

## Assess the relationship between insulin fasting levels and healthy obesity in adults in the province of Babylon, Iraq.

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### Abstract

The aim of this study was to evaluate the relationship between fasting insulin levels and obesity adults, who do not suffer from chronic diseases and evaluate insulin resistance by HOMA-IR in the same groups. This study was performed in AL-Qasim green university / College of Agriculture/ Laboratory bio-technology with department of nutrition at the Murjan teaching hospital / in Babylon province/Iraq, for the period from June 2015 to January 2016. This study included 89 (100%) were patients with obesity 52 (58.43 %) and 37(41.57%) were control for age range from (19-40) years. This study was found significant association( $P \leq 0.05$ ) between obese and control regarding the age (OR 7.46, CI (2.51-22.18)), gender (OR 3.16, CI (1.28-7.78)), WC (Fisher  $P \leq 0.05$ ), presence of history of previous obesity (OR 2.85, CI (1.19-6.82)), suffering from Psychosocial stress (OR 0.25, CI (0.10-0.63)) and eating more (OR 0.04, CI (0.01-0.13)) while there was no significant association ( $P \geq 0.05$ ) in exercise daily activity (OR 2.19, CI (0.89-5.33)). This study shows there was significant mean differences ( $P \leq 0.05$ ) of age, body mass index( $\text{Kg}/\text{M}^2$ ), waist circumference(cm) and FBG (mmol/L) between control with obese and in FBG (mmol/L) for both gender. While there were no significant mean differences ( $P \geq 0.05$ ) of age, body mass index( $\text{Kg}/\text{M}^2$ ), waist circumference(cm), insulin( $\text{ng}/\text{mL}$ ), HOMA-IR and TG (mmol/L) between obese and control groups for both gender. This study also shows that the significant mean differences ( $P \leq 0.05$ ) in obese group in levels of FBG (mmol/L), HOMA-IR and TG (mmol/L) at the three class of obesity in obese group, While there was no significant mean differences ( $P \geq 0.05$ ) differences in levels of Insulin( $\text{ng}/\text{mL}$ ), despite the high level at class III of obesity. Correlation analysis showed there was positive correlation between BMI with insulin fasting for the obese group. This study of Correlation analysis also showed positive correlation between BMI and insulin fasting and between BMI with HOMA-IR for female in obese group.

**Key words:** Body mass index, homeostasis model assessments, insulin resistance, obesity.

## **Introduction:**

Obesity is a complex, multifactorial, chronic disease involving environmental (social and cultural), genetic, physiologic, metabolic, behavioral and psychological components. Obesity, characterized by an increase in body weight that results in excessive fat accumulation, that it may cause a negative effect on health (1, 2). Where Overweight is defined as a BMI between 25.0 kg/m<sup>2</sup> and 29.9 kg/m<sup>2</sup> and at risk of becoming obese, and obesity is defined as a BMI higher than 30.0 kg/m<sup>2</sup>, whereas those with BMI of 18.5kg/m<sup>2</sup> to 24.9kg/m<sup>2</sup> are considered at low risk for morbidity, the category of obesity is further divided into subcategories of class I (BMI 30.0–34.9 kg/m<sup>2</sup>), class II (BMI 35.0–39.9 kg/m<sup>2</sup>), and class III (BMI ≥40 kg/m<sup>2</sup>) (3). The waist circumference (concentration of fat in the abdominal region) it is measured in overweight and obese adults to assess for abdominal obesity addition to measuring BMI where waist circumference (WC) of ≥40 in (102 cm) for men and ≥35 in (88 cm) for women are considered elevated and indicates increased risk of metabolic diseases and the disorder of energy balance (4). Overeating either due to hormonal imbalance or to bad food behavior and some others diet problems, increase body weight and deposition of fat in the body, when the increment of fat exceed the normal weight by 20% lead to the production of obesity (5,6). Studies have shown that the basal level of plasma insulin and the elevated level of insulin in response to increased blood glucose are related to the body fat mass (7), where Gloy et al., (2010) found although insulin level increases during forced weight gain, it returns to a normal level on the first day of recovery. Levels of insulin are determined to a great range by peripheral insulin sensitivity, and this is related to total body fat stores and fat distribution, with visceral fat being a key determinant of insulin sensitivity (9). Adipose tissue seems to play a key role in the pathogenesis of insulin resistance (IR) through several released metabolites, hormones, and adipocytokins that can affect different steps in insulin action (10) Adipocytes produce nonesterified fatty acids, which inhibit carbohydrate metabolism via substrate competition and impaired intracellular insulin signaling (10). Obese individuals develop insulin resistance to the cellular actions of insulin, characterized by an impaired ability of insulin to inhibit glucose output from the liver and to promote glucose uptake in fat and muscle (11). where insulin resistance develops gradually in adipocytes and is paralleled by an increased excretion of insulin from β-cells in the pancreatic islets of Langerhans to meet the demands of the adipose tissue, resulting in hyperinsulinemia. Insulin resistance in adipocytes precedes the development of insulin resistance in other insulin responsive tissues such as skeletal muscle and liver and estimate of insulin resistance derived from fasting glucose and insulin levels, with higher levels representing greater degrees of insulin resistance such measured with simple indices name the homeostasis model assessment-estimated insulin resistance (HOMA-IR) (12, 14).

## **Materials and method:**

The study was performed in AL-Qasim green university / College of Agriculture/ Laboratory biotechnology with department of nutrition at the Murjan teaching hospital / in Babylon province/Iraq for the period from June 2015 to January 2016 for aged 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, genetic predisposition to obesity in the family, registration of the person exercising the activity daily, and recording does the person's psychological pressure and

whether eating more food. 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  $BMI = \text{weight (kg)} / \text{height}^2 \text{ (m)}^2$  and classifying normal weight (BMI 18.5- 24.9), obesity (BMI 30-39.9) and morbid obesity (BMI > 40) (14). 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 (13, 15). The Insulin (INS) fasting 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 insulin serum. where the level of glucose were measured in fasting serum after 12 hours of fasting according to the principle of enzymatic oxidation presence of an enzyme Glucose \_Oxidase (GOD) (Kit) from Audit Company. The estimation of insulin resistance by the homeostasis model assessment-estimated insulin resistance (HOMA-IR), calculated multiplying fasting blood insulin (FBI) by fasting blood glucose (FBG), then dividing by the constant 22.5 (14). Measuring the level of triglycerides (TG) in fasting serum after 12 hours of fasting according to the principle of enzymatic hydrolysis from BIOLABO Company and the Rondo Company.

### **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, Standard Error, One –sample T Test, One – way ANOVA and the Chi– Square test, Odds ratio to identify the risk factors of obesity, Correlation coefficient and Linear regression was performed with the BMI, insulin and We calculated HOMA-IR as  $\text{plasma glucose [mmol/l]} / 22.5$  (14). Values were considered statistically significant if the associated P values were lower than 0.05.

### **Results:**

Results of statistical analysis showed there was significant association ( $P \leq 0.05$ ) between obese and control regarding the age (OR 7.46, CI (2.51-22.18)), gender (OR 3.16, CI (1.28-7.78)), WC (Fisher  $P \leq 0.05$ ), presence of history of previous obesity (OR 2.85, CI (1.19-6.82)), suffering from Psychosocial stress (OR 0.25, CI (0.10-0.63)) and eating more (OR 0.04, CI (0.01-0.13)), while there was no significant association ( $P \geq 0.05$ ) in exercise daily activity (OR 2.19, CI (0.89-5.33)) shown in table (1). This study shows there was significant mean differences ( $P \leq 0.05$ ) of age, body mass index ( $\text{Kg}/\text{M}^2$ ), waist circumference (cm) and FBG (mmol/L) between control with obese and in FBG (mmol/L) for both gender. While there were no significant mean differences ( $P \geq 0.05$ ) of age, body mass index ( $\text{Kg}/\text{M}^2$ ), waist circumference (cm), insulin (ng/mL), HOMA-IR and TG (mmol/L) between obese and control groups for both gender, as shown in table (2).

This study also shows that the significant mean differences ( $P \leq 0.05$ ) in obese group in levels of FBG (mmol/L), HOMA-IR and TG (mmol/L) at the three class of obesity in obese group, While there was no significant mean differences ( $P \geq 0.05$ ) differences in levels of Insulin (ng/mL), despite the high level at class III of obesity, show in table (3). Correlation analysis showed there was positive correlation between BMI with insulin fasting ( $y = -0.77 + 0.79 * \text{BMI}$ ,  $r = 0.32$ ,  $P \leq 0.05$ ) and between BMI with HOMA-IR ( $y = -1.87 + 0.21 * \text{BMI}$ ,  $r = 0.37$ ,  $P \leq 0.05$ ) for the obese group. This study of Correlation analysis also showed positive correlation between BMI and insulin fasting ( $y = -0.77 + 0.79 * \text{BMI}$ ,  $r = 0.31$ ,  $P \leq 0.05$ ) and between BMI with HOMA-IR ( $y = -1.93 + 0.22 * \text{BMI}$ ,  $r = 0.41$ ,  $P \leq 0.05$ ), for female in obese group, Show in Figure (1), (2).

Table (1) The association of study groups by study variables.

Variable	Control (n=37)	Obese (n=52)	$\chi^2$	P- value	Odds ratio	95% CI
<b>Age</b>						
(19-29) year	32(86.49%)	24 (46.15%)	14.90	<0.001*	7.46	2.51-22.18
(30-40) year	5 (13.51%)	28 (53.85%)				
<b>Gender</b>						
Male	19 (51.35%)	13 (25.0%)	6.44	0.01*	3.16	1.28-7.78
Female	18 (48.65%)	39 (75.0%)				
<b>WC</b>						
High ( $\geq 102$ cm) Male	0(0%)	8(61.54%)		<0.001* <sup>a</sup>		
High $\geq 88$ cm Female	0(0%)	39(100%)				
<b>family history</b>						
Absent	23(62.16%)	19(36.54%)	5.63	0.018*	2.85	1.19-6.82
Present	14(37.84%)	33(63.46%)				
<b>Exercise</b>						
YES	26(70.27%)	27(51.92%)	2.99	0.08	2.19	0.89-5.33
NO	11(29.73%)	25(48.08%)				
<b>Psychosocial stress</b>						
YES	17(45.9%)	40(76.92%)	8.91	0.003*	0.25	0.10-0.63
NO	20(54.1%)	12(23.08%)				
<b>Eat more</b>						
YES	6(16.22%)	43(82.69%)	38.17	<0.001*	0.04	0.01-0.13
NO	31(83.78%)	9(17.31%)				

\*p value  $\leq 0.05$  was significant.

<sup>a</sup>: Fisher – exact test.

Table (2) The mean differences of some physio-biochemical parameters of for both gender in obese and control: -

Variable \ Group	Control (Mean ± S.E)		Obese (Mean ± S.E)		P value of gender	P value of group
	Male (n=19)	Female (n=18)	Male (n=13)	Female (n=39)		
Age( years)	23.27±1.09	23.83±1.22	27.80±1.78	20.77±1.03	0.13	0.05*
BMI(Kg/M <sup>2</sup> )	23.03±0.48	22.30±1.77	28.76±0.79	27.23±0.72	0.39	0.05*
WC (cm)	82.00±1.47	77.77±1.77	107.23±3.74	108.72±2.25	0.07	0.05*
Insulin(ng/mL)	2.07±22.84	2.71±21.43	3.03±24.57	1.82±27.99	0.11	0.19
FBG (mmol/L)	4.41±0.07	4.18±0.10	4.78±0.17	4.79±0.12	0.03*	0.05*
HOMA-IR	4.77±0.47	4.10±0.49	5.91±0.38	0.94±0.39	0.16	0.06
TG (mmol/L)	0.79±0.00	0.75±0.06	1.26±0.07	1.38±0.04	0.11	0.18

\*P value is significant ≤ 0.05 level. .

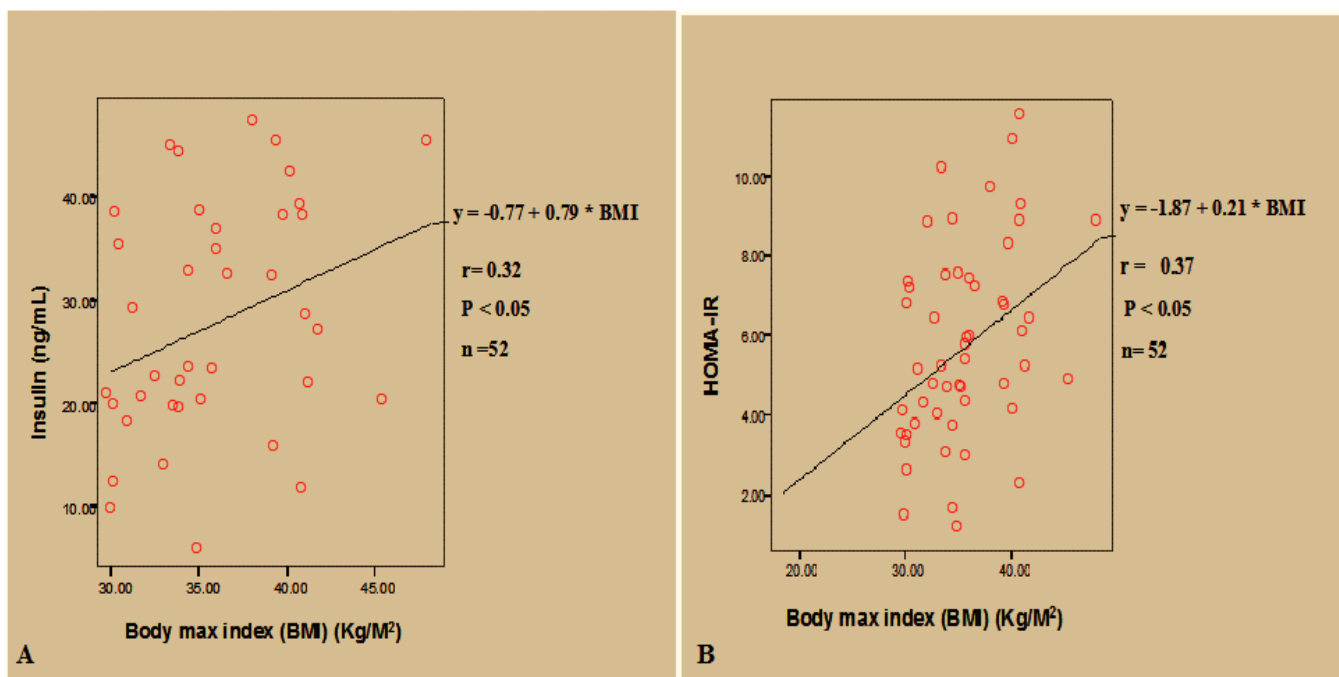
S.E: Standard error

**Table (3) The Mean differences of Some Physio-biochemical parameters at the three class of obesity in obese group.**

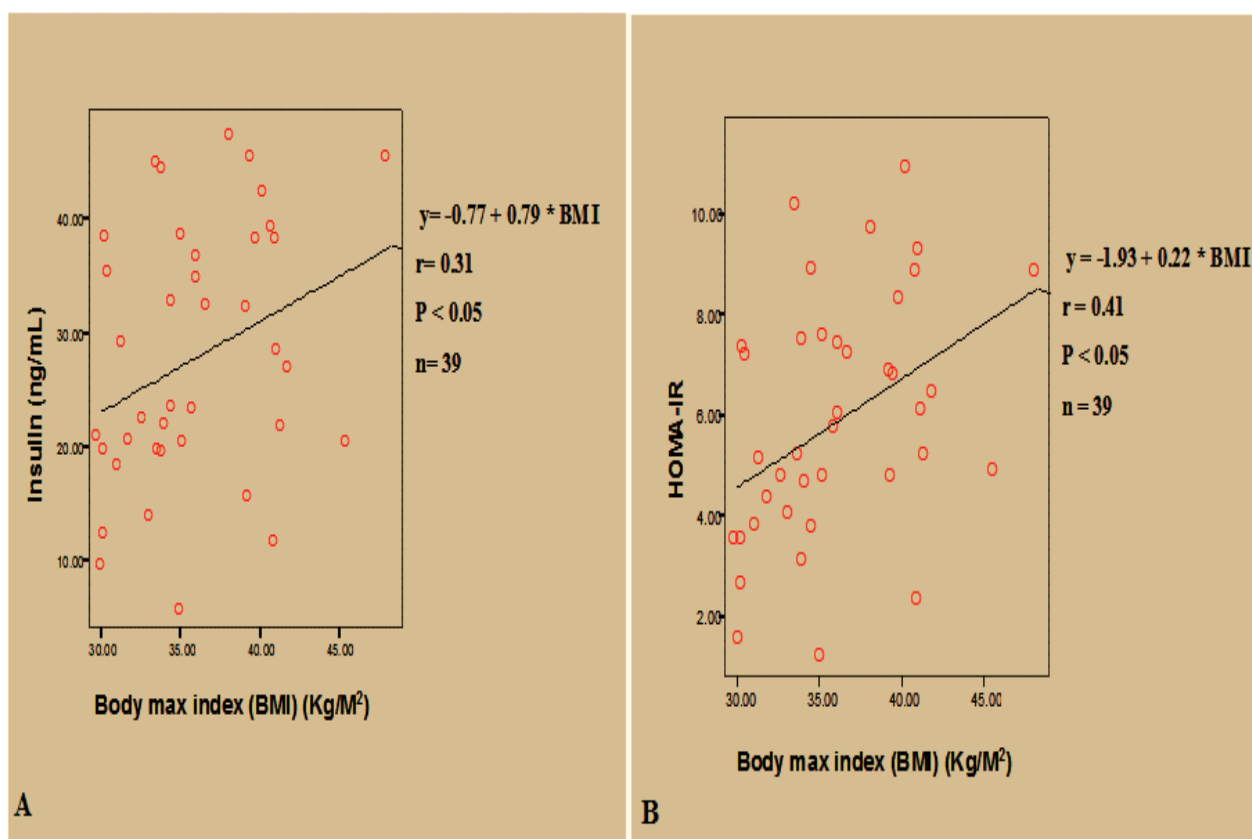
Group parameters	Obese group at three class (Mean ± S.E.)			P value of group
	Class I )n=24(	Class II )n=17(	Class III )n=11(	
Insulin(ng/mL)	2.15±24.03	2.64±29.18	3.76±30.79	0.17
FBG (mm\l)	0.14±.72±	0.19±4.67	0.14±.22±	0.000*
HOMA-IR	0.47±.11±	0.49±.11±	0.51±.11±	0.000*
TG (mm\l)	0.04±.10±	0.07±.12±	0.05±.10±	0.000*

\*P value is significant ≤ 0 .05 level.

S.E: Standard error.



**Figure (1): The relationship between Body max Indexes (BMI) (Kg/M2) with **A:** insulin hormone (ng/mL) and **B:** homeostasis model assessment of insulin resistance (HOMA-IR) of obese group.**



**Figure (5):** The relationship between Body mass Indexes (BMI) (Kg/M<sup>2</sup>) with **A:** insulin hormone (ng/mL) and **B:** homeostasis model assessment of insulin resistance (HOMA-IR) of female obese group.

## Discussion:

This study showed there was significant association between obese and control regarding the age, gender, presence of history of previous obesity, suffering from Psychosocial stress and eating more, while there was no significant association in exercise daily activity, shown in table (1). In our study, we dealt with young population the highest percentage (86.49%) for age (19-29) years and we began investigation for the risk of obesity factors, where at the global level, obesity prevalence is increasing continuously, with differences from one country to another, from one region of the country to another, according to sex, age, ethnicity and social-economic status with consideration the fact that the obesity risk for the population with a medical family history of obesity is 2.2 higher than in the population without a family history, we can presume that heredity has an important role in the emergence and maintenance of obesity (16). Data from large population studies show that mean body weight BMI gradually increase during most of adult life and reach peak values at 50–59 years of age in both men and women, where after 20–30 years of age, fat-free mass (FFM) progressively decreases and fat mass increases (16). While Joshua et al (2015) observed higher total physical activity in healthy than in unhealthy obese adults is evident only when measured objectively, which suggests that physical activity has a greater role in promoting

health among obese populations than previously (17 ) thus results of this study agree with many previous studies suggested that obesity is associated with impaired physical, psychosocial, overeating, increasing abdominal obesity (WC), genetic predisposition to obesity(16 -20).

The result of this study shows there was significant mean differences of age, body mass index, waist circumference and FBG between control with obese and in FBG for both gender, but there were no significant mean differences of the age, body mass index, waist circumference, insulin, HOMA-IR and TG between obese and control groups for both gender, as shown in table (2). Also result shows that there was significant mean differences in obese group in levels of FBG, HOMA-IR and TG at the three class of obesity in obese group, while there was no significant mean differences in levels of Insulin, despite the high level at class III of obesity show table (3). While we found positive correlation between BMI with insulin fasting and with HOMA-IR for the obese group and female group, show in Figure (1), (2). Studies have shown that the basal level of plasma insulin and the elevated level of insulin in response to increased blood glucose are related to the body fat mass (7), and other studies have shown that overfeeding on a high-fat diet for only 5 days induced hepatic insulin resistance and hyperinsulinemia while glucose disposal was unaffected (21). Where although insulin level increases during forced weight gain, it returns to a normal level on the first day of recovery (8). While several of evidence suggest that Insulin resistance develops over the years, but can be seen in young and old people, where 23% of people with a BMI <25 kg/m<sup>2</sup> appeared to be insulin resistant (22). The frequency of insulin resistance is 48.7% in overweight and 66.3% in obese patients (22). In general, the longer the duration of the obesity and the higher the BMI, the more insulin resistant a patient will be, although genetic tendency cannot be ruled out, seeing the fact that obesity and type 2 diabetes are strongly hereditary conditions (24). furthermore a sedentary lifestyle or psychosocial stress is strongly related to insulin resistance and promotes developing insulin resistance and low free fat mass (25). Weight gain of 10% has been shown to increase liver fat, stored as triglycerides, by 2.5-fold within 4 weeks (26). Where several lines of evidence suggest that the association of obesity with IR is not only related to degree of obesity but also seems to be critically dependent on fat distribution, thus, individuals with greater degrees of central adiposity develop IR more frequently (27). Through a study of non-diabetic Korean adult's shows that insulin resistance was associated with high triglycerides (28).

In addition, a Japanese study shows that triglyceride level was uniquely higher in insulin-resistant subjects, suggesting that the increased level of triglyceride would play a role in association with insulin resistance in the non-obese Asian people (29). Where normally insulin stimulates the synthesis and release of lipoprotein lipase from the endothelial cells of blood vessels causing lipolysis of triglycerides in the blood and release of free fatty acids (FFA). Insulin enhances the transport of FFA to the fatty cells (adipocytes) to be stored as lipids and elevated triglyceride was significantly higher in insulin-resistant subjects in both genders in western countries (30). The HOMA-index correlates insulin resistance with fasting concentrations of insulin and glucose in the blood, where a higher value indicates insulin resistance (31). Where studies the regression models of obesity in U.S. for adolescents shows that included age and weight status, girls had higher HOMA-IR than boys and Mexican-American children had higher HOMA-IR levels than white children (32). The many studies found a significant association between insulin resistance and weight loss but are inconsistent with the results for other studies reporting that insulin resistance



was predictive of weight gain these incongruous results could be due to age and ethnic differences between study populations (33).

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تقييم العلاقة بين مستويات الانسولين عند الصيام والسمنة صحية في البالغين في محافظة بابل،  
العراق

أميرة كمال محمد وعلاء جواد حسن

كلية العلوم | قسم علوم الحياة | جامعة بابل

## الخلاصة:

الهدف من هذه الدراسة لتقييم العلاقة بين مستويات الانسولين عند الصيام والكبار والسمنة، والذين لا يعانون من أمراض مزمنة وتقييم مقاومة الأنسولين في نفس المجموعات. وقد أجريت هذه الدراسة في -قاسم الجامعة الخضراء / كلية الزراعة / مختبر التكنولوجيا الحيوية مع قسم التغذية في مستشفى تعليمي مرجان / في محافظة بابل / العراق، للفترة من يونيو ٢٠١٥ إلى يناير ٢٠١٦. وشملت هذه الدراسة ٨٩ (وكانت ١٠٠٪) المرضى الذين يعانون من السمنة ٥٢ (٥٨.٤٣٪) و ٣٧ (٤١.٥٧٪) وكانت الرقابة على الفئة العمرية من (١٩-٤٠) سنة.

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