

## Effects of Vitamin D injection and natural products on Blood Glucose Level of Diabetic Patient With Vitamin D Deficiency

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### Abstract

DM is the 4<sup>th</sup> leading cause of global mortality. By the year 2025 the disease incidence is expected to increase to over 380 million, 80% would be in developing countries. It has been proposed that vitamin D deficiency plays an important role in insulin resistance resulting in diabetes. The study was conducted to evaluate the effect of use some foods high in vitamin D content especially (parsley) and drug source of the same vitamin on diabetics and healthy. A selection of the sample is done by analyzing vitamin D in the blood and selecting patients with vitamin D deficiency. 100 people who suffered from type2 diabetes and 50 do not have diabetes and all of them suffer from vitamin D deficiency. Each group was divided into, group I is diabetic patients and vitamin D deficiency and are treated with vitamin D injection only. Group II is diabetics and vitamin D deficiency and are treated with vitamin D plus parsley injections. Group 3 is non-diabetics patients with vitamin D deficiency and are only injecting vitamin D. Group 4 is non-diabetics patients with vitamin D deficiency and need to inject vitamin D plus parsley. The treatment was done for 3 months. The results found that after treatment groups 2 and 4 which received 100 g fresh parsley with ampoules of vit. D were having the highest values statistical significantly ( $P \leq 0.05$ ) of serum vit. D. Calcium level of group 4 non-diabetic injected by vit. D and consumed 100g of fresh parsley was the highest value statistical significantly ( $P \leq 0.05$ ). HbA1C was high statistical significantly ( $P \leq 0.05$ ) in diabetic and non-diabetic patients before treatment. While, after injected by vit. D for groups (1 and 3) and vit. D plus 100g fresh parsley for groups 2 and 4 results showed reduction in serum HbA1C for all groups. The study concluded that there is a relationship between the level of vitamin D and improve the level of blood sugar in patients with diabetes type II. Eating rich vitamin D-containing food with fresh parsley gave the best results to type 2 diabetics and vitamin D deficiency patients.

## Introduction

Diabetes Mellitus (DM) is a metabolic disorder characterized by the presence of chronic hyperglycemia accompanied by greater or lesser impairment in the metabolism of carbohydrates, lipids and proteins (**Ahmed, 2002**). DM is the 4<sup>th</sup> leading cause of global mortality. Around 250 million people worldwide are living with disease. By the year 2025 the disease incidence is expected to increase to over 380 million and of this, 80% would be in developing countries (**Roglic, 2005**).

Type 2 Diabetes Mellitus is an epidemic disease and is associated with several chronic disorders. The number of patients with type 2 diabetes is rapidly increasing throughout the world, rising from approximately 171 million in 2000 to a projected 366 million in 2030 (**WHO, 2010**) (**Yang et al., 2010**).

Vitamin D (calciferol) comprises a group of fat soluble seco-sterols found naturally only in a few foods, such as fish-liver oils, fatty fish, mushrooms, egg yolks, and liver. The two major physiologically relevant forms of vitamin D are D2 (ergocalciferol) and D3 (cholecalciferol). Vitamin D3 is photosynthesized in the skin of vertebrates by the action of solar ultraviolet (UV) B radiation on 7-dehydrocholesterol (**Fieser and Fieser, 1959**). Vitamin D may play an important role in modifying the risk of cardio metabolic outcomes, including diabetes mellitus (DM), hypertension, and cardiovascular disease. The incidence of type 2 DM is increasing worldwide and results from a lack of insulin or inadequate insulin secretion following increases in insulin resistance. Therefore, it has been proposed that vitamin D deficiency plays an important role in insulin resistance resulting in diabetes (**Christensen et al., 2003**). Evidence linking vitamin D to insulin resistance and diabetes, several studies have indicated a relationship between vitamin D status and the risk of diabetes or glucose intolerance. Vitamin D has been proposed to play an important role and to be a risk factor

in the development of insulin resistance and the pathogenesis of type 2 DM, by affecting either insulin sensitivity or  $\beta$ -cell function, or both (Chiu *et al.*, 2004).

Parsley (*Petroselinum crispum*) as a spice is produced in vegetable garden, it is a very rich source of vitamins C and E, carotene, thiamin and organic minerals, because of its high water content (78-82%,w\w.) Parsley is ordinarily dried for market, in order to inhibit microorganisms growth and prevent degradation caused by biochemical reactions (Soysal, 2004). Yanardag *et al.*, (2003) observed that parsley extract did not increase insulin release from  $\beta$  cells of the pancreas but decreased blood glucose levels by causing usage of glucose via extra pancreatic ways.

Parsley leaves, which are commonly used and found everywhere with absolutely no reported toxicity, possess excellent antihyperglycemic effect and this may be mediated by  $\alpha$  glycosidase inhibition. Thus these leaves can offer a more reliable and potential alternative to acarbose which is currently the most widely used  $\alpha$  glycosidase inhibitor for diabetes mellitus. Anti hyperglycemic activity of parsley (*Petroselinum crispum*) is not due to improvement and regeneration of secretory granules and  $\beta$ -cells of pancreas islets. Furthermore, parsley(*Petroselinum crispum*) improves hyperglycemia-induced heart and aorta oxidative damage via its antioxidant activity in the heart and aorta tissue. However, it did not showed significant effect on non-enzymatic glycosylation of skin proteins in diabetic rats (Popović *et al.*, 2007). Therefore, the study aimed to evaluate the relationship between vitamin D intake and level of blood glucose in diabetic patient and the effect of parsley intake on health status.

## **SUBJECTS and METHODS**

### **Subjects:**

**Ethical issues:** The study was conducted after explaining to the participants the steps of the study and its objectives. Only those who agreed were included . Informed consent was taken at the beginning of the study from all the participants in the

study according to Helsinki deceleration s of biomedical ethics (Temple, 2003). The study was registered with Clinical Trials GOTH1 (no. I N000084) ON 21/12/2015.

**Studied Sample:** All sample were collected by using simple random sample technique from the outpatient clinic, at Egyptian National Nutrition Institute (N.N.I.- Cairo-Egypt) and the National Institute of Diabetes and Endocrinology (NIDE- Cairo-Egypt ). The sample was selected from among 600 people who underwent the necessary analysis determined HbA1c , Vitamin D deficiency and collected 50 diabetic and 50 non diabetic subjects. **Inclusion criteria include** type II diabetic adult, ages eligible for study between 20 to 45 Years, genders eligible for study: females, matched sample for BMI and metabolic control, acceptance of patients. aged between 20 -45 years of both gender chosen to meet the study requirements, Having low 25 hydroxy Vitamin D below (**20 ng / ml**). Subjects were excluded if they had significant renal, liver, cancer disease, and thyroid disease. Although controlled hypertension was acceptable if blood pressure was less than 140/90 mm Hg on medications.

**Study design:** This study was an experimental (intervention) clinical studied the relationship between vitamin D intake and diabetes mellitus and the effect of vitamin D supplement injection with or without low caloric dietary intervention on diabetic subjects After determination of vitamin D at baseline for six hundreds subjects, subjects under 20 ng/ml of vitamin D excluded, also, subject suffering from any disease like liver ,kidney , menopause ,lactating , pregnancy ,cancer and thyroid Hormone were excluded Reminding 100 hundred subject (females). Suffering from vitamin D were divided into main four groups as follow:

**Group (1):** Twenty five diabetic female aged 25:45 years received vitamin D injection by devarol ampoule (200,000) IU for 3 months.

**Group(2):** Twenty five diabetic female aged 25:45 years injected vitamin. D by ampule devarol(200,000) IU

beside 100g of fresh parsley content about 449.77mg (18000 IU) from vitamin .D for 3 months .

**Group(3):** Twenty five non - diabetic female aged 25:45years deficiency in vitamin . D less than 20ng/ml were injected by devarol ampoule(200,000) IU for 3 months.

**Group(4):** Twenty five non - diabetic female aged 25:45years deficiency in vitamin .D taken devarol ampoule(200,000) IU + 100g fresh Parsley contain 449.77mg(18000 IU) vitamin D for 3 months.

### **Data collection and intervention phase:**

Subjects on vitamin D supplementation trial lasting 3 months. The study consisted of 4 visits Zero time and 3 subsequent follow up visits at 0 ,4 ,8 and 12 weeks. were all subjects were injected by Vitamin D (200.000 IU). at Zero,4 time and 8 weeks also with measuring the weight , BMI. While they were measured for weight and BMI, HbA1C only without injection at the Zero time and 12 weeks . At all visits, anthropometrics were measured and blood was drawn at base line and at the end of the study for testing.

### **3. 1. method:**

**Data collection technique:** An interviewing questionnaire was designed to collect the data and it was revised by the researcher . Personal and family socio-demographic characteristics: personal data (name, age, address, telephone number, marital status) and the socio-demographic characteristics. The socioeconomic score was calculated according to **(Park and Park, 1979) (Appendix 2)**. This score is based on the assumption that the level of parent's occupation and education can determine the social standard of the family. The social standard is based on the classification of the overall scale into three levels;

1. Low level if the overall score <9.
2. Intermediate level if the overall score ranged from 9 to 18.
- 3- High level if the overall score ranged from 19 to 28

**Anthropometric measurements** were made while the subjects were minimally clothed and bare foot using standard equipment (Moayeri *et al.*,2006) Height and body weight were measured and body mass index (BMI) was calculated from the following equation : $BMI = \text{Weight (Kg) /height (cm)}^2$  Participants were classified as there is some debate about where on the BMI scale the dividing lines between categories should be placed (Bauer, Henry ,(2018) Commonly accepted BMI ranges are underweight: under 18.5 kg/m<sup>2</sup>, normal weight: 18.5 to 25, overweight: 25 to 30, obese: over 30. People of Asian descent have different associations between BMI, percentage of body fat, and health risks than those of European descent, with a higher risk of type 2 diabetes and cardiovascular disease at BMIs lower than the WHO cut-off point for overweight, 25 kg/m<sup>2</sup>, although the cutoff for observed risk varies among different Asian populations (WHO, 2014).

Twenty-Four hour recall, for the exact foods and beverages intake during 24 hour period for 3 days ,including weekend day and the previous or next 2 days( Wednesday ,Thursday , Friday )or (Friday , Saturday , Sunday )Energy and nutrients intakes were calculated by using a computer program based on food composition table of the National Institute of Nutrition (NNI, 2006).Results were compared with Dietary References Intakes (DRIs , 2002).

#### **Laboratory analysis:**

**Blood picture:** in all subjects groups, Venous blood samples were collected from the ante-cubital vein of all individuals by a trained Nurse at the clinic for the purpose of the study were collected after 12 hours fasting . The blood was centrifuged for 10 minutes at 3000 r.p.m to separate serum, serum was carefully separated into clean eppendorf and keep in deep frozen for analysis as described by (Schermer, 1967).

**1,25-hydroxy Vitamin D:** Collection took place in a serum collection tube and sent to the NNI lab. The serum level of 25(OH)D was used as an indicator for vitamin D status

because this metabolite reflects the supply of vitamin D metabolites both in the diet and through skin synthesis. Moreover, hydroxylation of 25(OH)D to 1.25(OH)2D3 (active vitamin D) occurs in several tissues: the half-life of 25-OH-D is 2–3 weeks while the half-life of 1.25(OH)2D3 is approximately 6 h (Chris G. Velentzas,2012). We used the EUROIMMUN Enzyme-linked Immune-sorbent Assays (ELISA) test kits for determination of 25 vitamin D as detailed.

**Glycosylated hemoglobin (HbA1C) estimation** Percentage of (HbA1C) were determined 25(OH)D were measured using Enzyme-linked Immune sorbent Assays (ELISA) including greater convenience (fasting not required) greater pre – analytical stability according to ADA(2010) The World Health Organization (WHO) suggests the following diagnostic guidelines for diabetes:

- **HbA1c below 42 mmol/mol (6.0%):** Non-diabetic
- **HbA1c between 42 and 47 mmol/mol (6.0–6.4%):** Impaired glucose regulation (IGR) or Pre diabetes
- **HbA1c of 48 mmol/mol (6.5%) or over:** Type 2 diabetes.

**Determination of lipids profile:** (total cholesterol, triglycerides, free fatty acids and HDL-cholesterol Cholesterol determined calorimetrically according to Allain, (1974). free fatty acids and HDL-cholesterol by Singh et al.,( 1991).

Triglycerides will be carried out according to Fossati and Prencipe (1982).

Low – density lipoprotein (LDL)cholesterol according to Friedewald, et al ., (1972) calculated with a few restriction , as follows:  $LDL(mg/dl) = total\ cholesterol - HDL - total\ triglycerides / 5$  .

**Determination of Chemical Composition of parsley:** protein, fat, moisture and ash contents were determined in fresh parsley according to AOAC (2010) .The carbohydrate was

calculated by difference. Calcium and Iron were determined using flame atomic absorption spectrophotometry (model 5100 pc , perkin – Elmer Norwalk CT) According to **Metger and Nielsen ,.(2017)**

**Statistical Analysis:** results were expressed as the mean  $\pm$  SD .Data for multiple variable comparisons were analyzed by one – way analysis of variance (ANOVA).For the comparison of significance between groups , Duncan´ s test was used as a post hoc test according to the statistical package program. (**P. Armstage and Berry,1987**).

### Results and Discussion

Data present in table (1) showed the chemical composition of fresh parsley. The results indicated that moisture (84.6g/100g), vitamin D (449.77mg/100g) fresh parsley and Calcium (210mg/100g) fresh parsley were high in fresh parsley while Ash(2.2g), protein(3.3g), fat(0.4g), Carbohydrates(8.2 g) and iron (6.55g) were in low.

**Table (1) Chemical composition of parsley as a mean  $\pm$  SD 100g fresh Weight:**

Parameter	Fresh parsley
Moisture(g/100g).	84.6 $\pm$ 2.3
Ash(g/100g).	2.2 $\pm$ 0.1
Protein(g/100g).	3.3 $\pm$ 0.21
Fat(g/100g).	0.4 $\pm$ 0.35
Carbohydrate(g/100g).	8.2 $\pm$ 0.23
vitamin D(mg/100g).	449.77 $\pm$ 0.24(18000IU)
Calcium(mg/100g).	210 $\pm$ 0.51
Iron (mg/100g).	6.55 $\pm$ 0.26

(USDA., 2010). (National Center for Agricultural Research, (2017)

The characteristics of the studied women as illustrated in table 2 indicated that no significant between age and efficient score. (**Lindquist, et al., 2000**) suggested that the use of the tape recorded for estimating energy intake does not result in accurate assessments among adult, although this technique may be useful for specific subgroups. From this study it is cleared that no significant differences between age and efficient score



of food record in the age under from sample. Therefore, the present results were in agreement. Also, the study results showed that no significant between efficient score and adult sex. The results are agreement with **Salamoun, et al., (2005)** which stated that body had a significantly higher mean daily calcium intake and vitamin D. in addition, the data represents that there are no significant between the efficient score and parent's education levels. Our findings are similar to **Perron and Endres., (1985)** they stated that no significant correlation was found between knowledge and attitudes, such as a concern for weight and a dependence on parents for food selections.

**Table (2): Characteristics of the studied women.**

Socio demographic characteristics	No=(100)	%
<b>Age (years)</b>		
20-< 30	54	54
30-< 40	40	40
40 ≥	6	6
<b>Marital status</b>		
Single	5	5
Married	85	85
Widow	5	5
Divorced	5	5
<b>Family size</b>		
≤ 4	7	7
> 4	93	93
<b>Occupation</b>		
Rural	80	80
Urban	20	20
<b>*Social status</b>		
Low	75	75
Middle	23	23
High	2	2

\*Social classification according to **Park and Park., (1979)** .

**Anthropometric Measurements of groups:**

**Table (3): Means of Anthropometric Measurements, (Weight, Body Mass Index(BMI) and Kilo calories(K.Cal.):**

Groups	Wight (Kg)		BMI		K. CaL	
	Before	After	Before	After	Before	After
	m±S.D	m±S.D	m±S.D	m±S.D	m±S.D	m±S.D
1	84.84±17.14 <sup>a</sup>	81.04±13.98 <sup>a</sup>	31.92±6.91 <sup>a</sup>	30.49±5.65 <sup>a</sup>	2091.59±498.7 <sup>a</sup>	1555.4±101.2 <sup>a</sup>
2	81.44±15.64 <sup>a</sup>	75.60±12.54 <sup>ab</sup>	28.83±4.10 <sup>b</sup>	26.83±3.27 <sup>b</sup>	2171.8±375.1 <sup>a</sup>	1562.0±106.3 <sup>a</sup>
3	76.84±10.11 <sup>a</sup>	73.65±9.74 <sup>b</sup>	27.29±3.16 <sup>b</sup>	26.10±3.06 <sup>b</sup>	2292.0±475 <sup>a</sup>	1540.0±93.5 <sup>a</sup>
4	82.60±14.11 <sup>a</sup>	77.5±11.60 <sup>ab</sup>	29.30±4.63 <sup>ab</sup>	27.52±3.60 <sup>b</sup>	2407.0±53.3 <sup>a</sup>	1523.0±88.4 <sup>a</sup>

\*kg:kilo gram

- ✓ **Group 1: Diabetes female with vitamin D deficiency and injected with Devarol .**
- ✓ **Group2: Diabetes female with vitamin D deficiency and injected with Devarol + Parsley(100g /per day)**
- ✓ **Group3: non-diabetic female with Vitamin D deficiency and injected with Devarol**
- ✓ **Group4: non-diabetic female with Vitamin D deficiency and injected with Devarol + Parsley(100g /per day)**

Concerning of weight data in Table (3) showed that there were slightly decreased insignificantly ( $P>0.05$ ) between groups 1 and 2 before and after treatment with means±SD were  $84.84\pm17.14$ ,  $81.44\pm15.64$  vs.  $81.04\pm13.98$  and  $75.60\pm12.54$  kg, respectively. Also, the same results showed that there were slightly decreased insignificantly ( $P>0.05$ ) in weights between groups 3 and 4 non-diabetic patients before and after treatment. Concerning of BMI data in Table (3) showed that there were decrease statistical significant differences ( $P\leq0.05$ ) between groups 1 and 2 after treatment with values of mean ± SD. were  $31.92\pm6.91$  and  $26.83\pm3.27$  respectively. While there were statistical significant differences ( $P\leq0.05$ ) between groups 3 and 4 after treatment with values of mean±SD were  $26.10\pm3.06$  and  $27.52\pm3.60$ , respectively. Moreover, group 2 after treatment was the lowest value  $26.83\pm3.27$  for BMI.

Results in Table (3) contained the values of K. cal. which were  $2091.59 \pm 498.7$ ,  $2171.8 \pm 375.01$ ,  $2292 \pm 475.0$ ,  $2407.53 \pm 53.3$ ,  $1555.4 \pm 106.3$ ,  $1562.0 \pm 106.3$ ,  $1540.0 \pm 93.5$  and finally  $1523.0 \pm 88.4$  for groups 1, 2, 3 and 4 before and after treatments respectively. From the above results we can be observed that there were no statistical significant differences ( $P > 0.05$ ) between all groups under study.

**Table (4): Means of Dietary Intake (Protein, Fat and Carbohydrate) for Different Groups:**

Groups	Protein( g)		Fat( g)		Carbohydrate (g)	
	Before	After	Before	After	Before	After
	m±S.D	m±S.D	m±S.D	m±S.D	m±S.D	m±S.D
1	94.8±17.1 <sup>a</sup>	81.0±13.9 <sup>b</sup>	69.13±11.0 <sup>ab</sup>	34.5±2.2 <sup>a</sup>	313.3±74.8 <sup>a</sup>	233.2±15.07 <sup>a</sup>
2	91.4±15.6 <sup>a</sup>	75.6±12.5 <sup>c</sup>	65.33±8.3 <sup>ab</sup>	34.6±2.3 <sup>a</sup>	325.2±56.2 <sup>a</sup>	234.2±15.8 <sup>a</sup>
3	76.8±10.1 <sup>a</sup>	73.3±9.2 <sup>b</sup>	56.7±10.4 <sup>b</sup>	34.1±2.05 <sup>a</sup>	343.2±71.2 <sup>a</sup>	230.8±13.9 <sup>a</sup>
4	82.6±14.1 <sup>a</sup>	77.7±11.1 <sup>ab</sup>	80.2±11.8 <sup>a</sup>	33.8±1.9 <sup>a</sup>	360.6±80.01 <sup>a</sup>	228.3±12.8 <sup>a</sup>

- ✓ **Group 1: Diabetes female with vitamin D deficiency and injected with Devarol .**
- ✓ **Group2: Diabetes female with vitamin D deficiency and injected with Devarol + Parsley(100g /per day)**
- ✓ **Group3: non-diabetic female with Vitamin D deficiency and injected with Devarol**
- ✓ **Group4: non-diabetic female with Vitamin D deficiency and injected with Devarol + Parsley(100g /per day)**

Concerning protein intake results in Table (4) showed that there were statistical significant differences ( $P \leq 0.05$ ) between groups 1 and 2 after treatment with mean  $\pm$  S.D values were  $81.0 \pm 13.9$  vs.  $75.6 \pm 12.5$  g, respectively. While there were no statistical significant differences ( $P > 0.05$ ) between the same groups before treatments. Values of protein intake were  $94.8 \pm 17.1$ ,  $91.4 \pm 15.6$ ,  $81.0 \pm 13.9$  and  $75.6 \pm 12.5$ g for groups 1 and 2 before and after treatments in diabetic patient respectively. Regarding fat consumed results in the above table showed that there were statistical significant differences ( $P \leq 0.05$ ) between diabetic and non-diabetic 1, 2, 3 and 4 groups before and after

treatments in fat intake. Values were  $69.13 \pm 11.0$ ,  $65.33 \pm 8.3$ ,  $56.7 \pm 10.4$  and  $80.2 \pm 11.8$  before treatment vs.  $34.5 \pm 2.2$ ,  $34.6 \pm 2.3$ , and  $34.1 \pm 2.05$  and  $33.8 \pm 1.9$  g after treatment for groups 1, 2, 3 and 4 respectively. Moreover, carbohydrate consumed results in table (4) showed that there were no statistical significant differences ( $P \leq 0.05$ ) between diabetic groups before and after treatment the values mean  $\pm$  S.D were  $313.3 \pm 74.8$  and  $325.2 \pm 56.2$  vs  $233.2 \pm 15.07$  and  $234.2 \pm 15.8$  g, respectively. Also there were no statistical significant differences ( $P \leq 0.05$ ) for non-diabetic patient between groups 3 and 4 before and after treatment with values of mean  $\pm$  S.D were  $343.2 \pm 71.2$  and  $343.2 \pm 71.2$  vs.  $230.8 \pm 13.9$  and  $230.8 \pm 13.9$  g, respectively.

**Table. (5): Means of Mineral Intake (Iron (Fe) and Calcium ( Ca)).**

Group	Fe mg		Ca mg	
	Before	After	before	After
	M $\pm$ S.D	M $\pm$ S.D	M $\pm$ S.D	M $\pm$ S.D
1	10.04 $\pm$ 3.51 <sup>a</sup>	15.52 $\pm$ 3.12 <sup>a</sup>	215.3 $\pm$ 8.8 <sup>a</sup>	233.3 $\pm$ 7.97 <sup>b</sup>
2	9.28 $\pm$ 4.32 <sup>a</sup>	16.08 $\pm$ 2.93 <sup>a</sup>	214.2 $\pm$ 4.57 <sup>a</sup>	258.6 $\pm$ 8.5 <sup>a</sup>
3	10.55 $\pm$ 3.87 <sup>a</sup>	15.15 $\pm$ 2.50 <sup>a</sup>	215.14 $\pm$ 4.7 <sup>a</sup>	260.8 $\pm$ 5.8 <sup>a</sup>
4	11.33 $\pm$ 6.43 <sup>a</sup>	15.40 $\pm$ 2.69 <sup>a</sup>	225.54 $\pm$ 7.9 <sup>a</sup>	259.1 $\pm$ 6.06 <sup>a</sup>

- ✓ **Group 1: Diabetes female with vitamin D deficiency and injected with Devarol .**
- ✓ **Group2: Diabetes female with vitamin D deficiency and injected with Devarol + Parsley(100g /per day)**
- ✓ **Group3: non-diabetic female with Vitamin D deficiency and injected with Devarol**
- ✓ **Group4: non-diabetic female with Vitamin D deficiency and injected with Devarol + Parsley(100g /per day)**

Results in Table (5) showed that there were no statistical significant differences ( $P \leq 0.05$ ) in Fe (mg) intake between diabetic patients for groups 1 and 2 before and after treatment. Values of mean  $\pm$  SD were  $10.04 \pm 3.51$ ,  $9.28 \pm 4.32$  vs.  $15.52 \pm 3.12$  and  $16.08 \pm 2.93$  mg before and after treatment

for diabetic patients suffered deficiency in vit.D. Besides that, results for diabetic patient group 2 was the lower value before treatment with means  $\pm$  SD 9.28  $\pm$  4.32 respectively. The study results were agreement with those obtained by **Tate et al.,(2007)** and **Weiss et al., (2007)** they found increment in iron intake by 22% after modified diet while the increment in our study iron was increased by 42- 47% this result may be referred to vitamin D injection and fresh parsley intake as a source of vitamins D and C. Concerning calcium intake results in the above found that there were higher significant differences ( $P \leq 0.05$ ) between group1 after treatment and among all groups. Values of mean  $\pm$  SD were 215.3  $\pm$  8.8, 214.2  $\pm$  4.57, 215.14  $\pm$  4.7, 225.54  $\pm$  7.9 vs. 233.3  $\pm$  7.97, 258.6  $\pm$  8.5, 260.8  $\pm$  5.8 and finally 259.1  $\pm$  6.06 mg for groups from 1 to 4 respectively. The increment in calcium intake ranging from 8.6 for group 1 to 16% for group 3 this increment referred to supplementation of vitamin D and vitamin C in fresh parsley. Our results were confirmed with **Holick and Chen., (2008)** they found that vitamin c caused increased calcium absorption about 15% to 22% from the diet, the different between our result and this result referred to source of vitamin.

**Table (6): Means of Serum Vitamin D (V.D), Calcium(Ca) and glycated Hb( HbA1c).**

Groups	V.D (ng/ml)		Ca (mg/dl)		HbA1c (mg/dl)	
	Before	After	Before	After	Before	After
	m $\pm$ S.D	m $\pm$ S.D	m $\pm$ S.D	m $\pm$ S.D	m $\pm$ S.D	m $\pm$ S.D
1	16.55 $\pm$ 7.52 <sup>a</sup>	75.54 $\pm$ 7.86 <sup>b</sup>	9.18 $\pm$ 2.12 <sup>a</sup>	9.17 $\pm$ 1.18 <sup>b</sup>	7.26 $\pm$ 1.21 <sup>a</sup>	5.0 $\pm$ 0.59 <sup>a</sup>
2	14.09 $\pm$ 3.31 <sup>a</sup>	102.97 $\pm$ 8.27 <sup>a</sup>	8.92 $\pm$ 1.06 <sup>a</sup>	9.94 $\pm$ 0.50 <sup>a</sup>	7.65 $\pm$ 1.19 <sup>a</sup>	4.27 $\pm$ 0.63 <sup>bc</sup>
3	14.40 $\pm$ 5.15 <sup>a</sup>	82.07 $\pm$ 5.76 <sup>b</sup>	8.42 $\pm$ 0.84 <sup>a</sup>	9.02 $\pm$ 1.20 <sup>b</sup>	4.85 $\pm$ 0.61 <sup>b</sup>	4.51 $\pm$ 0.82 <sup>b</sup>
4	13.91 $\pm$ 2.60 <sup>a</sup>	102.97 $\pm$ 8.27 <sup>a</sup>	8.69 $\pm$ 0.60 <sup>a</sup>	10.05 $\pm$ 0.98 <sup>a</sup>	5.14 $\pm$ 0.44 <sup>b</sup>	4.07 $\pm$ 0.57 <sup>c</sup>

- ✓ **Group 1: Diabetes female with vitamin D deficiency and injected with Devarol .**
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- ✓ **Group3: non-diabetic female with Vitamin D deficiency and injected with Devarol**
- ✓ **Group4: non-diabetic female with Vitamin D deficiency and injected with Devarol + Parsley(100g /per day)**
- ✓ **Reference range: Vitamin D (N>=30 mg/ml-80 , Insufficiency 20-30 ,Deficient<20ng/ml).**
- ✓ **Calcium (N:9.2 – 11.0 mg%).**
- ✓ **HbA1c (Normal range:4.2-6.2%,good control:5.5-6.8%,fair control:6.8-7.6%,poor control:above:7.6%).**

Results in Table (6) contained the values of vitamin D which were  $16.55 \pm 7.52$ ,  $14.09 \pm 3.31$ ,  $75.54 \pm 7.86$ ,  $102.97 \pm 8.27$ ,  $14.40 \pm 5.15$ ,  $13.91 \pm 2.60$ ,  $82.07 \pm 5.76$  and  $94.75 \pm 7.17$  ng/ml for groups 1, 2 before and after treatments diabetic patient with injected by vitamin D ampule and received 100 g of fresh parsley. While, groups 3 and 4 before and after treatments non-diabetic patient and suffering from deficiency in vitamin D and injected by ampule vit. D with 100 g fresh parsley for group 4 respectively. From the above results we can be noticed that all groups before treatment were the lowest values of serum vit. D for diabetic and non-diabetic patients. While, after treatment groups 2 and 4 which received 100 g fresh parsley with ampules of vit. D were the highest values statistical significantly ( $P \leq 0.05$ )  $102.97 \pm 8.27$  and  $102.97 \pm 8.27$  ng/dl respectively. Concerning serum calcium results in Table (6) showed that there were statistical significant differences ( $P \leq 0.05$ ) between diabetic patients before and after injected by ampule and ampule plus 100 g of fresh parsley. Patient who injected by ampule and consumed 100 g fresh parsley group 2 had the higher value statistical significantly than before treatment  $9.94 \pm 0.50$  Vs.  $8.92 \pm 1.06$  mg/ dl. Besides that, group 4 non-diabetic injected by vit. D ampule and consumed 100g of fresh parsley was the highest value significantly ( $P \leq 0.05$ ) compared to group 4 before treatment with value  $10.05 \pm 0.98$  vs.  $8.69 \pm 0.60$  mg/dl. Regarding glycated hemoglobin a fraction HbA1C is the most interest serving as a retrospective indicator of the average

glucose concentration. HbA1C is recommended as an essential indicator for the monitoring of blood glucose control. The blood HbA1c  $\geq 6.5\%$  is considered as diabetes (Selvin, *et al.*, 2010). Concerning the results of HbA1C were higher significantly ( $P \leq 0.05$ ) in diabetic and non-diabetic patients before treatment with values  $7.26 \pm 1.21$ ,  $7.65 \pm 1.19$ ,  $7.65 \pm 1.19$ , and  $7.65 \pm 1.19$  mg/dl While, after injected by vit. D for groups (1 and 3) and vit. D plus 100g fresh parsley for groups 2 and 4 results showed that decreased in serum HbA1C for all groups. Beside that groups 2 and 4 who consumed 100g fresh parsley with injected vit.D were the lowest values in serum HbA1C. The values of HbA1C after treatment were  $5.0 \pm 0.59$ ,  $4.27 \pm 0.63$ ,  $4.51 \pm 0.82$  and  $4.07 \pm 0.57$  mg/dl for groups 1, 2, 3 and 4 respectively. Our results were agreement with those obtained by Chiu, *et al.*, (2004) who observed that a positive correlation of 25-hydroxyvitamin D concentrations with insulin sensitivity a lot of studies have indicated a relationship between vitamin D status and the risk of diabetes or glucose intolerance. vitamin D has been proposed to play an important role and be a risk factor in the development of insulin resistance and the pathogenesis of type 2 DM by  $\beta$ -cell function (Chiu, *et al.*, 2004; Deleskog, *et al.*, 2012 and Forouhi, *et al.*, 2012). The presence of a vitamin D response element in the insulin gene promoter (Maestro, *et al.*, 2003). Activates peroxisome proliferator activator receptor gene (Dusso, *et al.*, 2005). Also, many studies have examined the predictive value of vitamin D on future risks of type 2 diabetes mellitus (Mattila, *et al.*, 2007; Forouhi, *et al.*, 2012 and Knekt, *et al.*, (2008). Also the results in this study agree with Tuorkey and Abdul-Aziz., (2010) who noticed that vitamin D improves glucose tolerance. Vitamin D could also prevent type 2 Diabetes through its role as efficient antioxidant, type 2 diabetes is associated with systemic inflammation so that it has been linked primary to insulin resistance but elevated cytokines may also play a role in  $\beta$ -cell apoptosis. Vitamin D may improve insulin sensitivity and promote  $\beta$ -cell survival by

directly modulating the generation and effects of cytokines (Milner, *et al.*, 2010). Previous studies have reported significant inverse associations of vitamin D with IR (Alemzadeh, *et al.*, 2008) and  $\beta$ -cell dysfunction (Wu, *et al.*, 2009) including cross-sectional study in the current cohort (Kayaniyil, *et al.*, 2010). Only two prospective studies have been conducted to date, both of which reported significant inverse associations of baseline 25(OH)D with IR after 5 and 10 years of follow-up, respectively, in largely white cohorts (Gagnon, *et al.*, 2011). On the other hand, there were relationship between insufficient vitamin D, calcium status and type 2 DM. However, the available human data are limited because most observational studies are cross-sectional, whereas prospective studies have not measured 25-OHD concentration, and there is a paucity of randomized controlled trials with vitamin D and/or calcium supplementation specifically designed for outcomes related to type 2 DM. Although the evidence to date suggests that vitamin D and calcium deficiency influences postprandial glycaemia and insulin response while supplementation may be beneficial in optimizing these processes, our understanding of the exact mechanisms by which vitamin D and calcium may promote  $\beta$ -cell function or ameliorate insulin resistance and systemic inflammation is incomplete.

HbA1C, blood pressure, and cholesterol goals may need to be adjusted for the individual based on age, duration of diabetes, health history, and other present health conditions. Further recommendations for individualization of goals can be found in the ADA Standards of Medical Care in Diabetes (ADA, 2014). Effective nutrition therapy interventions may be a component of a comprehensive group diabetes education program or an individualized session. Andrews, *et al.*, (2011) reported HbA1C reductions are similar or greater than what would be expected with treatment with currently available pharmacologic treatments for diabetes. The reduction of



HbA1C for type 1 diabetes was 20.3 to 21% and type 2 diabetes was 20.5 to 22% (Coppell, *et al.*, 2010).

**Table (7): Means of Lipid profile(Low density lipoprotein(LDL), High Density Lipoprotein (HDL),Cholesterol, Triglycerides(TG)and Very low – Density Lipoproteins cholesterol(VLDL) for different groups.**

Groups	LDL (mg/dl %)		HDL (mg/dl %)		Cholesterol (mg/dl %)		TG (mg/dl %)		VLDL (mg/dl %)	
	Before	After	Before	After	Before	After	Before	After	Before	After
	m±S.D	m±S.D	m±S.D	m±S.D	m±S.D	m±S.D	m±S.D	m±S.D	m±S.D	m±S.D
1	91.68±10.06 <sup>a</sup>	39.13±9.59 <sup>a</sup>	38.44±3.99 <sup>a</sup>	51.36±4.84 <sup>b</sup>	179.16±9.5 <sup>b</sup>	103.8±5.46 <sup>a</sup>	245.16±9.08 <sup>b</sup>	70.12±8.34 <sup>a</sup>	49.03±1.81 <sup>b</sup>	27.62±1.66 <sup>a</sup>
2	96.28±10.81 <sup>a</sup>	23.99±5.78 <sup>b</sup>	38.28±6.12 <sup>a</sup>	59.48±5.23 <sup>a</sup>	185.48±8.65 <sup>ab</sup>	93.92±3.12 <sup>c</sup>	254.56±8.87 <sup>a</sup>	49.04±5.79 <sup>b</sup>	50.91±1.77 <sup>a</sup>	26.4±1.15 <sup>ab</sup>
3	82.87±10.63 <sup>a</sup>	36.0±7.89 <sup>a</sup>	45.92±6.05 <sup>a</sup>	52.05±5.16 <sup>b</sup>	180.64±5.97 <sup>ab</sup>	100.95±6.18 <sup>b</sup>	259.24±6.26 <sup>a</sup>	53.00±8.03 <sup>a</sup>	51.84±1.25 <sup>a</sup>	25.32±1.60 <sup>b</sup>
4	94.59±9.44 <sup>a</sup>	25.17±9.23 <sup>b</sup>	43.80±5.11 <sup>a</sup>	59.43±5.12 <sup>a</sup>	187.2±5.99 <sup>a</sup>	95.56±5.53 <sup>c</sup>	244.04±5.59 <sup>b</sup>	50.46±7.55 <sup>b</sup>	48.8±1.11 <sup>b</sup>	20.7±1.51 <sup>c</sup>

- ✓ **Group 1: Diabetes female with vitamin D deficiency and injected with Devarol .**
- ✓ **Group2: Diabetes female with vitamin D deficiency and injected with Devarol + Parsley(100g /per day)**
- ✓ **Group3: non-diabetic female with Vitamin D deficiency and injected with Devarol**
- ✓ **Group4: non-diabetic female with Vitamin D deficiency and injected with Devarol + Parsley(100g /per day)**
- ✓ **Reference range :LDL(Optimal<100,Borderline high130-159,high160-189,very high>=190) (mg/dl %).**
- ✓ **HDL(M:->40,F:->50) (mg/dl %).**
- ✓ **Cholesterol (Desirable<200,Borderline high200-239,high>=240) (mg/dl %).**
- ✓ **TG (Normal<150, Borderline high150-199, high200-499,very high>499) (mg/dl %).**

Data in Table (7) showed that there were statistical significant differences ( $P \leq 0.05$ ) between groups 1, 2 before and after treatment in serum LDL with means  $\pm$  S.D were  $91.68 \pm 10.06$  and  $96.28 \pm 10.81$  vs.  $39.13 \pm 9.59$  and  $23.99 \pm 5.78$  mg/dl, respectively. Also there were significant differences ( $P \leq 0.05$ ) between groups 3 and 4 before and after treatment

with means  $\pm$  S.D were  $82.87 \pm 10.63$  and  $94.59 \pm 9.44$  vs.  $36.0 \pm 7.89$  and  $25.17 \pm 9.23$  mg/dl respectively. Moreover, group 2 and group 4 after treatment were the lowest values statistical significantly ( $P \leq 0.05$ )  $23.99 \pm 5.78$  and  $25.17 \pm 9.23$  mg/dl respectively. From the above results we can be observed that patients who injected by vit. D ampule and consumed 100g of fresh parsley for diabetic and non-diabetic patients were lowest values than groups' injected vit. D ampule only. Concerning of serum HDL data in Table (9) showed that there were statistical significant differences ( $P \leq 0.05$ ) between groups 1, 2 before and after treatment with mean  $\pm$  S.D were  $38.44 \pm 3.99$ ,  $38.28 \pm 6.12$ , VS  $51.36 \pm 4.84$  and  $59.48 \pm 5.23$  mg/dl respectively. Also there were statistical significant differences ( $P \leq 0.05$ ) between groups 3, 4 before and after treatment with mean  $\pm$  S.D were  $45.92 \pm 6.05$ ,  $43.80 \pm 5.11$  VS.  $52.05 \pm 5.16$  and  $59.43 \pm 5.12$  mg/dl , respectively. Concerning of serum total cholesterol results in Table (9) showed that there were statistical significant differences ( $P \leq 0.05$ ) between groups 1, 2 before and after treatment with means  $\pm$  S.D  $179.16 \pm 9.5$ ,  $185.48 \pm 8.65$ ,  $103.8 \pm 5.46$  and  $93.92 \pm 3.12$  mg/dl , respectively. On the other hand group 2 after treatment was the lowest value  $93.92 \pm 3.12$  mg/dl. Also, from the same table there were statistical significant differences ( $P \leq 0.05$ ) between all normal groups which the lowest value  $95.56 \pm 5.53$  mg/dl for group 4 after treatment. Beside that data found that serum T.G were statistical significant differences ( $P \leq 0.05$ ) between diabetic groups while group 2 after treatment was the lowest value significantly ( $P \leq 0.05$ )  $49.04 \pm 5.79$  mg/dl. Moreover, data in the same table showed that there were statistical significant differences ( $P \leq 0.05$ ) between normal groups before and after treatment. On the other hand group 4 after treatment was the lowest value  $50.46 \pm 7.55$  mg/dl, respectively. Data in table (7) found that serum VLDL were no statistical significant differences ( $P > 0.05$ ) between groups 2 and (3 and 4) after treatment with values of mean  $\pm$  S.D were  $9.80 \pm 1.15$ ,  $10.60 \pm 1.60$ ,  $10.09 \pm 1.51$  mg/dl, respectively. We could be

observed that the lowest value in group 2 after treatment. Beside that there were statistical significant differences ( $P \leq 0.05$ ) between groups 1 and 2 before treatment and group 1 after treatment with values of mean  $\pm$  S.D were  $49.03 \pm 1.81$ ,  $50.91 \pm 1.77$  and  $14.02 \pm 1.66$  VS.  $51.84 \pm 1.25$ ,  $48.8 \pm 1.11$  mg/dl, respectively. The lipid abnormalities associated with type 2 diabetes is defined by a high concentration of TG and small dense LDL and a low concentration of HDL cholesterol. Serum LDL cholesterol levels are generally normal, insulin resistance is believed to this Atherogenic dyslipidemia by increasing the hepatic secretion VLDL and other Apolipoprotein  $\beta$  containing lipoprotein particles as result of increased free fatty acid flux to the liver (**Krauss and Siri, 2004 and Lewis, et al., 2002**). This may also be result of a diminished supportive effect of insulin on Apo  $\beta$  secretion either at the level of the regulation of Apo  $\beta$  degradation or inhibition of microsomal TG transfer protein activity (**Malmstrom, et al., 1997**). This result was agree with our result in Table (9) the insulin receptor substrate -2 mediated insulin signaling pathway in the insulin – resistance states, and subsequent simulation of de novo lipogenesis in the liver leads to increased intracellular availability of triglycerides ,promoting fatty liver .This also increases VLDL assembly and secretion (**Lewis, et al., 2002**). Cholesterol ester protein – mediated transfer of TG from VLDL to LDL contributes to the formation of small dense LDL particles (**Berneis and Krauss, 2002**). Also a lot of studied concluded that Vit. D3 might be useful especially toxicity by significantly TG, carbohydrate in diabetic rats. Moreover plasmatic non-enzymatic antioxidant level of HDL- cholesterol increased after VD administration (**Hamden, et al ., 2009**).

**Table (8): CBC "complete blood count" (White Blood Cells, Lymphocytes, Red Blood Cells) in serum for different groups.**

Groups	WBC (10 <sup>3</sup> ) ul		LY(10 <sup>3</sup> )ul		RBC (million/ul)	
	Before	After	Before	After	Before	After
	m±S.D	m±S.D	m±S.D	mean±S.D	mean±S.D	mean±S.D
1	7.37±2.07 <sup>a</sup>	6.98±2.44 <sup>a</sup>	2.55±0.71 <sup>a</sup>	2.37±0.06 <sup>a</sup>	4.56±0.32 <sup>a</sup>	4.34±0.34 <sup>a</sup>
2	6.47±2.16 <sup>a</sup>	7.20±1.84 <sup>a</sup>	2.33±0.79 <sup>a</sup>	2.30±0.44 <sup>a</sup>	4.63±0.54 <sup>a</sup>	3.98±0.20 <sup>b</sup>
3	6.19±1.83 <sup>a</sup>	2.32±0.90 <sup>b</sup>	2.36±0.79 <sup>a</sup>	1.48±0.20 <sup>b</sup>	4.41±0.48 <sup>a</sup>	4.49±0.32 <sup>a</sup>
4	6.22±1.49 <sup>a</sup>	5.9±1.42 <sup>a</sup>	2.18±0.56 <sup>a</sup>	2.07±0.67 <sup>a</sup>	4.53±0.45 <sup>a</sup>	4.38±2.26 <sup>a</sup>

- ✓ **Group 1: Diabetes female with vitamin D deficiency and injected with Devarol .**
- ✓ **Group2: Diabetes female with vitamin D deficiency and injected with Devarol + Parsley(100g /per day)**
- ✓ **Group3: non-diabetic female with Vitamin D deficiency and injected with Devarol**
- ✓ **Group4: non-diabetic female with Vitamin D deficiency and injected with Devarol + Parsley(100g /per day)**
- ✓ **Reference range :WBC(4-10) (10<sup>3</sup>) ul .**
- ✓ **LY(1-3) (10<sup>3</sup>) ul .**
- ✓ **RBC(3.8-4.8)(Million/ul).**

Results in table (8) contained the values of WBC in which were 7.37±2.07, 6.47±2.16, 6.19±1.83, 6.22±1.49 , 6.98±2.44, 7.20±1.84, 2.32±0.90 and 5.9±1.42 for all groups 1, 2, 3 and 4 before and after treatments respectively. From the above results we can be noticed that there were no statistical significant differences (P>0.05) between all groups before and after treatment except group 3 after treatment which statistical significantly lower value in WBC and LY. As meanwhile, the values of serum lymphocyte were 2.55±0.71, 2.33±0.79, 2.37±0.06 and 2.30±0.44 (10<sup>3</sup>) ul for groups 1, 2 before and after treatment. Moreover, results concerning RBC in Table (8) showed that there were no statistical significant differences (P>0.05) between all groups 1, 2, 3 and 4 before treatment and

groups 3 and 4 after treatment. However, groups 3 and 4 after treatments were lower statistical significantly ( $P \leq 0.05$ ) among all groups under study.

**Table (9): Means of Hemoglobin(Hb), Hematocrit(HCT), Red blood width(RDW) for different groups.**

Groups	Hb (g/dl)		HCT (%)		RDW (%)	
	Before	After	Before	After	Before	After
	m±S.D	m±S.D	m±S.D	m±S.D	m±S.D	m±S.D
1	12.35±1.03 <sup>a</sup>	12.06±1.03 <sup>b</sup>	36.84±2.67 <sup>ab</sup>	35.48±2.68 <sup>b</sup>	15.41±0.86 <sup>a</sup>	15.75±0.61 <sup>a</sup>
2	12.84±1.73 <sup>a</sup>	12.92±0.61 <sup>a</sup>	38.32±4.65 <sup>a</sup>	38.22±1.35 <sup>a</sup>	15.38±0.80 <sup>a</sup>	14.49±0.28 <sup>b</sup>
3	12.06±1.5 <sup>a</sup>	11.97±1.07 <sup>b</sup>	35.89±3.82 <sup>b</sup>	34.39±2.45 <sup>c</sup>	15.42±0.72 <sup>a</sup>	15.73±0.97 <sup>a</sup>
4	12.24±1.46 <sup>a</sup>	12.85±1.27 <sup>a</sup>	36.43±3.93 <sup>ab</sup>	39.09±1.67 <sup>a</sup>	15.42±0.66 <sup>a</sup>	14.69±0.67 <sup>b</sup>

- ✓ **Group 1: Diabetes female with vitamin D deficiency and injected with Devarol .**
- ✓ **Group2: Diabetes female with vitamin D deficiency and injected with Devarol + Parsley(100g /per day)**
- ✓ **Group3: non-diabetic female with Vitamin D deficiency and injected with Devarol**
- ✓ **Group4: non-diabetic female with Vitamin D deficiency and injected with Devarol + Parsley(100g /per day)**
- ✓ **Reference range :Hb(12-15)(g/dl).**
- ✓ **HCT(36-47)%.**
- ✓ **RDW(11.5-14.5)%.**

Data in Table (9) contained the results of Hb the values of mean±S.D were 12.35±1.03, 12.84±1.73, 12.06±1.03, 12.92±0.61, 12.06±1.5, 12.24±1.46, 11.97±1.07, 12.85±1.27g/dl for groups 1, 2 diabetic patient before and after treatment and groups 3, 4 non diabetic patient before and after treatment respectively. From the previous results we can be noticed that there were no statistical significant differences ( $P > 0.05$ ) between groups 1, 2 before and after treatment except of group 1 after injected by vitamin D ampule which lower statistical significantly than before treatment. On the other hand, there were significant differences ( $P \leq 0.05$ ) between

groups 3 and 4 non diabetic patient after treatment Hb and HCT. Concerning HCT results in Table (9) showed that there were statistical significant differences ( $P \leq 0.05$ ) between group 1 after treatment and group 2 before and after treatment vs. group 3 after treatment and group 4 before and after treatment. Moreover, groups 1 and 3 after treatment were the lowest values  $35.48 \pm 2.68$  vs.  $34.39 \pm 2.45\%$  respectively. Concerning RDW results in the same table showed that group 4 after treatment of non-diabetic patient was lowest statistical significantly ( $P \leq 0.05$ ) among all groups  $14.49 \pm 0.28\%$ .

**Table (10): Means of Corpuscular Volume(MCV), Mean Corpuscular Hemoglobin(MCH), Mean Corpuscular Hemoglobin Concentration(MCHC) and Platelet in Serum of Groups.**

Groups	MCV (FL)		MCH (Pg)		MCHC (g/dl)		PLT ( $10^9$ /ul)	
	Before	After	Before	After	Before	After	Before	After
	m±S.D	m±S.D	m±S.D	m±S.D	m±S.D	m±S.D	M±S.D	M±S.D
1	$81.11 \pm 7.55^a$	$82.06 \pm 5.23^b$	$27.13 \pm 2.96^a$	$27.82 \pm 2.61^a$	$33.47 \pm 1.005^a$	$33.94 \pm 1.27^a$	$245.0 \pm 1251^c$	$243.16 \pm 4.96^b$
2	$82.06 \pm 7.5^a$	$85.51 \pm 1.95^a$	$27.68 \pm 2.74^a$	$27.47 \pm 2.86^a$	$33.40 \pm 0.85^a$	$33.32 \pm 0.45^b$	$273.08 \pm 13.17^a$	$263.48 \pm 6.90^a$
3	$81.76 \pm 8.37^a$	$79.37 \pm 6.86^c$	$27.28 \pm 3.36^a$	$25.21 \pm 3.05^a$	$33.48 \pm 1.05^a$	$32.82 \pm 0.86^b$	$266.28 \pm 16.18^{ab}$	$246.59 \pm 5.48^b$
4	$80.58 \pm 5.73^a$	$83.88 \pm 3.86^a$	$26.83 \pm 2.71^a$	$27.01 \pm 2.77^a$	$33.51 \pm 1.05^a$	$33.42 \pm 0.29^b$	$263.5 \pm 17.45^b$	$244.46 \pm 8.66^b$

- ✓ **Group 1: Diabetes female with vitamin D deficiency and injected with Devarol .**
- ✓ **Group2: Diabetes female with vitamin D deficiency and injected with Devarol + Parsley(100g /per day)**
- ✓ **Group3: non-diabetic female with Vitamin D deficiency and injected with Devarol**
- ✓ **Group4: non-diabetic female with Vitamin D deficiency and injected with Devarol + Parsley(100g /per day)**
- ✓ **Reference range :MCV(83-101)FL.**
- ✓ **MCH(27-32)Pg.**
- ✓ **MCHC(31.5-34.5)g/dl.**
- ✓ **PLT(150-450)  $10^9$ /ul.**

In addition results in Table (10) for MCV the values were  $81.11 \pm 7.55$ ,  $82.06 \pm 7.5$ ,  $82.06 \pm 5.23$ ,  $85.51 \pm 1.95$ (fl) for groups 1, 2 diabetic patient before and after treatment

respectively. From above results we can be observed that there were no statistical significant differences ( $P>0.05$ ) between groups 1 and 2 before treatment, while there was statistical significant difference ( $P\leq 0.05$ ) in group 2 between before and after treatment. Also results in the same table found there was no statistical significant difference ( $P>0.05$ ) between groups 3 and 4 before treatment. While, there was statistical significant difference ( $P\leq 0.05$ ) between groups 3 and 4 in MCV after treatment. Concerning MCH we can noticed that there we no significant differences ( $P>0.05$ ) among all groups except group 3 after treatment which lower statistical significantly ( $P\leq 0.05$ ). Moreover, results concerning MCHC showed that there were statistical significant differences ( $P\leq 0.05$ ) between groups 1, 2 after treatment with values,  $\pm$  S.D were  $33.94\pm 1.27$  and  $33.32\pm 0.45$ pg, respectively. Also results in the same table in normal groups showed that there were statistical significant differences ( $P\leq 0.05$ ) between groups 3 and 4 after treatment with values  $32.82\pm 0.86$ ,  $33.42\pm 0.29$ pg, respectively on the other hand there were no statistical significant differences ( $P>0.05$ ) between other groups under study.

The values of mean  $\pm$  S.D for PLT were  $245.0\pm 13$ ,  $273.08\pm 13.17$ ,  $243.16\pm 4.96$  and  $263.48\pm 6.90$  for groups 1, 2 before and after treatment g/dl, respectively. From the previous results, there were statistically significant differences ( $P\leq 0.05$ ) between diabetic groups, besides that, there were statistically significant differences ( $P\leq 0.05$ ) between normal groups before and after treatment values were  $266.28\pm 16.18$ ,  $263.5\pm 17.45$ VS.  $246.59\pm 5.48$ ,  $244.46\pm 8.66$  respectively. While, group 1 after treatment was the lowest value statistical significantly. The cells in the CBC (white blood cells, red blood cells, and platelets) have unique functions. Generally, white blood cells are an essential part of the immune system and help the body fight infections. Each different component of the white blood cell (the WBC differential) plays a specific role in the immune system. Red blood cells are essential in transporting oxygen to all the cells in the body to serve their

functions. The hemoglobin molecule in the red blood cell is the vehicle for the transportation of oxygen. Platelets are a part of the blood clotting system in the body and help in preventing bleeding (**John and Sara., 2021**). It can also be done as a part of an evaluation based on a patient's symptoms. A high WBC count (leukocytosis) may signify an infection somewhere in the body or, less commonly, it may signify an underlying malignancy. A low WBC count (leukopenia) may point toward a bone marrow problem or related to some medications, such as chemotherapy. A doctor may order the test to follow the WBC count in order to monitor the response to a treatment for an infection. The components in the differential of the WBC count also have specific functions and if altered, they may provide clues for particular conditions. A low red blood cell count or low hemoglobin may suggest anemia, which can have many causes. Possible causes of high red blood cell count or hemoglobin (erythrocytosis) may include bone marrow disease or low blood oxygen levels (hypoxia) a low platelet count (thrombocytopenia) may be the cause of prolonged bleeding or other medical conditions that affect the production of platelets in the bone marrow. Conversely, a high platelet count (thrombocytosis) may point toward a bone marrow problem or severe inflammation (**John and Sara., 2021**).

### **Conclusion**

The study concluded that there is a relationship between the level of vitamin D and improve the level of blood sugar in patients with diabetes type II. In addition, the injection of vitamin D is absorbed faster than the digestion through the digestive system, which explains the effect of the injection with the intake of parsley. Also, eating rich vitamin D-containing food with fresh parsley gave the best results to type 2 diabetics and vitamin D deficiency patients. Finally, HbA1C was improved due to insulin resistance, because most of the research shows the work of vitamin D as a hormone, which improves the entry of glucose into cells and improves the function of insulin receptors in cells.



**Recommendations**

The study recommend that eating food rich in vitamin D content and use them, especially fresh parsley on a daily basis and is an accessible source for all its availability and cheap price and availability throughout the year. Also preserving the level of vitamin D in its normal rate helps diabetic patient to decrease the rate blood sugar and reduce its complications of diabetes and improve the absorption of calcium and improve the fatigue and stress of diabetic .

**References**

- ADA :American Diabetes Association (2010):** Diagnosis and classification of *Diabetes Mellitus*. *Diabetes Care* 33 Suppl. 1: S62-69.
- Ahmed, A.M. (2002):** History of *Diabetes Mellitus*. *Saudi Med. J.* 23: 373-378.
- Alemzadeh R, Kichler J, Babar G, Calhoun M. (2008):** Hypovitaminosis D in obese children and adolescents: relationship with adiposity, insulin sensitivity, ethnicity, and season. *Metabolism.*57:183–191.
- Allain, C. C. (1974):** Cholesterol enzymatic colorimetric method. *J. of Clin. Chem.*, (20):470.
- American Diabetes Association.(2014);** Standards of medical care in diabetes, *Diabetes Care*;37(Suppl. 1):S14 - S80.
- Andrews RC, Cooper AR, Montgomery AA ,(2011):** Diet or diet plus physical activity versus usual care in patients with newly diagnosed type 2 diabetes: the Early ACTID randomized controlled trial. *Lancet* 129:139-378.
- AOAC (2010)** Official Methods of Analysis of Association of Official Analytical Chemists. 18th Edition, Washington, DC.
- Bauer, Henry (2018).** Essay Review. Fake Medical News Doctoring Data: How To Sort Out Medical Advice from Medical Nonsense by Malcolm Kendrick. *Journal of Scientific Exploration.* 32. 764-772. 10.31275/2018/1377.
- Berneis KK and Krauss RM.(2002):** Metabolic origins and clinical significant of LDL heterogeneity . *J Lipid Res.*,43:1363- 1379.
- Chiu, K. C., Chu A., Go V. L. W., and Saad M. F. (2004):** Hypo vitaminosis D is associated with insulin resistance and  $\beta$  cell dysfunction,” *The American Journal of Clinical Nutrition*, 79, ( 5): 820–825.

- Chris G. Velentzas ,(2012):** The Hydroxylation of Vitamin D on C25 in Thyrotoxicosis: The Role of the Activity of Microsomal Liver Enzymes. *International Journal of Clinical Medicine* Vol.3 No.4(2012), Article ID:20872,5 pages DOI:10.4236/ijcm.2012.34057.
- Christensen E. I., Devuyst O., Dom G. (2003):** Loss of chloride channel ClC-5 impairs endocytosis by defective trafficking of megalin and cubilin in kidney proximal tubules,” *Proceedings of the National Academy of Sciences of the United States of America*. 100 ( 14): 8472–8477.
- Coppell KJ, Kataoka M, Williams SM, Chisholm AW, Vorgers SM, Mann JI, (2010):** Nutritional intervention in patients with type 2 diabetes who are hyperglycaemic despite optimised drug treatment Lifestyle Over and Above Drugs in Diabetes (LOADD) study: randomised controlled trial. *BMJ*;341:c3337.
- Deleskog A., Hilding A., Brismar K., Hamsten A., Efendic S., and Ostenson C. G.,(2012):** Low serum 25-hydroxyvitamin D level predicts progression to type 2 diabetes in individuals with prediabetes but not with normal glucose tolerance. *Diabetologia*. ( 55): 1668–1678.
- DRI, Dietary Reference Intakes (2002):** for Energy, Carbohydrate, Fiber, Fat, Fatty Acids, Cholesterol, Protein, and Amino Acids.
- Dusso A. S., Brown A. J., and Slatopolsky E. (2005):** Vitamin D. *American Journal of Physiology*. 289( 1): 8–28.
- Fieser, L.F. and Fieser, M. (1959):** Vitamin D. In: *Steroids*. 1st ed. New York: Reinhold Publishing Corporation; USA. p. 90-168.
- Forouhi, N. G.;Ye Z.and Rickard A. P. (2012):** Circulating 25- hydroxyvitamin D concentration and the risk of type 2 diabetes: results from the European Prospective

- Investigation into Cancer (EPIC)-Norfolk cohort and updated metaanalysis of prospective studies. *Diabetologia*, 55, ( 8): 2173–2182.
- Fossati, P. and Prencipe, L. (1982)** Serum Triglycerides Determined Colorimetrically with an Enzyme that Produces Hydrogen Peroxide. *Clinical Chemistry*, 28, 2077-2080.
- Friedewald WT, Levy RI, Fredrickson DS.(1972):** Estimation of the concentration of low-density lipoprotein cholesterol in plasma, without use of the preparative ultracentrifuge. *Clin Chem.* 1972 Jun;18(6):499-502. PMID: 4337382.
- Gagnon, C; Lu, ZX and Magliano , DJ.(2011):** Serum 25-hydroxyvitamin D, calcium intake, and risk of type 2 diabetes after 5 years: results from a national population-based prospective study (the Australian Diabetes, Obesity and Lifestyle study). *Diabetes Care*;34:1133–1138.
- Hamden K , Carreau S , Jamoussi K , Miladi S, Lajmi S, Aloulou D, Ayadi F and El Feki A .(2009):** 1 $\alpha$ 25 Dihydroxyvitamin D3:Therapeutic and preventive Effects against oxidative Stress , Hepatic , pancreatic and renal injury in Alloxan Induced diabetic in rates , *J. of Nutritional Science and Vitaminology* .55(3):215-222.
- Holick M.F. and Chen T. C.,(2008):** Vitamin D deficiency: a worldwide problem with health consequences. *The American Journal of Clinical Nutrition*. 87( 4): 1080S–1086S.
- John H. Fountain; Sarah L. Lappin.(2021):** Physiology, Platelet(book shelf) . Treasure Island (FL): StatPearls Publishing; 2022 Jan.
- Kayaniyil, S; Vieth, R. and Retnakaran R. (2010):** Association of vitamin D with insulin resistance and  $\beta$ -cell dysfunction in subjects at risk for type 2 diabetes. *Diabetes Care*;33:1379–1381.

- Knekt P, Laaksonen M, Mattila C, Harkanen T, Marniemi J, Heliovaara M, et al.** Serum vitamin D and subsequent occurrence of type 2 diabetes. *Epidemiology* 2008; 19:666–671.
- Krauss RM , and Siri PW .(2004):** Dyslipidemia in type 2 Diabetes" , *J. Med Clin Nut Am .*, 88:897-909.
- Lewis GF , Carpenter A, Khosrow A and Giacca A . (2002):** Disordered fat storage and mobilization in the pathogenesis of insulin resistance and type 2 diabetes. *J.Endocr Rev .*, 23:201-229.
- Lindquist CH ,Cummings T, Goran MI , (2000):** Use of tap recorded food records in assessing children's dietary intake Department of nutrition science, school of health Related procession . University of Alabama at Birmingham ,USA . Jan ; 8(1):2-11.
- Maestro B., Campi'on J., D'ávila N., and Calle C. (2003):** Stimulation by 1,25-dihydroxyvitamin D3 of insulin receptor expression and insulin responsiveness for glucose transport in U-937 Journal of Biomedicine and Biotechnology 11 humanpromonocytic cells.*Endocrine Journal.* 47, (4): 383–391.
- Malmstrom R, Packard CJ,Caslake M, Bedford D , Stewart P , Yki- Järvinen H,Shepherd J and Taskinen MR.(1997):**Defective regulation of triglyceride metabolism by insulin in the liver in NIDDM. *J . Diabetologia .* 40:454-462.
- Mattila C, Knekt P, Männistö S, Rissanen H, Laaksonen MA, Montonen J, Reunanen A:** Serum 25-hydroxyvitamin D concentration and subsequent risk of type 2 diabetes. *Diabetes Care.* 2007 Oct;30(10):2569-70. doi: 10.2337/dc07-0292. Epub 2007 Jul 12. PMID: 17626891.
- Metzger LE, Nielsen SS.(2017):** nutrition labeling .In: Nielsen SS editor. *Food analysis .5 th ed.* Indiana : springer. 35-43

- Milner, K.L.; Van Der Poorten, D , Trenell ,M ; Jenkins ,A.B ;Xu ,A ; Smythe ,G ; Dore , G.J. and Zekry, A.(2010):** Chronic hepatic C is associated with peripheral rather than hepatic insulin resistance.J. Gastroenterology.138(3):932-41.
- Moayeri, H., Bidad, K., Aghamohammadi, A., Rabbani, A., Anari, S., Nazemi, L., et al. (2006):** Overweight and Obesity and Their Associated Factors in Adolescence in Tehran, Iran, 2004-2005. European Journal of Pediatrics, 165, 489-493.  
<http://dx.doi.org/10.1007/s00431-006-0101-8>
- National Center for Agricultural Research,(2017):** The analysis was carried out in the Laboratory at the Institute of Food Technology on HPLC Fraction ,Application 954 pp.257
- NNI (2006):** National Nutrition Institute Food Composition Tables, Cairo, A.R.E., 2006.
- P. Armitage and G. Berry, Blackwell Scientific :**Statistical methods in medical research Publications, Oxford, U.K., 1987. No. of pages: 559.
- Park, J.E. and Park, K. (1979):** Textbook of preventive social medicine, 7th ed Hesers Banner Sides, Phnat publisher, 1268 Napier town, p.81.
- Perron M and Endres J, (1985):** Knowledge, attitudes and dietary practices of female athletes. American Diabetic Association May . 85 (5):573-6.
- Popović, M.; Kaurinović, B. and Jakovljević, V. (2007):** Effect of parsley (*Petroselinum crispum* (Mill.) Nym. ex A.W. Hill, Apiaceae) extracts on some biochemical parameters of oxidative stress in mice treated with CCl(4). Phytother Res. 21(8): 717-723.
- Roglic, G. (2005):** The burden of mortality attributable to diabetes. Diabetes Care, 28: 2130

- Salamoun MM , Kizrian AS ,Tannous RI , Nabulsi MM,Choucair MK , (2005):**"Low calcium and vitamin D intake in healthy children and adolescent and their correlates . calcium Metabolism and Osteoporosis program ,Department of Internal Medicine , American University of Beirut , Lebanon . Feb ; 59(2) : 177 – 84.
- Schermer, S. (1967). The blood morphology of laboratory animals. Philadelphia: Davis**
- Selvin, E.; Steffes, M.W.; Zhu, H.; Matsushita, K. and Wagenknecht, L. (2010):** Glycated hemoglobin, diabetes, and cardiovascular risk in non diabetic adults. N. Engl .J. Med. 362: 800.
- Shaw ,J.E. Sicree, R.A. Zimmet, P.Z: Global estimates of the prevalence of diabetes for 2010 and 2030. Diabetes Research and Clinical Practice Volume 87, Issue 1, January 2010, Pages 4-14**
- Singh, K., Sundarro, K., Tinkerame, J., Kaluwin, C. and Matsuoka, T. (1991):** Lipid content fatty acid and mineral composition of Mud Crabs (*Seylla serrata*) from Papua new Guinea. Journal of Food Composition and Analysis, 4 (3): 276 - 280.
- Soysal, Y. (2004):** Microwave drying characteristics of parsely, Bio.Eng. 89, 167-73.
- Tate, D.; Jeffery, R. and Sherwood, N. (2007):** Long-term weight losses associated with prescription of higher physical activity goals. Are higher levels of physical activity protective against weight regain. Am. J. Clin. Nutr., 85 (4): 954-9.
- Temple R .(2003):** Impact of the Declaration of Helsinki on medical research from a regulatory perspective. Address to the Scientific Session, World Medical Association General Assembly, September.
- Tuorkey MJ, Abdul-Aziz KK:** Strategies for diabetes and pathways of vitamin D. Diabetes and Metabolic Syn-drome: Clinical Research and Reviews 2010;4(2):101-110.

- USDA ,U.S. Department of Health and Human Services and U.S. Department of Agriculture. Dietary Guidelines for Americans, (2010):**[Internet]. Available from [www.health.gov/dietaryguidelines](http://www.health.gov/dietaryguidelines) Accessed 30 June 2011.
- Weiss, E.; Galuska, D. and Kettel-Khan, L. (2007):** Weight regain in U.S. adults who experienced substantial weight loss, 1999-2002". July; Am. J. Prev. Med., 33 (1): 34-40.
- WHO (2014a):** "BMI classification". World Health Organization. Retrieved 15 February 2014.
- WHO (2014b):** Obesity and overweight. World Health Organization, Fact sheet N°311, Updated August 2014 .
- Wu, T; Willett, WC. and Giovannucci, E.(2009):** Plasma C-peptide is inversely associated with calcium intake in women and with plasma 25-hydroxy vitamin D in men. J Nutr;139:547–554 .
- Yanardag, R.; Bolkent, S.; Tabakoglu-Oguz, A. and Ozsoy-SacanO(2003):** Effects of Petroselinum crispum extract on pancreatic B cells and blood glucose of streptozotocin-induced diabetic rats. Biol Pharm Bull,26: 1206
- Yang, W.; Lu, J.; Weng, J.; Jia, W.; Ji, L. and Xiao, J. (2010):** Prevalence of diabetes among men and women in China. New.



## تأثير المأخوذ من فيتامين د حقنا ومنتجات طبيعية على مستوى جلوكوز الدم لدى مرضى السكر المصابين بنقص فيتامين د

### الملخص العربي

يعتبر مرض البول السكري هو رابع سبب رئيسي للوفيات في العالم. بحلول عام ٢٠٢٥ ، من المتوقع أن يزداد معدل الإصابة بالمرض إلى أكثر من ٣٨٠ مليوناً ، ٨٠ ٪ سيكون في البلدان النامية. لقد أثبتت الدراسات السابقة أن نقص فيتامين (د) يلعب دوراً مهماً في مقاومة الأنسولين مما يؤدي إلى الإصابة بمرض السكري. أجريت الدراسة الحالية لتقييم تأثير استخدام بعض الأطعمة التي تحتوي على نسبة عالية من فيتامين د وخاصة (البقدونس) ومصدر دوائي من نفس الفيتامين على مرضى السكر والأشخاص الصحيين. تم اختيار العينة عن طريق تحليل فيتامين د في الدم واختيار المرضى الذين يعانون من نقص فيتامين د. ١٠٠ شخص يعانون من مرض السكري من النوع ٢ و ٥٠ غير مصابين بالسكري وكلهم يعانون من نقص فيتامين (د). تم تقسيم كل مجموعة إلى المجموعة الأولى مرضى السكري ونقص فيتامين د ويتم علاجهم بحقن فيتامين د فقط. المجموعة الثانية هي مرضى السكر ونقص فيتامين د ويتم علاجهم بفيتامين د بالإضافة إلى حقن البقدونس. المجموعة ٣ هي مرضى غير مصابين بالسكري يعانون من نقص فيتامين د ويتم حقن فيتامين د فقط. المجموعة ٤ هي مرضى غير مصابين بالسكري يعانون من نقص فيتامين د ويحتاجون إلى حقن فيتامين د بالإضافة إلى البقدونس. استمر العلاج لمدة ٣ أشهر. ووجدت النتائج أنه بعد علاج المجموعتين ٢ و ٤ حيث تم تناول ١٠٠ جم من البقدونس الطازج مع أمبولات من الفيتامين. كانت قيمة D أعلى معنوياً ( $P \leq 0.05$ ) لفيتامين المصل. د. مستوى الكالسيوم من المجموعة ٤ غير السكري يحقن بفيتامين. كانت D و ١٠٠ جم من البقدونس الطازج أعلى قيمة إحصائية معنوية ( $P \leq 0.05$ ). كان الهيموجلوبين السكري ذو دلالة إحصائية عالية ( $P \leq 0.05$ ) في مرضى السكري وغير المصابين بالسكري قبل العلاج. بينما بعد حقنها بالفيتامين د للمجموعات (١ و ٣) أظهرت نتائج D plus 100g البقدونس الطازج للمجموعتين ٢ و ٤ انخفاضاً في الهيموجلوبين السكري في مصل الدم لجميع المجموعات. وخلصت الدراسة إلى وجود علاقة بين مستوى فيتامين (د) وتحسين مستوى السكر في الدم لدى مرضى السكري من النوع الثاني. أعطى تناول الأطعمة الغنية بفيتامين د مع البقدونس الطازج أفضل النتائج لمرضى السكر من النوع الثاني و نقص فيتامين د.