

Original Research Article

Investigation of Vitamin D Levels and Inflammatory Markers in Obese Adolescent: A Study from Ramadi City

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Abstract: *Objective:* The aim of the study was to assess the relationship between obesity measured by anthropometric measurements, vitamin D levels, and inflammatory status in Al-Ramadi obese adolescents. *Methods:* Between April and September of 2024, this study was carried out in the Maternity and Children Teaching Hospital in Ramadi, Iraq. A total of 60 teenagers were chosen, 30 of whom were obese (14 females and 16 males), and 30 of whom were normal weight as a control group. The InBody Device was used to evaluate anthropometric parameters such as body mass index (BMI), body fat percentage (BFP), skeletal muscle mass (SMM), waist-hip fat ratio (WHR), visceral fat, body fat, and basal metabolism. Insulin resistance (IR) and vitamin D levels were assessed using the linked enzyme immunoassay (ELISA) method. The Cobas e411 equipment was used to measure the levels of inflammatory cytokines, total cholesterol (TC), triglycerides (TG), low-density lipoprotein (LDL), and high-density lipoprotein (HDL). *Results:* Results showed significantly ($P \leq 0.05$) increases in anthropometric measurements, insulin resistance and pro-inflammatory cytokines (IL-6, TNF- α and IL-1 β), whereas vitamin D levels showed significantly ($P \leq 0.05$) decreases in obese adolescents male and female than adolescents with normal weight male and female. *Conclusion:* The study concluded that the decreased in vitamin D levels appeared to be associated with obesity, as well as the high levels of insulin resistance (IR), inflammatory mediators and lipid profile that associated with obesity.

Keywords: Obesity; Anthropometric measurements; Vitamin D; IR; inflammation cytokines.

1. INTRODUCTION

Vitamin D is a fat-soluble vitamin with a unique among vitamins because of can be produced in the human body in sufficient quantities on condition that that there is sufficient exposure to sunlight and proper related organ as: liver, skin, and liver, function, in addition to, it functions more the same as a hormone than a vitamin since its activity is based on interacting with a receptor [1].

Sufficient exposure to sunlight helps in the synthesis of vitamin D and thus there are sufficient amounts of vitamin D in the body, but in some geographical areas there is not sufficient exposure to sunlight due to the climate of those areas and also low exposure to sunlight is necessary to prevent skin cancer, and thus there is a need for vitamin D from food sources [2].

Humans primarily obtain vitamin D from their diet, which includes foods like liver oil, oily fish, and sunlight, as well as from supplements and fortification in foods like milk, orange juice, and cooking oil. Cholecalciferol, or vitamin D₃, is also produced exogenously from animal products and endogenously in skin exposed to UVB rays. The metabolism of vitamin D (Figure 1) is a multi-stage, intricate process [3]:

Vitamin D is mainly synthesized in the skin from 7-dehydrocholesterol when exposed to Ultraviolet B (UVB) sunlight. In addition to the dietary sources, and fortified foods. the initial form is in an inactive, known as vitamin D₃ (from the animal sources: cholecalciferol) or vitamin D₂ (from the plant sources: ergocalciferol) [4].

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Once vitamin D (D2 and D3) comes in the bloodstream it is either distributed and stored in the adipose and muscle tissue or metabolized in the liver by the 25-hydroxylase into 25 hydroxyvitamin D (25(OH)D or calcidiol) [5].

25(OH)D or calcidiol is the major circulating shape of vitamin D and is typically estimated in blood tests to evaluate vitamin D case.

In the final stage, in kidneys, 25(OH)D is further hydroxylated by the 1α -hydroxylase enzyme into 1,25 dihydroxyvitamin D ($1,25(\text{OH})_2\text{D}_3$ or calcitriol) [6].

Calcitriol is the biologically active form of vitamin D, plays a main role in calcium and phosphorus homeostasis. In the kidney, the conversion to calcitriol is regulated by parathyroid hormone (PTH), calcium, and phosphorus levels in the blood serum [7].

Obese adolescents are more likely to be vitamin D deficient than people of the same age but of normal weight. This may be due to numerous causes as decreased exposure to sunlight, distribution of the vitamin in adipose tissue, and changes in lifestyle and nutrition. This deficiency is related to several health issues, including poor bone health, increased risk of heart and metabolic diseases, and immune disorders [8].

It is worth noting, obesity is not just excessive fat accumulation, it is a chronic, low-grade inflammatory condition that affects many bodily functions. When adipose tissue, especially visceral adipose tissue, increases, it not only acts as an energy store, but also becomes metabolically active and releases inflammatory mediators cytokines [9].

These mediators promote a chronic, low-grade inflammatory response, leading to metabolic imbalance, as inflammatory cytokines interfere with insulin signaling, causing insulin resistance and increasing the risk of type 2 diabetes [10].

This study aims to evaluate the effect of obesity on vitamin D levels and investigate obesity-induced inflammation by measuring pro-inflammatory cytokines (IL-6, TNF- α and IL-1 β).

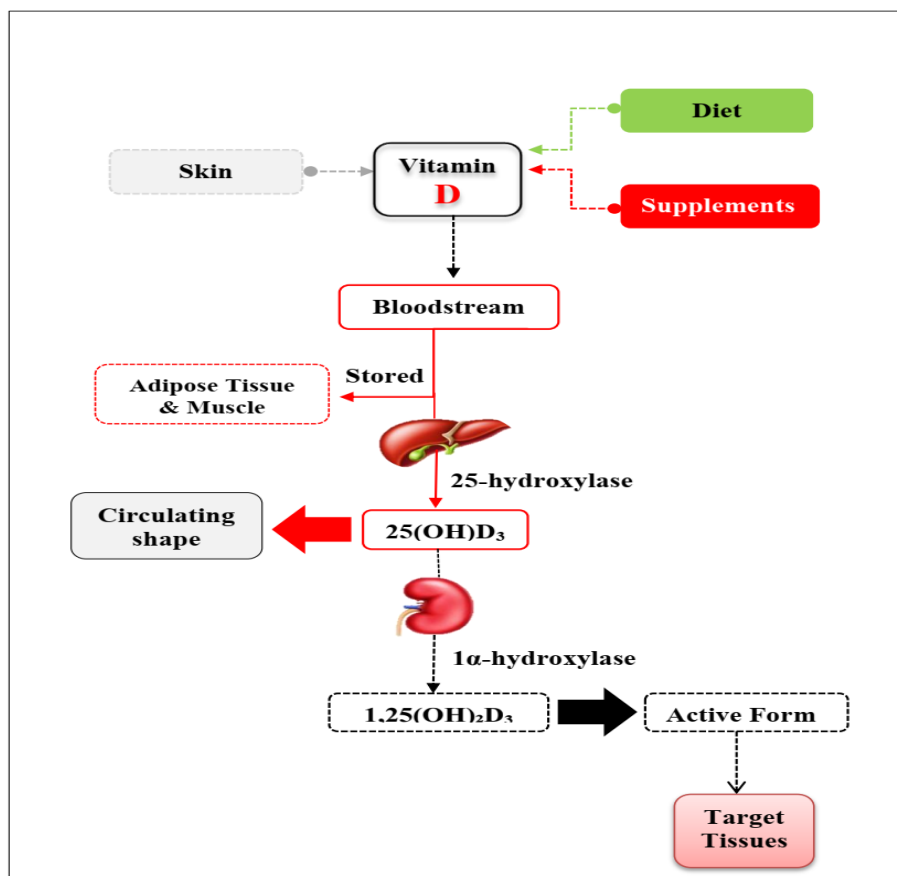


Figure 1: Schematic shows the major pathway of vitamin D metabolism (Designed by researcher)

2. MATERIAL AND METHODS

2.1. Study Participants

Sixty participants were recruited of the study, thirty obese adolescents (14 female/ 16 male) and thirty healthy adolescents (15 female/15 male), all participants with age 14 to 17 years old. Exclusion criteria which including obesity resulting from endocrine disorders, chronic diseases, and treatment with medicaments may affect vitamin D levels or body composition.

Participants were recruited from medical clinics, schools, and health centers, after obtaining consent from the parent and the adolescent himself.

2.2. Anthropometric Parameters

Participants' body composition was measured using the InBody device, a bioelectrical impedance analysis (BIA) body composition device. Measurements were done in a controlled environment according to typical rules, with participants asked to: Fasting for 2-3 hours before measurement, avoiding exercise during the previous 24 hours, removing any metal accessories (such as watches) during the examination.

The following data were collected for each participant:

- Body Mass Index (BMI) = weight (kg) / height squared (m²).
- Body Fat Percentage (BFP).
- Skeletal Muscle Mass (SMM).
- Waist-hip fat ratio (WHR)
- Visceral Fat Level.
- Body Fat
- Basal Metabolism

2.3. Biochemical Assessment

Participants' venous blood samples were obtained, and the serum was separated by centrifugation at 3000 rpm for 10 minutes. The samples were then kept at -20°C until the linked enzyme immunoassay (ELISA) method was used to evaluate vitamin D and insulin resistance (HOMA-IR). The Cobas e411 equipment was used to measure the lipid profile, which includes the following: total cholesterol (TC), triglycerides (TG), low-density lipoprotein (LDL), high-density lipoprotein (HDL), and inflammatory cytokines.

2.4. Statistical Analysis

All data were analyzed using SPSS (version 20), in which data were expressed as mean \pm standard error (Mean \pm SE). For comparison between the two groups (obese and healthy adolescents), the independent t-test was used for variables with a normal distribution. Statistically significant was considered $P \leq 0.05$.

3. RESULTS

Table 1 presented the summary of anthropometric and biochemical measures of participants, who was divided into group (14 female/16 male obese adolescents) and (15 female/ 15 male healthy adolescents).

The results anthropometric measurements in the table 1 showed significant differences between the obese adolescent group and the normal-weight adolescent group. The obese adolescent group recorded a significantly ($P \leq 0.05$) increased in BMI, BFP, SMM, WHR, and visceral fat, and body fat compared to normal weight adolescents, reflecting the clear difference in body composition between the two groups. AS well as, the obese adolescent group recorded a significant ($P \leq 0.05$) increased in Basal metabolic rate compared to normal weight adolescents.

The results in Table 1 showed the level of vitamin D and HOMA-IR in the group of obese and normal weight participants. There was a significant ($P \leq 0.05$) decrease in the level of vitamin D in obese adolescents compared to normal weight adolescents. On the other hand, there was a significant ($P \leq 0.05$) increase in the level of IR in obese adolescents compared to normal weight adolescents.

The lipid profile levels of the group of participants who were obese and those who were normal weight were displayed in Table 1. The results showed that the triacylglycerol (TAG), total cholesterol (TC), and low-density lipoprotein cholesterol (LDL-C) were significantly ($P \leq 0.05$) higher in the obese adolescents than in the normal weight adolescents, while the LDL-C was significantly ($P \leq 0.05$) lower in the obese adolescents than in the normal weight adolescents.

Regarding obesity-related inflammation which measured by pro-inflammatory cytokines, the results are shown in Table 1, there was a significant ($P \leq 0.05$) increase in the level of IL-6, TNF- α and IL-1 β in obese adolescents compared to normal weight adolescents.

Table 1: Summary of parameters in studied participants groups of obese with and non-obese adolescents

Parameter	Unit	Mean± SE Obese (n=30)	Mean± SE Non-obese (n=30)	p-value
Age	Years	15.34±0.12	16.08±0.52	N.S
Gender	M/F	16M/14F	15M/15F	N.S
BMI	Kg/m ²	35.13±0.48	24.09±1.07	0.0000*
BFP	%	43.06±1.02	22.19±0.16	0.0001*
SMM	kg	28.95±0.20	26.08±0.68	0.0007*
WHR	-	1.05±0.96	0.82. ±0.71	0.0008*
Visceral Fat	kg	10.02±0.26	5.32±1.02	0.0001*
Body Fat	kg	25.85±0.57	16.98±1.04	0.0002*
Basal Metabolism	Kcal	1711.26±0.06	1392.03±0.92	0.0003*
HOMA-IR	-	2.94±1.15	1.06±0.80	0.0004*
Vitamin D	ng/mL	17.45±0.91	28.09±1.09	0.0000*
LDL	mg/dL	102.15±0.25	77.12±0.34	0.0000*
HDL	mg/dL	36.28±1.76	58.12±0.76	0.0002*
TC	mg/dL	189.64±0.29	170.91±0.17	0.0001*
TG	mg/dL	132.81±0.86	192.98±0.45	0.0005*
IL-1 β	pg/mL	4.01±0.91	1.23±0.61	0.0007*
TNF- α	pg/mL	3.38±0.21	2.03±0.45	0.0002*
IL-6	pg/mL	4.81±0.05	1.28±0.37	0.0000*

* $P \leq 0.05$ *SE: Standard Error * N.S: Not Significant * M/F: Male/Female * BMI: Body Mass Index *BFP: Body Fat Percentage *SMM: Skeletal Muscle Mass *WHR: Waist-hip fat ratio * HOMA-IR: Insulin Resistance *LDL: Low-Density lipoprotein *HDL: High-density lipoprotein *TC: Total Cholesterol *TG: Triglyceride * IL-1 β : Interleukin -1 beta * TNF- α : Tumor necrosis alpha *IL-6: Interleukin-6.

The figure below shows a comparison of vitamin D and IR levels between obese adolescents (males and females) and normal weight adolescents (males and females). There was a significant ($P \leq 0.05$) decrease in the level of vitamin D in obese adolescents (male and female) compared to normal weight adolescents (male and female). On the other hand, there was no significant ($P \leq 0.05$) differences in the level of vitamin D in male and female obese adolescents, as well the results in figure showed there was no significant ($P \leq 0.05$) differences in the level of vitamin D in male and female with normal weight adolescents.

On the other hand, there was a significant ($P \leq 0.05$) increase in the level of IR in obese adolescents (male and female) compared to normal weight adolescents (male and female). While, there was no significant ($P \leq 0.05$) differences in the level of IR in male and female obese adolescents, as well the results in figure showed there was no significant ($P \leq 0.05$) differences in the level of IR in male and female with normal weight adolescents.

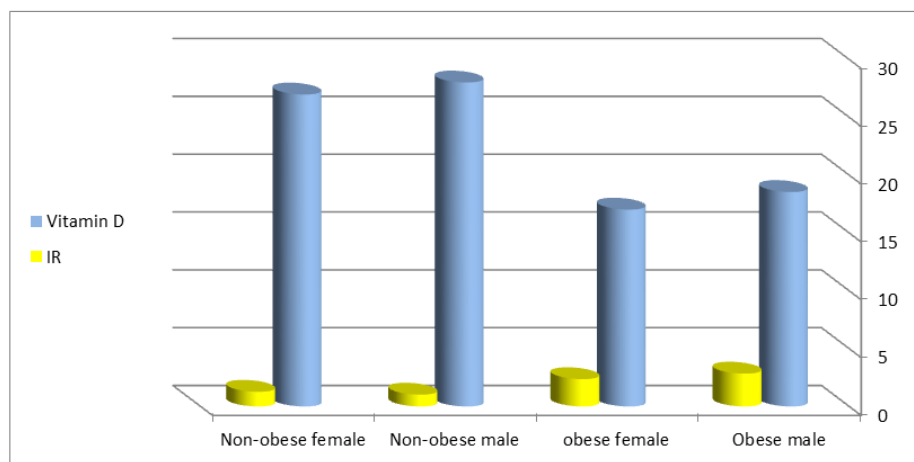


Figure 2: Show the levels of HOMA-IR and Vitamin D in obese adolescents (males and females) and normal weight adolescents (males and females)

The Figure below shows a comparison of inflammatory cytokine levels between obese adolescents (males and females) and normal weight adolescents (males and females). There was a significant ($P \leq 0.05$) increase in the level of IL-6, TNF- α and IL-1 β in obese adolescents (male and female) compared to normal weight adolescents (male and female). On the other hand, there was no significant ($P \leq 0.05$) differences in the level of IL-6, TNF- α and IL-1 β in male and female obese adolescents, as well the results in figure showed there was no significant ($P \leq 0.05$) differences in the level of IL-6, TNF- α and IL-1 β in male and female with normal weight adolescents.

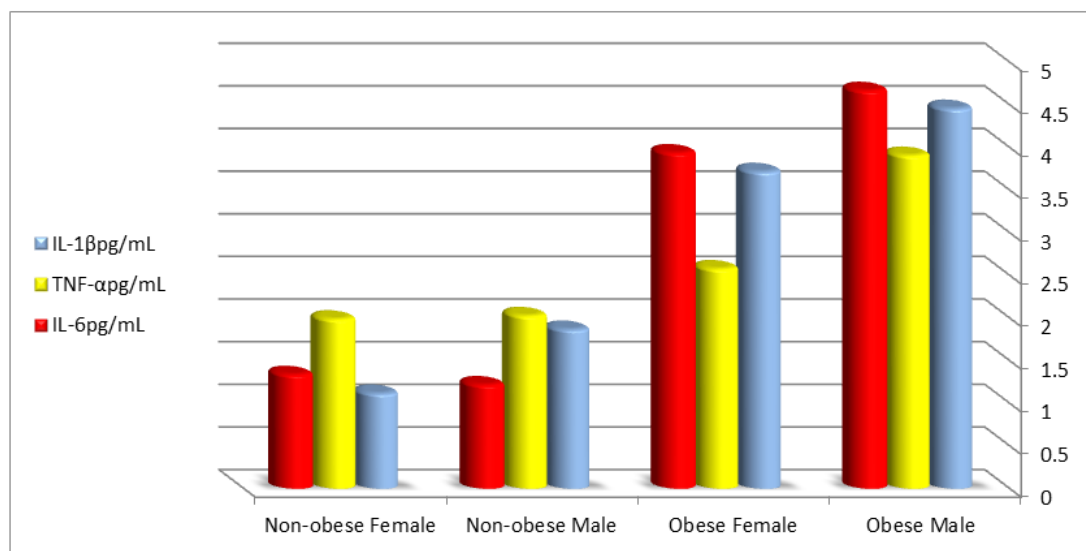


Figure 3: Show the levels of inflammatory cytokine in obese adolescents (males and females) and normal weight adolescents (males and females)

4. Correlations

Our study shows that IR correlates positively with BMI ($R=0.672$, $P \leq 0.05$). Also IR correlates positively with both TC ($R=0.361$, $P \leq 0.05$) but correlates negatively with HDL ($R= -0.395$, $P \leq 0.05$). Serum LDL correlates positively with IL-6 ($R=0.795$, $P \leq 0.01$).

5. DISCUSSION

Anthropometric measurements were compared between obese male and female adolescent participants and their normal-weight peers (males and females) to determine differences in body composition.

The results in the table showed that the obese group had significantly higher levels of BMI, BFP, SMM, WHR, and visceral fat, and body fat compared to the normal weight group. These differences suggest that physiological effects accompanying obesity may play a role in the metabolic and inflammatory changes associated with it [11].

The results (Table 1) obtained indicate that there is an increase in the body mass index (BMI), body fat percentage, and waist circumference in obese adolescents, and this is due to an energy imbalance, where the intake of calories exceeds the rate of their consumption, which leads to the accumulation of fat [12].

In obesity, hypertrophy and hyperplasia of fat cells occur, increasing adipose tissue mass, especially in visceral areas. This expansion of adipose tissue is associated with metabolic changes such as increased visceral fat storage, which affects body fat distribution and is associated with insulin resistance and chronic inflammation [13]. This is consistent with the results obtained (Table 1).

Furthermore, obesity affects body composition by increasing fat mass compared to muscle mass, resulting in a higher body fat percentage compared to individuals of normal weight. Waist circumference is also a key indicator of visceral fat accumulation, which is associated with increased health risks such as diabetes and heart disease [14].

The results also showed that there is an increase in the basal metabolic rate in obese adolescents compared to adolescents with normal weight, due to the increase in total body mass, which requires more energy to maintain organ functions [15]. Also, the high muscle mass in obese adolescents (Table 1), the increased activity of vital organs, and hormonal changes contribute to raising energy consumption even at rest [16].

The results in the Table (1) showed the level of vitamin D in the obese adolescents group and adolescents with normal weight group, there was a significant ($P \leq 0.05$) decreases in the level of vitamin D in the group of participants suffering from obesity compared to the group of participants with normal weight, this compatible with study by Mohammed and Allwsh [17].

Numerous of previous studies proving the reverse relation between Obesity and vitamin D. One of an important study related to this problem is claiming that there is an adverse associated between ration of body fat and vitamin D levels [18]. Similarly, Mohammed and his colleagues [19], have found out that level of vitamin D has inverse relation with BMI, PBF, visceral fat tissue and subcutaneous fat tissue.

Low levels of vitamin D in obese people are attributed to the fact that excess body fat stores vitamin D because it is a fat-soluble vitamin [20]. So, that the cholecalciferol created by the skin (when the body is exposed to ultraviolet B rays of the sun) or obtained from the diet is partly reserved by the body fat before being transported to the liver for first hydroxylation (by first hydroxylation the cholecalciferol converted to -hydroxyvitamin D-25) [21].

On the other hand, many previous studies have suggested that vitamin D deficiency can lead to increased obesity through the following mechanism: vitamin D deficiency leads to decreased calcium absorption, as a result, the secretion of parathyroid hormone (PTH) increases to compensate for this deficiency and increase calcium levels in the blood, which increases the flow of calcium into fat cells and thus increases fat formation (lipogenesis) and Reduce fat breakdown (lipolysis) [22, 23].

The HOMA-IR index, which measures insulin resistance via means of an equation employing fasting insulin and fasting glucose, was significantly increased in obese adolescents (male and female) (Table 1).

Increased insulin resistance in obese individuals, as our study results showed, is associated with the accumulation of free fatty acids in tissues, thus disrupting intracellular insulin signaling. Chronic inflammation resulting from the release of inflammatory cytokines from adipose tissue also contributes to the inhibition of insulin sensitivity [24].

The results of our study showed that the level of IL-6, TNF- α and IL-1 β among obese adolescents was higher than adolescents with normal weight with no significant difference between male and female obese adolescents, this result was in agreement with Sakran and his group [25]. The explanation for this result can be that the impact of obesity linked inflammation which is the special feature of the obese adolescents [26].

Obese adolescents had significantly low HDL-C, and significantly high LDL-C, TG and TC. Since, insulin resistance disrupts leptin signaling, reducing satiety and increasing appetite and thus leads to obesity [27]. Also, insulin resistance stimulate fat storage and inhibit fat oxidation, and thus promoting obesity [28].

6. CONCLUSION

Adolescents obese exhibited greater insulin resistance accompanied by decreased vitamin D levels and increased markers of chronic inflammation. These factors reflect a complex interplay between metabolism and immune disorders, underscoring the need for early and effective interventions to limit the progression of obesity and reduce its long-term health risks.

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