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# The Relationship Between Body Mass Index And Biochemical Parameters In Type 2 Diabetes Mellitus In Nineveh City In Iraq

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**Abstract**

Obesity and type 2 diabetes (T2DM) are public health issues that have caused worry across the world due to their health repercussions and economic costs. Diabetes is becoming more common at the same time that obesity is becoming more common. This twin pandemic has been dubbed "diabesity" by some specialists. T2DM was linked to an increased body mass index (BMI) and waist circumference (WC). Obesity has been linked to a higher risk of acquiring T2DM. Prenatal, early childhood, and teenage phases are all thought to be important in the development of obesity. Free fatty acids (FFA) are known to produce peripheral (muscle) insulin resistance, and most obese people have high plasma levels of them.

**Key word:** diabetes, obesity, body mass index, insulin, cardiovascular.

## General characteristics of the work

**Research object:** the effect of obesity in type 2 diabetes mellitus.

**Research subject:** to indicate the relationship between obesity and type 2 diabetes mellitus and how the obesity effected to the biochemical parameters in type 2 diabetes mellitus

## Aim of the study

1. to check the relationship between obesity and type 2 diabetes mellitus in Nineveh city In Iraq.

2.to indicate which type of biochemical parameters is more effected by body mass index in type 2 diabetes mellitus.

3.to check which type of obesity is more affected to the type 2 diabetes mellitus because we divided the obesity to the three group according to the body mass index (normal weight, overweight, obesity).

**Main part**

**Introduction**

Obesity and diabetes are two pandemics that have emerged in the century of twenty-first. Both are serious public health issues that affect people all over the world and are linked to severe, possibly life-threatening co-morbidities as well as massive financial consequences. Overweight (BMI between 25 and 30 kg/m2) [1] and obesity (BMI of 30 kg/m2 or above) [1] are becoming more common across the world, especially in developing nations. Obesity and type 2 diabetes have been linked for a long time. In both men and women, a meta-analysis of research on the relationship between these two variables revealed a greater relative risk with BMI and waist circumference [2].Although not all the people with type 2 diabetes (T2DM) are overweight or obese, and many obese people do not have diabetes, the majority of people with T2DM are overweight or obese. Diabetes affects a large percentage of fat people. Obesity, overweight, and T2DM may all be avoided by changing one's lifestyle and avoiding sedentary behaviors and excessive energy intake. Insulin resistance and atherogenic lipid profiles, such as elevated triglycerides and reduced HDL-C, are present in both obesity and T2DM. Human obesity has a multigenic rather than a monogenic genetic base that predisposes to insulin resistance and T2DM.Current clinical recommendations recognize the therapeutic value of exercise intervention in diabetes prevention and therapy.

**1 Criteria and Definitions for Diagnosis**

WHO defines "overweight" [1] as having a BMI of 25 or more, and "obesity" [1] as having a BMI of 30 or more. BMI is a measure of weight standards that is determined by dividing weight (kg) by height (m2) and then adjusting for height. The American Diabetes Association7 recommends the following criteria for diagnosing diabetes mellitus: 1. An A1C of 6.5 percent or a fasting plasma glucose [FPG] value of 126 mg/dL following an 8-hour fast, or a 2-hour post load glucose (PG) of 200 mg/dL (11.1 mmol/L) after an OGTT, or symptoms of diabetes mellitus and a random plasma glucose concentration of 200 mg/dl (11.1 mmol/L) Insulin resistance [3] is defined as the inability of target organs to respond to insulin in a normal manner. Insulin resistance syndrome (IRS) is a collection of disorders that are more frequent in people who are insulin resistant. The National Cholesterol Education Program Adult Treatment Panel III (NCEP ATP III) [5] defines metabolic syndrome as "a metabolic cluster disorders with insulin resistance as a primary hallmark." The presence of any three of the five components is enough to diagnose the condition. The metabolic syndrome consists of five components: 1) abdominal obesity (waist circumference > 102 cm [16] in men, >

88 cm [15] in women); 2) hypertriglyceridemia (150 mg/dL); 3) low HDL-C (40 mg/dL in men, 50 mg/dL in women); 4) high blood pressure (130/85 mm Hg); and 5) high fasting glucose.

**2 Impact of Obesity on Type 2 Diabetes**

In this study from the Asia-Pacific area, the relative risk of T2DM increases when BMI rises above 23 [6], and the link was found to be greater in younger age groups [7]. Early adulthood weight growth is linked to a greater risk and earlier development of type 2 diabetes than weight gain between the ages of 40 and 55 [8]. Diabetes risk increases linearly with BMI; diabetes prevalence rose from 2% in those with a BMI of 25 to 29.9 kg/m2, to 8% in those with a BMI of 30 to 34.9 kg/m2, and ultimately to 13% in those with a BMI more than 35 kg/m2 [9]. Despite the fact that the incidence of diagnosed diabetes has risen dramatically in the previous decade, the prevalence of undiagnosed diabetes and impaired fasting glucose (IFG) has remained relatively consistent [10]. Increases in waist circumference (WC), waist-to-hip ratio, visceral adiposity, or abdominal obesity enhance the risk of central obesity more than generalized obesity [14-17]. BMI or WC independently predicted or was linked with T2DM in a review of 17 prospective and 35 cross-sectional studies in persons aged 18 to 74 years [11]. BMI increases are a greater predictor of diabetes than weight gain. Prospective studies in non-diabetic overweight adults found that every 1 kg/year increase in body weight was associated with a 49 percent increase in diabetes incidence over 10 years, and similarly, each kg of weight lost annually over 10 years was associated with a 33 percent lower risk of diabetes over the following 10 years. Weight growth was only strongly connected to diabetes incidence in Pima Indians who were not already overweight (BMI less than 27.3 kg/m2), according to similar research [19,20]. Similarly, Mokdad et al. [12] found that every 1 kg increase in average self-reported weight was related with a 9% increase in the prevalence of diabetes [14] in the Behavioral Risk Factor Surveillance System (BRFSS) during 1991-1998. Visceral fat, rather than upper body subcutaneous fat, appears to be substantially linked to an aberrant metabolic profile. WC is used by the National Institutes of Health to identify those who are at risk [13]. Despite the fact that both visceral adiposity (VAT) and subcutaneous fat are linked to unfavorable cardio metabolic risk factors, VAT is more strongly linked [14].

**3 Insulin Resistance and Obesity in T2DM**

Normal glucose homeostasis is maintained by a precise balance between insulin secretion by pancreatic -cells and insulin sensitivity of peripheral tissues (muscle, liver and adipose tissue). Insulin resistance is a defining feature of the metabolic syndrome, which often results in T2DM. Reduced insulin sensitivity and reduced -cell function are the two key components of T2DM pathogenesis, according to long-term adult experience. Obesity and T2DM are linked primarily by insulin resistance. In the normal course of diabetes, obesity and insulin resistance arrive before high glucose levels. Reduced insulin-stimulated glucose

transport and metabolism in adipocytes and skeletal muscle, as well as inadequate hepatic glucose output reduction, are all signs of insulin resistance in both of these illnesses [17].

**4 Diabetes and Obesity's Effects on Cardiovascular Disease**

Cardiovascular disease (CVD) is a leading cause of morbidity and mortality in people with type 2 diabetes, accounting for up to 75% of all fatalities. When compared to age-matched people, the risk of CVD mortality in type 2 diabetes patients is more than doubled [18]. Persons with type 2 diabetes have the same risk of coronary artery disease (CAD) as nondiabetic subjects who have CAD [200,201], especially women [19]. Obesity and clinically significant coronary artery disease are clearly linked in two well cited prospective studies: the Framingham Heart Study [20] and the Nurses' Health Study [21Obese patients' elevated pro-inflammatory cytokine levels are mostly due to obesity rather than T2DM. Furthermore, surgery-induced weight loss lowers circulation levels of important pro-inflammatory molecules, which contributes to a reduction in cardiovascular co-morbidity after excessive weight loss [22-36]. The World Health Organization (WHO) frequently defines obesity and overweight in terms of excess weight for a certain height. Overweight was defined as a BMI of 25.0 to 29.9, while obesity was classified as a BMI of >30.0. BMI is a measure of weight standards that is determined by dividing weight (kg) by height (m2) and then adjusting for height.

**Material and method**

**1. Materials** (material and instruments used in the study). Negative control, Positive control, cotton, Cobas 111, Water shower, Centrifuge (differential)), Micro Pipettors (limit 5–40 µl, 40–200 µl, 200–1000 µl), Micro Pipettors (limit 5–40 µl, 40–200 µl, 200–1000 µl), Gauze wipes – for application on the site from which the needle is pulled back, Adhesive gauzes/tape – e ensures the vein cut site after accumulation, Gel tube, Alcohol Wipes – 70% isopropyl liquor, Gloves, syringe, Venus blood sample taken with a Tourniquet.

**2.1. Population Study of the Patient**

This study was conducted with the approval of the Department of Biochemistry at Grodno State University, Faculty of Biology and Ecology (Belarus), the diagnosis was provided by the senone general hospital and the Blood Bank in Nineveh city in Iraq for patients who suffer from type II diabetes and obesity.

**2.2. Period of sample collection**

the cases control study study was conducted in the Governorate of Nineveh: From 12 August 2021 to 8 September of the same year, the number of patients with type 2 diabetes was 78 patients is divided to three group of BM1(normal weight group, overweight group and obesity group) for each group is 26

samples of both sexes and different ages (45-65), male 39 and female also 39 samples, so also the same for control group using cobas(version111).

**Statistical analysis**

The data was translated into a computerized database interface and the patient information was taken for each of the sex, age, height and weight. All samples were collected and a database was created for each patient according to the information that was taken from the patient and the results of the patient samples. Statistical analyzes were performed using the statistical program SPSS version 20 in conjunction with a program Microsoft Excel version 2010 where a relationship was made with each disease for all patients, such variables are described by mean, SD (standard deviation) Receiver operating characteristic analysis and diagnostic test were performed to calculate the relationship between type 2 diabetes mellitus and obesity , estimate was considered statistically significant if its P value is 0.05 or near to this value.

**Result and discussion**

Through laboratory results that are then obtained and by making a relationship between BMI and biochemical parameters in type 2 diabetes mellitus we see there is a relationship between normal BMI and C. peptide and the C. peptide may be abnormal when the body mass index is normal also as shown as in the table below

| **Normal weight** | | | | | | |
|---|---|---|---|---|---|---|
| **Parameters** | | **BMI** | **HBa1c** | **RBS** | **Insulin resistant** | **C. peptide** |
| **BMI** | **Pearson Correlation** | 1 | .054 | .129 | -.122 | -.431* |
| | **Sig. (2-tailed)** | | .794 | .530 | .551 | .028 |
| | **N** | 26 | 26 | 26 | 26 | 26 |

In the overweight patients we can indicate there is a significant relationship between overweight patient and insulin RBS

| **Over weight** |
|---|

| Parameters | | BMI | HBa1c | RBS | Insulin resistant | C. peptide |
|---|---|---|---|---|---|---|
| BMI | Pearson Correlation | 1 | .155 | .394* | .161 | -.023 |
| | Sig. (2-tailed) | | .448 | .047 | .432 | .911 |
| | N | 26 | 26 | 26 | 26 | 26 |

In the obesity group there is a significant relationship between obesity with both of random blood sugar and HBA1C as shown as in the table below

| Obesity | | | | | | |
|---|---|---|---|---|---|---|
| parameters | | BMI | HBa1c | RBS | Insulin resistant | c. peptide |
| BMI | Pearson Correlation | 1 | .560** | .501** | -.368 | .154 |
| | Sig. (2-tailed) | | .003 | .009 | .064 | .452 |
| | N | 26 | 26 | 26 | 26 | 26 |

**Correlation is significant at the 0.01 level(2-tailed)**

**Correlation is significant at the 0.05 level(2-tailed)**

According to the descriptive statistics we can check the minimum value, maximum value, mean and standard division as shown as in the table of normal weight group, overweight group and obesity group for both of patients and control

| Normal weight | | | | | |
|---|---|---|---|---|---|
| Patient group | | | | | |
| Descriptive Statistics | | | | | |
| parameters | N | Minimum | Maximum | Mean | Std. Deviation |

| | | | | | |
|---|---|---|---|---|---|
| **BMI** | 26 | 19 | 25 | 21.85 | 1.85 |
| **HBa1c** | 26 | 5.9 | 10 | 8.02 | 0.99 |
| **RBS** | 26 | 110 | 480 | 222.88 | 92.57 |
| **Insulin resistant** | 26 | 3.2 | 7.5 | 6.22 | 1.20 |
| **c. peptide** | 26 | 0.3 | 2.7 | 1.53 | 0.73 |

| **Normal weight** | | | | | |
|---|---|---|---|---|---|
| **Control group** | | | | | |
| **Descriptive Statistics** | | | | | |
| **Parameters** | **N** | **Minimum** | **Maximum** | **Mean** | **Std. Deviation** |
| **BMI** | 26 | 18.5 | 24 | 21.69 | 1.59 |
| **HBa1c** | 26 | 4.1 | 6.4 | 5.05 | 0.72 |
| **RBS** | 26 | 18 | 190 | 129.42 | 35.08 |
| **Insulin resistant** | 26 | 4.1 | 7.3 | 6.22 | 0.91 |
| **c. peptide** | 26 | 0.3 | 2.4 | 1.49 | 0.60 |

| **Over weight** | | | | | |
|---|---|---|---|---|---|
| **Patient group** | | | | | |
| **Descriptive Statistics** | | | | | |
| **parameters** | **N** | **Minimum** | **Maximum** | **Mean** | **Std. Deviation** |
| **BMI** | 26 | 26 | 30 | 27.65 | 1.16 |
| **HBa1c** | 26 | 6.8 | 10.2 | 8.38 | 0.80 |
| **RBS** | 26 | 90 | 470 | 247.31 | 95.61 |
| **insulin** | 26 | 3.5 | 7.8 | 5.86 | 1.18 |

| | | | | | |
|---|---|---|---|---|---|
| **resistant** | | | | | |
| **c. peptide** | 26 | 0.4 | 3.1 | 1.79 | 0.60 |

| **Over weight** | | | | | |
|---|---|---|---|---|---|
| **Control group** | | | | | |
| **Descriptive Statistics** | | | | | |
| **parameters** | **N** | **Minimum** | **Maximum** | **Mean** | **Std. Deviation** |
| **BMI** | 26 | 26 | 30 | 27.65 | 1.26 |
| **HBa1c** | 26 | 5.3 | 6.7 | 6.05 | 0.29 |
| **RBS** | 26 | 85 | 180 | 138.35 | 28.25 |
| **insulin resistant** | 26 | 4.6 | 7.2 | 6.08 | 0.72 |
| **c. peptide** | 26 | 0.4 | 2.5 | 1.51 | 0.64 |

| **Obesity** | | | | | |
|---|---|---|---|---|---|
| **Patient group** | | | | | |
| **Descriptive Statistics** | | | | | |
| **Parameters** | **N** | **Minimum** | **Maximum** | **Mean** | **Std. Deviation** |
| **BMI** | 26 | 33 | 41 | 36.50 | 2.27 |
| **HBa1c** | 26 | 7.1 | 11.3 | 8.82 | 1.09 |
| **RBS** | 26 | 150 | 500 | 339.23 | 106.96 |
| **Insulin resistant** | 26 | 3.5 | 7.8 | 6.00 | 1.18 |
| **c. peptide** | 26 | 0.3 | 3.4 | 1.74 | 0.79 |

| Obesity | | | | | |
|---|---|---|---|---|---|
| Control group | | | | | |
| Descriptive Statistics | | | | | |
| Parameters | N | Minimum | Maximum | Mean | Std. Deviation |
| BMI | 26 | 31 | 42 | 35.69 | 2.71 |
| HBa1c | 26 | 4.8 | 6.5 | 5.97 | 0.40 |
| RBS | 26 | 85 | 167 | 124.42 | 26.08 |
| insulin resistant | 26 | 4.1 | 7.4 | 5.66 | 1.03 |
| c. peptide | 26 | 0.3 | 2.8 | 1.62 | 0.55 |

| T test and p value for normal group | | | |
|---|---|---|---|
| no | Parameters | T test | P value |
| 1 | BMI | 0.32 | 0.749 |
| 2 | HBa1c | 12.36 | 0.000 |
| 3 | RBS | 4.81 | 0.000 |
| 4 | insulin resistant | 0.03 | 0.979 |
| 5 | c. peptide | 0.19 | 0.853 |

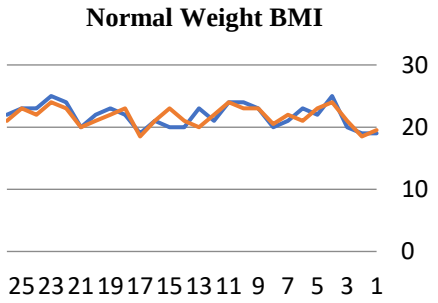
| T test and p value for overweight group | | | |
|---|---|---|---|
| | | T test | P value |
| 1 | BMI | 0.01 | 0.999 |
| 2 | HBa1c | 13.93 | 0.000 |
| 3 | RBS | 5.57 | 0.000 |
| 4 | insulin resistant | 0.84 | 0.409 |
| 5 | c. peptide | 1.63 | 0.110 |

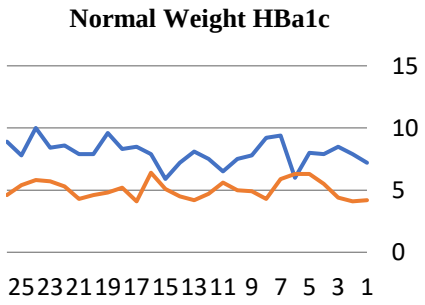
| | T test and P- value for Obesity Weight Group | | |
|---|---|---|---|
| | **parameters** | **T test** | **P value** |
| 1 | **BMI** | 1.17 | 0.249 |
| 2 | **HBa1c** | 12.57 | 0.000 |
| 3 | **RBS** | 9.95 | 0.000 |
| 4 | **insulin resistant** | 1.12 | 0.267 |
| 5 | **c. peptide** | 0.63 | 0.530 |

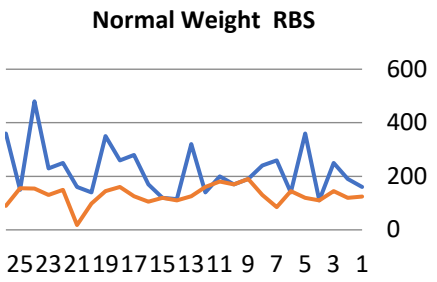
**T test and P- value for Obesity Weight Group**

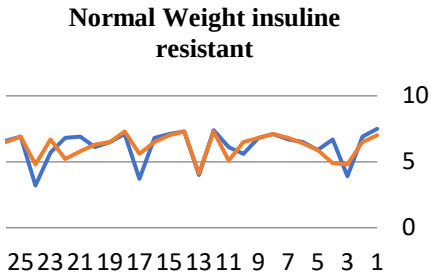
**Note: p- value less than 0.05 means significant**

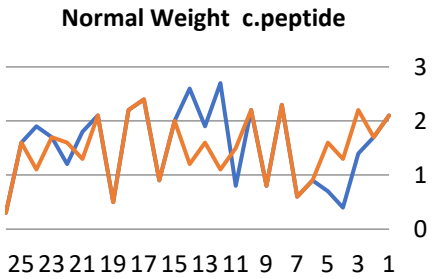
Throug the table above and by the T test and p value we can check there is a significant relationship between blood sugar and body mass index

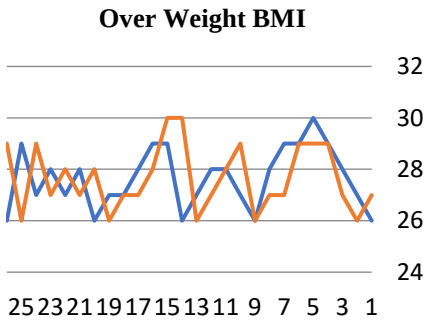


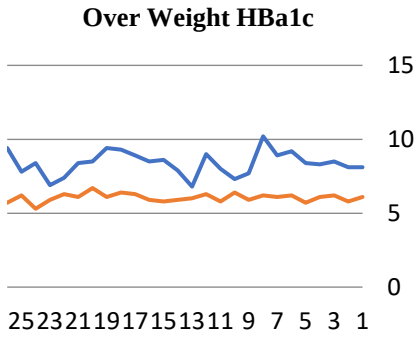


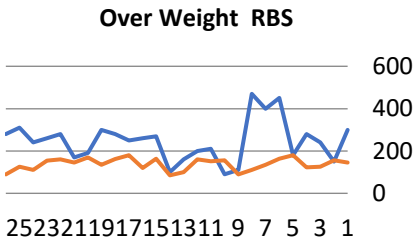



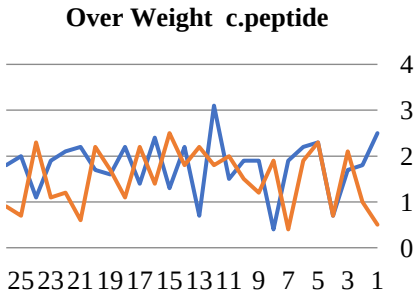



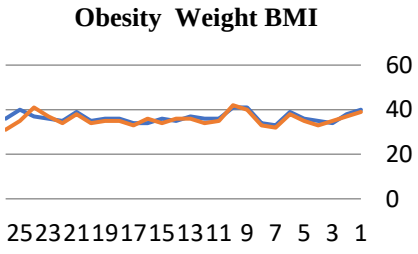




**Note : Blue for Patients Group and Red for Normal Group**

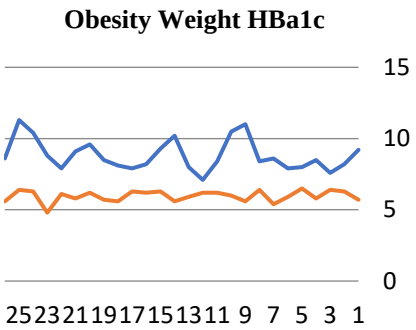


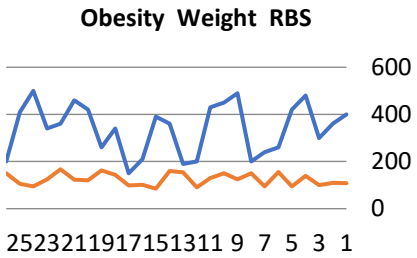



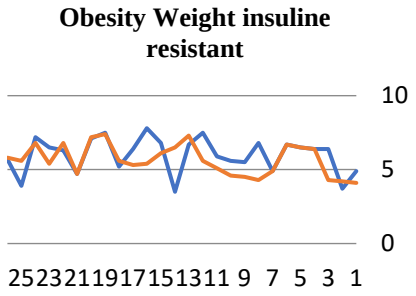



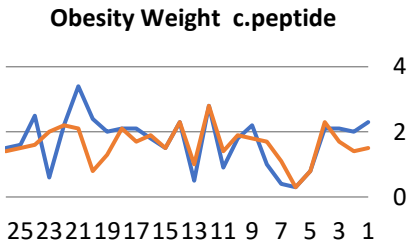




**Note : Blue for Patients Group and Red for Normal Group**




Through to the graphs we see a large divergence between the line of blood sugar and BMI

Also, there is significance relationship between HBA1c and type 2 diabetes mellitus.

**Conclusion**

1. there is significant correlation between body mass index (obesity groups) and less than correlation with overweight group with blood sugar in type 2 diabetes mellitus.

2. there is a strong relationship between body mass index (obesity group) with HBA1C and less than with overweight group.

3. C. peptide is affected by obesity group.

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