it included (112) woman aged between (15 to > 55) year, (84) of them were obese women and (28) of them were women with normal weight, depending on body mass index (BMI) value. They were divided into four age groups [(15-25), (25-35), (35-45), (45 to > 55) year]. All women (not suffering from diabetic mellitus or heart diseases). Blood samples were collected from (12-14 h) fasted women, serum samples were separated and stored in capped tubes, then Ghrelin hormone (Ghr), lipid profile [total cholesterol (TC), Triglyceride (TG), High density lipoprotein-cholesterol (HDL-c), Low density lipoprotein-cholesterol (LDL-c), Very low-density lipoprotein-cholesterol (VLDL-c), Atherogenic index (AI)], and C-peptide hormone beside Glucose had been measured. The results indicated that the level of ghrelin hormone decreased in obesity (the differences between control and obese groups were significant at P≤0.05) but lipid profile and insulin resistance increased except HDL decrease in obesity (the differences between control and obese groups were significant at P≤0.05).

Keywords: Obesity; Ghrelin; C-peptide; Lipid profile; Insulin resistance.

1. Introduction

Obesity is an increase in body fat that causes significant damage to health, and obesity occurs when the size and number of fat cells in the body increases. A person of normal weight has about 30-35 billion adipocytes, and when a person gain weight, these adipocytes increase in size first and then in number, and conversely, when a person begins to lose weight, the size of adipocytes decreases, but their number remains the same in general[1]. In parallel with the rising obesity epidemic growing the number of studies on the consequences of obesity has increased dramatically[2]. This interest has led to the formation of a relatively new branch of epidemiology focused on obesity. Obesity is defined as an abnormal or excessive accumulation of fat that may harm health, and a person with obesity is defined as that person who has excess fatty tissue and has a BMI ≥ 30 kg/m^2[3]. Body Mass Index or BMI is a simple and widely used method for estimating body fat mass[4]

BMI = Kg/m^2

Ghrelin (Ghr) is a peptide hormone that plays an important role in obesity, that has a unique structure with 28 amino-acids and an n-octanoyl ester (any fatty acid ester in which the carboxylic acid component is octanoic acid) at its third serine residue, which is essential for its potent stimulatory activity on somatotroph secretion [5].The pathophysiological mechanism responsible for Growth hormone hyposecretion in obesity is probably multifactorial, and there is probably a defect in ghrelin secretion [6]. Ghrelin levels in blood decrease during periods of feeding, due to its orexigenic (appetite stimulant) and metabolic effects, ghrelin has a potential benefit in antagonizing protein breakdown and weight loss in catabolic conditions such as cancer cachexia, renal and cardiac disease, and age-related frailty. Theoretically ghrelin receptor antagonists could be employed as anti-obesity drugs, blocking the orexigenic signal. By blocking the constitutive receptor activity, inverse agonists of the ghrelin receptor may lower the set-point for hunger, and could be used for the treatment of obesity [7, 8]. Abnormalities in lipid metabolism are very commonly observed in obese. Approximately 60-

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70% of patients with obesity are dyslipidemic, obesity has been associated with an increased risk for metabolic syndrome in adults [9]. The metabolic defects that ensue in obesity include increased levels of free fatty acids resulting from insulin resistance, increased total Cholesterol, LDL-Cholesterol, VLDL and triglycerides and decrease in HDL-Cholesterol. It is most likely that
presentation of increased free fatty acids and insulin in liver as a function of obesity is primarily responsible for over production of VLDL and this is probably the key to increased LDL [10]. Obesity increases the risk of cardiovascular diseases and diabetes [11], especially when the extra fat is accumulated to central and intra-abdominal depots [12]. The increased cardiometabolic risk in obesity is at least partly mediated through atherogenic dyslipidaemia characterized by an increase in serum triglycerides, large very low-density lipoprotein.
One of the conditions caused by obesity is insulin resistance it is a physiological condition in which the insulin hormone is less effective in reducing the level of glucose in the blood and resulting in cells losing the ability to absorb glucose and increasing its level in the blood [13]
Insulin and C-peptide are co-secreted from the pancreas in an equimolar ratio. This phenomenon has been exploited to assess prehepatic insulin secretion in humans. Unlike insulin, C-peptide is not significantly cleared by the liver and the kinetics of C-peptide are linear at physiological and supraphysiologic plasma C-peptide concentrations [14]. Therefore, it has been suggested that peripheral C-peptide levels more closely reflect pancreatic insulin secretion than do peripheral insulin levels. One study reported that stimulated serum C-peptide levels during a mixed-meal tolerance test are a gold-standard measure of endogenous insulin secretion. Based on these results, we hypothesized that the insulin resistance index based on C-peptide levels may be superior to an index based on insulin levels. Homeostatic model assessment (HOMA) is a method used to quantify insulin resistance and beta-cell function from basal (fasting) glucose and insulin or C-peptide concentrations. We can use c-peptide instead of insulin in homeostasis model assessment (HOMA) to estimate insulin resistance and beta cell function[15]. C-peptide hormone has a circulating half-life of 30 minutes, which is longer than the 5-10 minute half-life for insulin, due to of these differences in half-lives, we used c-peptide to estimate insulin resistance[13].

2.The aim of this study

Aims of this research are the comparison of ghrelin and Lipid profile beside the C-peptide and Insulin resistance levels between obese and normal women

3.Materials and methods

3.1. Subjects

The study included (112) women aged between (15 to > 55) year, (84) of them were obese (BMI > 30Kg/m^2) and (28) were normal weight (BMI < 25Kg/m^2) women.
All women (not suffering from diabetic mellitus and heart diseases and hypertension).
They were divided into four age groups [(15-25), (25-35), (35-45), (45 to > 55) year].
Each group was divided into each group Control (BMI < 25Kg/m^2), Obesity1 (BMI > 30Kg/m^2), Obesity2 (BMI > 35Kg/m^2), and Obesity3 (BMI > 40Kg/m^2).

3.2. Collection of blood samples

Blood samples (5ml) were collected from 12-14 h fasted women. Serum samples were separated and stored in capped tubes under (-20°C) temperature to be used later.

3.3 Estimation the level of Ghrelin hormone in serum

Ghrelin was estimated by using the ready-made analysis kit from Bioassay Technology Laboratory (BT LAB) Chinese company and depending on (Enzyme-Linked Immunosorbent Assay, ELISA technique) [16].

3.4. Estimation of Total Cholesterol (TC) in serum

Cholesterol was estimated by using ready-made analysis (kit) from the French company BIOLABO, based on the Enzymatic method [17].

3.5. Estimation of Triglycerides (TG) in serum

Triglycerides was estimated by using ready-made analysis (kit) from the French company BIOLABO, based on the Enzymatic method [18]

3.6. Estimation of High-Density Lipoprotein-cholesterol (HDL-c) in serum

High-density lipoprotein-cholesterol (HDL-c) was estimated by using ready-made analysis (kit) from the French company BIOLABO, based on the sedimentation method [19].

3.7. Estimation of Low-Density Lipoprotein-cholesterol (LDL-c) in serum

By using Friedewald equation LDL-c was calculated [19].

LDL Conc. = Conc. Of Cholesterol – HDL Conc. – TG/5
(mg/dl) or (mmol/l)

3.8. Estimation of Very Low-Density Lipoprotein-cholesterol (VLDL-c) in serum

The VLDL-c is calculated by using the following equation [20].
VLDL Conc. = Triglycerides/5
(mg/dl) or (mmol/l)

3.9. Estimation of Atherogenic Index (AI)in serum

The Atherogenic Index is calculated by using the following equation [20].
Atherogenic Index (AI) = TC/HDL-c

3.10. Estimation of C-peptide Hormone in serum

C-peptide was estimated by using ready-made analysis (kit) from the USA company Ortho Clinical Diagnostics, by using VITROS ECIQ device based on the Chemiluminescent immunoassay technique (CLIA) [21].

3.11. Estimation of Glucose in serum

Glucose concentration was estimated by using ready-made analysis (kit) from the UK company Randox based on the enzymatic method [22].

3.12. Estimation of Insulin resistance in serum

The Insulin resistance is calculated by using the following equation [23].
20/ (fasting C-peptide × fasting Glucose concentration)

4. Statistical analysis

The result were analyzed statistically by mean of the statistical program SPSS 14.0 The data were statistically analyzed according to the analysis of variance (ANOVA) test using the statistical program and the arithmetic means were compared using Duncan Multiple Range Test and below the level of significance p≤0.05

5.Results and Discussion

5.1. Ghrelin hormone

The result in the table (1) show that serum Ghrelin in all grades obesity groups was significantly decrease from the control group at P≤0.05

Table (1) Comparison of serum ghrelin between control group and grades obesity

Age (Year)	Mean ± SE Ghrelin hormone ng/ml			
	Control	Obesity 1	Obesity 2	Obesity 3
Group1 (n=6)	2.30±0.26 c	1.24±0.04 b	1.20±0.04 b	0.52±0.07 a
Group2 (n=6)	6.61±0.64 c	3.81±0.24 b	2.93±0.11 b	0.67±0.04 a
Group3 (n=6)	3.77±0.13 c	2.43±0.19 b	1.37±0.18 a	0.98±0.06 a
Group4 (n=6)	4.71±0.27 c	2.14±0.20 b	1.13±0.11 a	1.16±0.09 a

*The different letters in the same row indicate the presence of significant differences at the level P≤0.05.
* Similar letters in the same row indicate that there are no significant differences.

The differences in the same age group:

Group 1 and 2
The results obtained that there was a significant decrease at P≤0.05 between obesity 2 and obesity 3, while the differences between obesity1 and obesity2 was not significant.

Also, in group 3 and 4 we noted a significant decrease at P≤0.05 in obesity 1 and 2, while the differences between obesity2 and obesity3 was not significant.

This result is identical to previous research [8, 24, 25] which indicated that the Ghrelin hormone is lower in obese groups compared with control group. This decrease in Ghrelin concentration observed in obesity represent a physiological adaptation to the positive energy balance associated with obesity [26].

5.2. Lipid profile
5.2.1 Total Cholesterol

Table (2) Comparison of total cholesterol concentration between control group and grades obesity

Age (Year)	Mean ± SE TC mmol/L			
	Control	Obesity 1	Obesity 2	Obesity 3
Group1 (n=7)	1.81±0.18 a	3.71±0.25 b	4.08±0.59 b	6.10±0.59 c
Group2 (n=7)	1.35±0.10 a	3.67±0.36 b	2.81±0.31 b	4.92±0.57 c
Group3 (n=7)	1.59±0.19 a	2.62±0.16 b	3.33±0.08 c	4.42±0.40 d
Group4 (n=7)	1.31±0.15 a	2.97±0.17 b	4.38±0.39 c	6.08±0.75 d

*The different letters in the same row indicate the presence of significant differences at the level P≤0.05.
* Similar letters in the same row indicate that there are no significant differences.

The result in the table (2) show that the total cholesterol in all grades obesity groups was significantly increase from the control group at P≤0.05

The differences in the same age group:

Group1 and 2
The differences between (obesity2 and obesity3) was significant at P≤0.05 while the differences between (obesity1 and obesity2) was not significant.
Group 3 and 4
The differences between (obesity1 and obesity2), (obesity2 and obesity3) was significant at P≤0.05.

5.2.2. Triglyceride

Table (3) Comparison of triglyceride concentration between control group and grades obesity

Age (Year)	Mean ± SE TG mmol/L			
	Control	Obesity 1	Obesity 2	Obesity 3
Group1 (n=7)	0.55±0.11 a	1.41±0.31 b	1.60±0.21 b	2.34±0.20 c
Group2 (n=7)	0.50±0.03 a	1.55±0.09 b	1.30±0.14 b	2.46±0.25 c
Group3 (n=7)	0.42±0.02 a	1.88±0.31 b	3.21±0.30 c	4.62±0.63 d
Group4 (n=7)	0.44±0.05 a	1.52±0.10 b	3.31±0.34 c	4.70±0.63 d

*The different letters in the same row indicate the presence of significant differences at the level P≤0.05.
* Similar letters in the same row indicate that there are no significant differences.

The result in the table (3) show that the Triglyceride in all grades obesity groups was significantly increase from the control group at P≤0.05

The differences in the same age group:

Group1 and 2
The differences between (obesity2 and obesity3) was significant at P≤0.05 while the differences between (obesity1 and obesity2) was not significant.

Group 3 and 4
The differences between (obesity1 and obesity2), (obesity2 and obesity3) was significant at P≤0.05.

5.2.3. High-Density Lipoprotein-cholesterol (HDL-c)

Table (4) Comparison of HDL-c between control group and grades obesity

Age (Year)	Mean ± SE HDL-c mmol/L			
	Control	Obesity 1	Obesity 2	Obesity 3
Group1 (n=7)	1.47±0.08 c	1.01±0.15 b	0.96±0.14 b	0.30±0.06 a
Group2 (n=7)	1.38±0.09 c	0.97±0.09 b	0.87±0.19 b	0.29±0.06 a
Group3 (n=7)	1.31±0.06 c	0.90±0.10 b	0.44±0.08 a	0.28±0.03 a
Group4 (n=7)	1.08±0.07 c	0.78±0.07 b	0.35±0.06 a	0.27±0.03 a

*The different letters in the same row indicate the presence of significant differences at the level P≤0.05.
* Similar letters in the same row indicate that there are no significant differences.

The result in the table (4) show that HDL in all grades obesity groups was significantly decrease from the control group at P≤0.05

The differences in the same age group:
Group1 and 2
The differences between (obesity2 and obesity3) was significant at P≤0.05 while the differences between (obesity1 and obesity2) was not significant.

Group 3 and 4
The differences between (obesity1 and obesity2) was significant at P≤0.05 while the differences between (obesity2 and obesity3) was not significant.

5.2.4 Low-Density Lipoprotein-cholesterol (LDL-c)

Table (5) Comparison of LDL-c between control group and grades obesity

Age (Year)	Mean ± SE LDL-c mmol/L			
	Control	Obesity 1	Obesity 2	Obesity 3
Group1 (n=7)	1.87±0.51 a	2.80±0.22 b	4.12±0.16 b	6.58±0.24 c
Group2 (n=7)	2.80±0.13 a	4.02±0.37 b	5.15±0.19 b	7.08±0.53 c
Group3 (n=7)	2.70±0.35 a	4.41±0.36 b	6.45±0.32 c	9.08±0.27 d
Group4 (n=7)	3.88±0.39 a	5.90±0.02 b	7.75±0.34 c	10.82±0.68 d

*The different letters in the same row indicate the presence of significant differences at the level P≤0.05.
* Similar letters in the same row indicate that there are no significant differences.
The result in the table (5) show that the LDL-c in all grades obesity groups was significantly increase from the control group at P≤0.05

The differences in the same age group:
Group1 and 2
The differences between (obesity2 and obesity3) was significant at P≤0.05 while the differences between (obesity1 and obesity2) was not significant.
Group 3 and 4
The differences between (obesity1 and obesity2), (obesity2 and obesity3) was significant at P≤0.05

5.2.5 Very Low-Density Lipoprotein (VLDL)

Table (6) Comparison of VLDL between control group and grades obesity

Age (Year)	Mean ± SE VLDL mmol/L			
	Control	Obesity 1	Obesity 2	Obesity 3
Group1 (n=7)	0.11±0.02 a	0.28±0.06 b	0.32±0.04 b	0.47±0.04 C
Group2 (n=7)	0.10±0.01 a	0.31±0.01 b	0.26±0.03 b	0.49±0.05 C
Group3 (n=7)	0.08±0.01 a	0.37±0.06 b	0.64±0.06 c	0.92±0.13 D
Group4 (n=7)	0.08±0.01 a	0.30±0.02 b	0.66±0.07 c	0.94±0.13 D

*The different letters in the same row indicate the presence of significant differences at the level P≤0.05.
* Similar letters in the same row indicate that there are no significant differences.

The result in the table (6) show that the VLDL in all grades obesity groups was significantly increase from the control group at P≤0.05

The differences in the same age group:

Group1 and 2
The differences between (obesity2 and obesity3) was significant at P≤0.05 while the differences between (obesity1 and obesity2) was not significant.

Group 3 and 4
The differences between (obesity1 and obesity2), (obesity2 and obesity3) was significant at P≤0.05.

5.2.6 Atherogenic Index (AI)

Table (7) Comparison of AI between control group and grades obesity

Age (Year)	Mean ± SE AI			
	Control	Obesity 1	Obesity 2	Obesity 3
Group1 (n=7)	1.42±0.34 a	3.14±0.59 b	3.85±0.40 b	6.42±0.53 c
Group2 (n=7)	2.71±0.18 a	4.28±0.29 b	4.42±0.43 b	6.42±0.53 c
Group3 (n=7)	1.71±0.30 a	3.14±0.34 b	4.42±0.37 c	5.57±0.61 d
Group4 (n=7)	1.42±0.20 a	3.28±0.18 b	4.57±0.30 c	6.85±0.42 d

*The different letters in the same row indicate the presence of significant differences at the level P≤0.05.
* Similar letters in the same row indicate that there are no significant differences.

The result in the table (7) show that the AI in all grades obesity groups was significantly increase from the control group at P≤0.05

The differences in the same age group:

Group1 and 2
The differences between (obesity2 and obesity3) was significant at P≤0.05 while the differences between (obesity1 and obesity2) was not significant.

Group 3 and 4
The differences between (obesity1 and obesity2), (obesity2 and obesity3) was significant at P≤0.05.
These results in table (2-7) are agree with [27], which indicate the alterations in body fat distributions are associated with changes in lipids and lipoproteins . Obesity is a risk factor for adult coronary heart disease and is in increasing order among young people and adults [28, 29]. Various lipid/lipoprotein abnormalities have been observed in obese individuals, including elevated total cholesterol, triglycerides, LDL-c, VLDL, and AI while lower HDL-c levels. Of these indicators, changes in triglyceride and HDL-c levels are most consistent and pronounced [30].

5.3. Insulin Resistance by estimation of C-peptide and glucose conc.

5.3.1. C-peptide hormone

Table (8) Comparison of C-peptide hormone between control group and grades obesity

Age (Year)	Mean ± SE C-peptide ng/ml			
	Control	Obesity 1	Obesity 2	Obesity 3
Group1 (n=6)	1.58±0.17 a	2.60±0.35 b	3.43±0.23 c	3.80±0.31 c
Group2 (n=6)	1.11±0.04 a	2.46±0.34 b	3.98±0.45 c	4.40±0.36 c
Group3 (n=6)	1.78±0.39 a	3.28±0.13 b	3.85±0.34 b	5.05±0.56 c
Group4 (n=6)	2.10±0.19 a	3.30±0.21 b	4.06±0.17 b	5.03±0.51 c

*The different letters in the same row indicate the presence of significant differences at the level P≤0.05.
* Similar letters in the same row indicate that there are no significant differences.

The result in the table (8) show that the C-Peptide in all grades obesity groups was significantly increase from the control group at P≤0.05

The differences in the same age group:

Group1 and 2
The differences between (obesity1 and obesity2) was significant at P≤0.05 while the differences between (obesity2 and obesity3) was not significant.
Group 3 and 4
The differences between (obesity2 and obesity3) was significant at P≤0.05 while the differences between (obesity1 and obesity2) was not significant.

5.3.2. Glucose Concentration

Table (9) Comparison of glucose concentration between control group and grades obesity

Age (Year)	Mean ± SE Glucose mmol/L			
	Control	Obesity 1	Obesity 2	Obesity 3
Group1 (n=7)	5.18±0.12 a	5.90±0.31 b	6.68±0.23 c	6.82±0.23 c
Group2 (n=7)	4.97±0.15 a	6.57±0.32 b	7.37±0.17 c	7.56±0.39 c
Group3 (n=7)	4.91±0.14 a	6.92±0.51 b	7.55±0.49 b	9.21±0.20 c
Group4 (n=7)	5.12±0.18 a	6.90±0.35 b	7.64±0.29 b	8.45±0.29 c

*The different letters in the same row indicate the presence of significant differences at the level P≤0.05.
* Similar letters in the same row indicate that there are no significant differences.

The result in the table (9) show that the Glucose in all grades obesity groups was significantly increase from the control group at P≤0.05.

The differences in the same age group:

Group1 and 2
The differences between (obesity1 and obesity2) was significant at P≤0.05 while the differences between (obesity2 and obesity3) was not significant.

Group 3 and 4
The differences between (obesity2 and obesity3) was significant at P≤0.05 while the differences between (obesity1 and obesity2) was not significant.

5.3.3. Insulin Resistance

Table (10) Comparison of insulin resistance between control group and grades obesity

Age (Year)	Mean ± SE Insulin Resistance			
	Control	Obesity 1	Obesity 2	Obesity 3
Group1 (n=6)	2.64±0.34 c	1.76±0.33 b	0.91±0.09 a	0.76±0.04 a
Group2 (n=6)	3.93±0.18 c	1.46±0.31 b	0.81±0.17 a	0.62±0.08 a
Group3 (n=6)	4.55±0.27 c	2.20±0.09 b	2.06±0.32 b	0.50±0.05 a
Group4 (n=6)	2.15±0.24 c	1.33±0.11 b	1.13±0.20 b	0.39±0.03 a

*The different letters in the same row indicate the presence of significant differences at the level P≤0.05.
* Similar letters in the same row indicate that there are no significant differences.

The result in the table (10) show that the Insulin resistance in all grades obesity groups was significantly different from the control group at P≤0.05

The differences in the same age group:

Group1 and 2
The differences between (obesity1 and obesity2) was significant at P≤0.05 while the differences between (obesity2 and obesity3) was not significant.

Group 3 and 4
The differences between (obesity2 and obesity3) was significant at P≤0.05 while the differences between (obesity1 and obesity2) was not significant.
These results in table (8,9,10) are agree with [31], which indicate obesity is associated with an increased risk of developing insulin resistance. In obese individuals, adipose tissue releases increased amounts of non-esterified fatty acids, glycerol, hormones, pro-inflammatory cytokines and other factors that are involved in the development of insulin resistance. When insulin resistance is accompanied by dysfunction of pancreatic islet β-cells — the cells that release insulin — failure to control blood glucose levels results. Abnormalities in β-cell function are therefore critical in defining the risk and development of type 2 diabetes.

6. Conclusions

The results of this research revealed that serum Ghrelin decreased and negatively correlated with obesity. Beside lipid profile parameters except HDL had been raised and were positively correlated with obesity that cause insulin resistance and leads to diabetes.

7. References

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