

Investigation the Development of Metabolic Syndrome for Type 2 Female Diabetic through Serpin E1, Chemoattractant Protein-1 and Adiponectin

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Abstract

In this study, 54 female patients with type 2 diabetes and 36 non-diabetics took part, with obese and non-obesity persons compared to an apparently healthy control group. The serpin E1 and monocyte chemoattractant protein-1 were measured by ELISA sandwich while the colorimetric methods were used for calculating HbA1C, serpin E1 (serine proteinase inhibitors) and lipid peroxidation. The levels of serpin E1 (PAI-1) and monocyte chemo attractant protein -1 in the blood of non-obese women differed significantly from those of obese individuals ($p=0.05$). Similarly, there was a significant difference between the receptor levels of no obese female patients and serum controls from obese females ($p=0.05$). The values of serpin E1, monocyte chemoattractant protein -1, HbA1C were increased in patients with type 2 diabetes who have the highest TG, TC, vLDL, LDL and also decreased for HDL and adiponectin in obese females. In this study, parameter was compared with adiponectin under review.

Keywords: Type 2 diabetes mellitus; Obesity; Serpin E1; Monocyte Chemoattractant Protein-1(MCP); HbA1C; Lipid peroxidation

Introduction

Research into the role of the C-C chemokine Monocyte Chemoattractant Protein 1 (MCP-1) plays in the development of metabolic syndrome and type 2 diabetes has skyrocketed during the past two decades. The purpose of this article is to present a synopsis of this chemokine, including its development, regulatory mechanisms, functions, and therapeutic approaches. This research emphasizes the role of MCP-1 in the onset of obesity, diabetes, cardiovascular disease, insulinitis, diabetic nephropathy, and diabetic retinopathy. In white adipose tissue, simultaneous ablation of AdipoR1 and R2 resulted in elevated MCP-1 expression and the macrophage marker MAC-1 (WAT). Reduced adiponectin signaling could be an upstream mechanism for increased MCP-1 production and inflammation in WAT. Obesity, a chronic condition characterized by adipose tissue growth and inflammatory components, has been associated to the development of a number of metabolic illnesses. Insulin

resistance and metabolic syndrome are more frequent in people who have abdominal obesity and excess Visceral Adipose Tissue (VAT). VAT plays a critical role in obese inflammation. These abnormalities increase before the development of T2D and are strongly linked to NAFLD (Nonalcoholic Fatty Liver Disease). Obesity, Cardio Vascular Disease (CVD), and atherosclerosis are all linked to high cholesterol levels. Chemokines mediate immunological responses to these circumstances, resulting to an innate immune response characterized by the generation of cytokines. HbA1c measures eight to twelve weeks of mean plasma glucose. It may be done anytime and does not need fasting. Due to its features, it's the key test for glycemic control in diabetics. Newly, there has been a lot of interest in employing it as a diabetes analytical test and a diabetes showing test for high risk persons. The danger of developing prediabetes and type 2 diabetes over a 10 years follow-up period in a community based sample was similarly found to be associated with higher PAI-1 and lower adiponectin levels.

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Recent research has connected PAI-1 to hyperlipidemia and revealed a role for PAI-1 in lipid metabolism. Further, pharmaceutical PAI-1 inhibition protects against hepatic steatosis, dyslipidemia, and reduces total chop. Increased adipose tissue in obese people causes hypoxia and the initiation of inflammasomes, which in turn stimulates the synthesis of adipocytes and the initiation of monocytes, resultant in chronic low grade inflammation. In addition, low grade inflammation contributes to the development of atherosclerosis *via* processes such as changes in monocyte activity. In individuals with metabolic illnesses like T2D, monocytes production a crucial role in initiating endothelial and vascular dysfunction, which are major predictors of thrombosis and death. As a result, chronic immunological activation is being addressed in an attempt to decrease CVD risk in people with metabolic illnesses by inhibiting the production of cellular mediators of inflammation and thrombosis, such as monocytes. MCP-1 activation may cause type 2 diabetes in obese patients. Increased adhesion molecules and chemokines in the endothelium may accompany pathogenic changes, activating and attracting circulating monocytes into sub endothelial space where they develop into macrophages [1-5] (Figure 1).

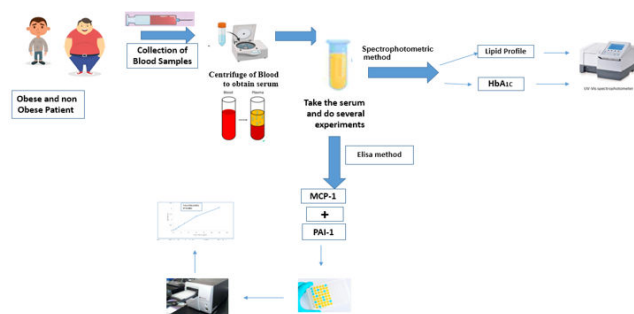


Figure 1: The study, parameter were compared with adiponectin under review.

This process promotes atherosclerosis and subsequent CVDs. Fructose ingestion exacerbated the already elevated levels of MCP-1 in morbidly obese people. To add insult to injury, MCP-1 signaling has a direct function in the expansion of obesity. Due to its antigenic action on endothelial cells, MCP-1 may aid in the development and reshaping of fat tissues. Obesity related disorders, including insulin resistance and type 2 diabetes, can be improved by reducing MCP-1 overproduction, which must be done reduced proportion converses activity is associated with increased cholesterol levels [6,7].

Materials and Methods

Method: In this study, participants were separated into two groups: diabetics and non-diabetics, the patients were separated into two groups (obese and non-obese). Also the

control groups divided into two matching with patients group, in the study divided into two groups (Figure 2).

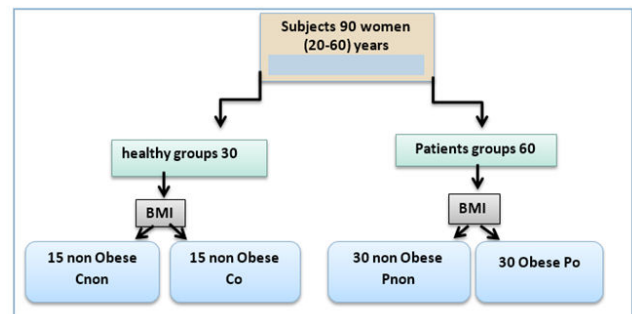


Figure 2: Subjection of 90 women ages from (20-60).

The investigation was carried out in the endocrinology center of Al-Murjan Al-Talafi Hospital in Al-Hilla, Babylon, Iraq, for a case control study. The following are patient and healthy person lists: type 2 diabetes patients (ages 26 to 60) who came to the diabetes research center Individuals and sick were both subjected to interrogators and legislation. The investigation was completed. To collect, use parameters such as pathological sex and. Background and weights According to the guidelines, (BMI, kg/m^2) was calculated as average weight (23.11-26.69) (kg/m^2) or obesity ($>30 \text{ kg}/\text{m}^2$).

Blood sampling

Patients and observers had 5 ml blood taken. Before being centrifuged at 4500 rpm for 10 minutes, blood samples might clot. The path was split and buried. Hold till analysis is done.

Statistical analysis

The data was analyzed using SPSS Statistics. The findings were analyzed using means, standard deviations, and ANOVA to determine the difference between groups. Students' assessments were compared to two average semesters. Pearson's correlation coefficients were used to compare the relationships between variables. P-values the test had two ends, and a P-value of 0.05 was thought to be significant.

Results and Discussion

Clinical properties

Biochemical and clinical factors experienced by the study's participants are listed in Tables 1 and 2. Table 1 shows the clinical characteristics of DM2 patients and healthy controls. The age difference between the two groups was not statistically significant ($p>0.05$), and while FBG levels were higher in the patients group than in the controls group, the BMI difference was not statistically significant ($p>0.05$).

Table 1: Clinical properties for DM2 patients and controls.

Parameters	Patients	Control	P-value
Age (years)	48.80 $\pm$ 13.13	52.48 $\pm$ 13.41	0.011

BMI (kg/m ²)	29.46 ± 5.05	27.53 ± 6.05	0.149
HbA1C	9.34 ± 1.27	5.17 ± 0.79	0.00001
T.C (mg/dL)	266.71 ± 62.13	130.07 ± 30.08	0.00001
T.G (mg/dl)	253.52 ± 100.64	118.35 ± 23.66	0.00001
HDL-C (mg/dL)	44.23 ± 7.13	50.98 ± 9.16	0.00001
LDL (mg/dL)	171.77 ± 55.94	55.41 ± 32.81	0.00001
VLDL (mg/dL)	50.70 ± 20.12	23.67 ± 4.73	0.00001
Duration of disease (years)	5.39 ± 2.69	-	
Duration of medication (years)	4.83 ± 2.17	-	
MCP-1	214.60 ± 141.91	85.58 ± 15.10	0.00001
PAI-1	5.14 ± 2.53	2.11 ± 1.55	0.00001

The mean difference is Significant at p ≤ 0.05.

Measurement of HbA1C and BMI

Table 2 shows the results of a comparison of HbA1C and body mass index between patient and control groups. Obese patients have high HbA1C (9.44 ± 1.38), with BMI (33.543.31 ±) compared to non-obese patients HbA1C (9.23

± 1.16), with BMI (25.37 ± 2.53), according to criteria compared to obese control HbA1C (4.93 ± 0.75), with BMI (32.97 ± 2.99) compared another non obese control HbA1C (5.71 ± 0.78), with BMI (22.42 ± 2.82).

Table 2: Fasting serum glucose, HbA1C and BMI in the study subjects.

Parameter	N	Groups	Mean ± SD	CI 95%		Compared groups	P value	
				Lower	Upper			
BMI	30	Po	3.31 ± 33.54	32.3	34.78	Po	Pnon	0.00001
							Co	0.543
							Cnon	0.00001
	30	Pnon	2.53 ± 25.37	24.42	26.32	Pnon	Co	0.00001
							Cnon	0.002
HbA1C	15	Co	2.99 ± 32.9 7	31.32	34.63	Co	Cnon	0.00001
	15	Cnon	2.82 ± 22.42	20.85	23.98			
	30	Po	9.44 ± 1.38	8.93	9.96	Po	Pnon	0.47
							Co	0.00001
							Cnon	0.00001
	30	Pnon	9.23 ± 1.16	8.79	9.66	Pnon	Co	0.00001
							Cnon	0.00001
	15	Co	4.93 ± 0.75	4.51	5.35	Co	Cnon	0.253
	15	Cnon	5.41 ± 0.78	4.97	5.84			

Note: PO: Patient Obese; Pnon: Patient non obese; Co: Control obese; Cnon: Control non obese, The mean difference is significant at P≤0.05

Determination of monocyte chemoattractant protein 1 (pg/mL)

Table 3 shows that compared to both obese and non-obese individuals, MCP-1 levels are lower in obese patients. However, this difference is not statistically significant. When comparing the levels of Monocyte Chemoattractant Protein 1 (MCP-1) between groups, those with obesity had higher

levels than those without obesity ($p=0.0001$). Likewise, the obese patients contrasted favorably to the non-obese controls ($p0.001$). Although MCP-1 levels were much higher in the Pnon than in the no obese controls, the difference was not statistically significant ($p=0.253$). In addition to Pnon vs. Co ($p=0.846$).

Table 3: The difference between the ADIPOR1 patient groups and the control groups (ng/mL).

Parameter	N	Groups	Mean $\pm$ SD	CI 95%		Compared groups		P value
				Lower	Upper			
MCP-1 pg/ml.	30	Po	329.01 $\pm$ 117.52	285.13	372.9	Po	Pnon	0.00001
							Co	0.00001
							Cnon	0.00001
	30	Pnon	100.19 $\pm$ 8.97	96.84	103.54	Pnon	Co	0.846
							Cnon	0.253
	15	Co	95.97 $\pm$ 12.57	89.01	102.93	Co	Cnon	0.41
	15	Cnon	75.20 $\pm$ 9.12	70.15	80.25			

Note: Po: Patients obese; pnon: Patient non-obese; Co: Control obese; Cnon: Control non obese

MCP-1 levels have also been linked to atherosclerosis-related problems such as ischemic stroke, myocardial infarction and cardiovascular disease mortality. Obese people appear to have stronger correlation than people of normal weight. High plasma MCP-1 levels have been related with a poor prognosis in progressive heart failure and death after acute coronary syndromes. According to research by Piemonti L, et al., the vast majority of people who develop diabetes have type 2, which is related with excess body fat and IR. Research has shown that those with type 2 diabetes had significantly greater MCP-1 blood levels. Comorbidities associated with obesity are in part caused by blood monocytes participation in cytokine generation, migration into inflammatory adipose tissue, and vascular adhesion. Monocyte Chemoattractant Protein-1 (MCP-1) and other cytokines and chemokines. While there is growing evidence that adiponectin has anti-inflammatory effects, a few *in vitro* studies have shown otherwise. These studies were conducted in endothelial and monocytic cell lines, and while the precise kind of adiponectin employed and the stimulation settings

may make a difference, the findings revealed that adiponectin truly had pro-inflammatory activities. Studies have indicated that adiponectin acts as a local inflammatory regulator in adipose tissue. According to research individuals gain weight, monocyte numbers and activation levels rise, a condition known as diabetes. MCP-1 also has a role in the production of thrombin, the development of clots, the control of tissue factors, and the activity of (PAI-1) [8-14].

Determination of Plasminogen activation inhibitor-1 (ng/ml)

In Table 4 PAI-1 is no significantly reduced in patients obese associated to obese healthy, ($p>0.05$). A non-obese patients ($p>0.05$) respectively. Plasminogen activation inhibitor-1 (PAI-1) level comparison between different groups. PAI-1 was significantly increased in the patient role obese compared to patients no obese, ($p=0.00001$). While PAI-1 levels were no significant elevated in the Pnon compared to control obese, ($p=0.379$).

Table 4: PAI-1 (ng/mL) comparison between patients and healthy subjects.

Table 1: PAI-1 (ng/ml) comparison between patients and healthy subjects.								
Parameter	N	Groups	Mean ± SD	CI 95%		Compared groups	P value	
				Lower	Upper			
SREPE-1 (PAI-1) ng/ml	30	Po	6.25 ± 2.34	5.37	7.13	Po	Pnon	0.00001
							Co	0.00001
							Cnon	0.00001

30	Pnon	4.03 ± 2.23	3.2	4.86	Pnon	Co	0.379
						Cno	0.00001
15	Co	3.49 ± 0.94	2.97	4.01	Co	Cnon	0.00001
15	Cnon	0.72 ± 0.16	0.63	0.81			

Note: Po: Patients obese; pnon: Patient non-obese; Co: Control obese; Cnon: Control non obese

Leurs P, et al. found a metabolic syndrome and diabetes, plasminogen activator inhibitor. In individuals with type 2 diabetes, circulating PAI-1 levels are elevated, which contributes significantly to prothrombotic and proatherosclerotic alterations. Furthermore, plasma PAI-1 levels are elevated across the insulin resistance continuum, from the metabolic syndrome to prediabetes (a state of reduced glucose tolerance) to diabetes. Because both obese and non-obese animals had elevated plasma PAI-1 levels in these conditions, obesity appears to play a substantial role. PAI-1 was shown to be linked to CVD and diabetes by Lyon C and Hsueh W, et al. which they say is indicative of the prothrombotic and proinflammatory environment present in IR. Plasma PAI-1 is mostly determined by adipose mass and circulating insulin levels; losing weight and increasing exercise are therefore useful strategies for reducing PAI-1. Along with the current suggestions for antiplatelet medication. Important therapies for reducing prothrombotic and proinflammatory states in diabetes include Peroxisome Proliferator-Activated Receptor (PPAR) agonists/ligands and renin angiotensin system inhibitors, as demonstrated by research by Lyon and Koliaki C the part of these agents in prediabetes or the metabolic syndrome is not yet clear. In humans, the mature adipocyte is a major contributor to the blood circulation of PAI-1. When a person is overweight or

insulin resistant, their plasma levels of PAI-1 rise, and these elevated levels are positively connected with characteristics of the MS and are good predictors of future risk for cardiovascular disease and type 2 diabetes. Adiponectin and PAI-1 levels and their connection through different parameters in people with MS suggests that PAI-1 plays a role in the development of insulin resistance and obesity and may be a causative relationship between obesity and CVD (Alessi MC, Juhan-Vague I). Both PAI-1 and MCP-1 are preferentially produced from visceral adipose tissue in humans, and visceral adipose mass is a primary determinant of PAI-1 levels. Therefore, the increased PAI-1 and MCP-1 levels reported in fructose consumers may be explained by their increased visceral adipose tissue deposition [15-18].

Measurement of serum lipid profile

Table 5 shows that the levels of TC, TG, and LDL-C were substantially (P<0.05) higher in the obese diabetes patients group compared to the no obese diabetic patients group. HDL levels did not differ significantly (P>0.05) between the obese and non-obese diabetes patient groups. As can be shown in Table 5, the mean serum HDL levels of the obese diabetes patients group were lower than the mean levels of the no obese diabetic patients group.

Table 5: Lipid profile parameters in patients and control groups.

Parameter	N	Groups	Mean ± SD	CI 95%		Compared groups		P value
				Lower	Upper			
T.G (mg/dL)	30	Po	292.30 ± 123.88	246.04	338.56	Po	Pnon	0.00001
							Co	0.382
							Cnon	0.004
	30	Pnon	214.75 ± 46.37	197.43	232.07	Pnon	Co	0.028
							Cnon	0.0.001
	15	Co	129.81 ± 15.20	121.39	138.23	Co	Cnon	0.422
15	Cnon	106.90 ± 25.44	92.81	120.99				
T.C (mg/dL)	30	Po	301.32 ± 68.04	275.91	336.72	Po	Pnon	0.00001
							Co	0.00001
							Cnon	0.00001

HDL (mg/dL)	30	Pnon	232.11 ± 27.33	221.91	242.32	Pnon	Co	0.00001
							Cnon	0.00001
	15	Co	135.00 ± 26.97	120.06	149.93	Co	Cnon	0.558
	15	Cnon	125.14 ± 33.09	106.82	143.47			
	30	Po	41.58 ± 6.91	39	44.16	P	Pnon	0.006
							Co	0.014
							Cnon	0.00001
	30	Pnon	46.88 ± 6.42	44.48	49.28	Pnon	Co	0.858
							Cnon	0.001
	15	Co-	47.39 ± 6.41	43.84	50.94	Co	Cnon	0.009
	15	Cnon	54.57 ± 10.25	48.89	60.25			
	30	Po	201.27 ± 62.62	177.89	224.65	Po	Pnon	0.00001
LDL (mg/dL)							Co	0.00001
							Cnon	0.00001
	30	Pnon	142.28 ± 25.43	132.78	151.77	Pnon	Co	0.00001
							Cnon	
	15	Co	61.64 ± 30.46	44.77	78.51	Co	Cnon	0.435
	15	Cnon	49.19 ± 34.92	29.85	68.53			
	30	Po	58.46 ± 24.77	49.2	67.71	Po	Pnon	0.001
							Co	0.00001
							Cnon	0.00001
	30	Pnon	42.95 ± 9.27	39.48	46.41	Pnon	Co	0.001
							Cnon	0.00001
	15	Co	25.96 0 ± 3.04	24.27	27.64	Co	Cnon	0.422
vLDL (mg/dL)	15	Cnon	21.38 0 ± 5.08	18.56	24.19			

Note: Po: Patients obese; pnon: Patient non-obese; Co: Control obese; Cnon: Control non obese

When compared to the healthy obese group and the healthy no obese group, diabetic obesity persons had significantly higher T.C, LDL, VLDL and T.Gs levels. When compared to the healthy obese and healthy no obese groups, substantial increases in T.C, LDL-C, VLDL-C, and T.Gs levels were recognized in the diabetic no obese patients group. While HDL-C levels show a nonsignificant variation in these four groups. BMI, waist-hip ratio, body fat mass, body fat percentage, and all of the MS parameters were favorably correlated with plasminogen activation inhibitor-1 (PAI-1), whereas Adiponectin was negatively correlated with all of these variables except for HDL, where the trend was inverted. Multiple regression analysis showed that waist hip height ratio and triglyceride levels were independent predictors of adipocytes, and that subjects with MS had lower adiponectin and greater PAI-1 levels than healthy controls.

Public health efforts to stop the current epidemic of diabetes and CVD are urgently needed because of the evidence linking changes in lifestyle to improvements in several aspects of MS. Garg, et al.; Ivanova, et al. provide two examples. LDL (-) in type 2 diabetics' plasma was greater than in controls, even after glycemic correction. LDL (-) demonstrated a lower binding affinity to LDLr in cultured fibroblasts than LDL (+), therefore endothelial cells released more MCP-1 and IL-8. Diabetes atherosclerosis is caused by interactions between LDLs and arterial wall cells. LDL and IDL from type 2 diabetics encouraged human endothelial cells to generate MCP-1 mRNA [19-23].

Correlation analysis

The relevance of adiponectin with the biochemical parameters in the diabetic obese patients: Table 6 shows nonsignificant positive correlations of diabetic obese patients

obtained from LH, HDL, FSH, TAC, and ADIPOR1. Nonsignificant negative correlations were obtained from Age, FBS, BMI, T.G, ADIPO, MDA, LDL, MCP-1, PAI-1, TOS and vLDL.

Table 6: The correlation of adiponectin with concentrations of biochemical parameters in the diabetic obese patients.

Parameters	ADIPO (ng/mL)	
	r	P value
Age (years)	-0.122	0.519
BMI (kg/m ²)	-0.441	0.015
FBG (mg/dl)	-0.898	0.00001
T.C (mg/dl)	-0.686	0.00001
T.G (mg/dl)	-0.7	0.00001
HDL-C (mg/dl)	0.473	0.00001
LDL (mg/dl)	-0.754	0.00001
vLDL (mg/dl)	-0.655	0.00001
MDA (μmol/L)	-0.509	0.004
TAC (μmol/L)	0.817	0.00001
TOS (μmol/L)	-0.179	0.341
ADIPOR1	0.811	0.00001
HbA1C	-0.394	0.031
PAI-1	-0.83	0.0001
MCP-1	-0.641	0.0001
FSH	0.471	0.009
LH	0.24	0.201

Fasting blood glucose, adiponectin, and resisting all have a strong inverse correlation with serum adiponectin levels. (*Chi square*=0.013; *r*=-7.9) *P*=0.019, *r* =-6.6, significance level d. On the other hand, a significant positive correlation was found between serum resisting and fasting blood glucose. (*Chi square*=0.015; *r*=6.0).

The relevance of mcp-1 with the biochemical parameters in the diabetic obese patients: Table 7 shows non-

significant negative correlations of diabetic obese patients obtained from HDL, LH, FSH, TAC, and ADIPOR1. Non-significant positive correlations were obtained from Age, FBS, BMI, T.G, ADIPO, MDA, LDL, PAI-1, TOS and vLDL.

Table 7: The correlation of MCP-1with concentrations of biochemical parameters in the diabetic obese patients.

Parameters	MCP-1 (ng/mL)	
	r	P value
Age (years)	0.251	0.181
BMI (kg/m ²)	0.428	0.018
FBG (mg/dl)	0.715	0.00001
T.C (mg/dl)	0.336	0.069
T.G (mg/dl)	0.333	0.006

HDL-C (mg/dl)	-0.333	0.072
LDL (mg/dl)	0.703	0.00001
vLDL (mg/dl)	0.436	0.016
HbA1C	0.308	0.09
MDA (μmol/L)	0.398	0.029
TAC (μmol/L)	-0.619	0.00001
TOS (μmol/L)	0.199	0.291
ADIPOR1	-0.676	0.00001
FSH	-0.364	0.048
LH	-0.263	0.159
PAI-1	0.00001	

The relevance of pai-1 with the biochemical parameters in the diabetic obese patients: Table 8 shows non-significant negative correlations of diabetic obese patients

obtained from HDL, LH, FSH, TAC, and ADIPOR1. Non-significant positive correlations were obtained from Age, FBS, BMI, T.G, MDA, LDL, TOS and vLDL.

Table 8: The correlation of PAI-1with concentrations of biochemical parameters in the diabetic obese patients.

Parameters	PAI1-1 (ng/mL)	
	r	P value
Age (years)	0.304	0.102
BMI (kg/m ²)	0.286	0.141
FBG (mg/dl)	0.755	0.00001
T.C (mg/dl)	0.593	0.001
T.G (mg/dl)	0.611	0.00001
HDL-C (mg/dl)	-0.342	0.064
LDL (mg/dl)	0.697	0.00001
vLDL (mg/dl)	0.577	0.001
HbA1C	0.534	0.002
MDA (μmol/L)	0.195	0.3
TAC (μmol/L)	-0.563	0.001
TOS (μmol/L)	0.238	0.204
ADIPOR1	-0.735	0.00001
FSH	-0.452	0.012
LH	-0.084	0.658

The relevance of ADIPOR1 with concentrations of biochemical parameters in the diabetic obese patients: Table 9 shows nonsignificant positive correlations of diabetic obese patients obtained from LH, FSH and TAC. Nonsignificant

negative correlations were obtained from Age, FBS, BMI, T.G, ADIPO, HDL, MDA, LDL, MCP-1, PAI-1, TOS and vLDL.

Table 9: The correlation of ADIPOR1 with concentrations of biochemical parameters in the diabetic obese patients.

Parameters	ADIPOR1 (ng/mL)
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	r	P value
Age (years)	-0.065	0.73
BMI(kg/m ²)	-0.608	0.00001
FBG (mg/dl)	-0.831	0.00001
T.C (mg/dl)	-0.54	0.004
T.G (mg/dl)	-0.637	0.00001
HDL-C (mg/dl)	0.615	0.00001
LDL (mg/dl)	-0.792	0.00001
vLDL (mg/dl)	-0.554	0.001
HbA1C	-0.425	0.019
MDA (μmol/L)	-0.636	0.00001
TAC (μmol/L)	0.66	0.00001
TOS (μmol/L)	-0.091	0.63
FSH	0.451	0.012
LH	0.322	0.082

Conclusion

Excessive adiposity is the direct cause of type 2 diabetes, and the chance of developing the disease increases in a linear fashion along with body mass index. The increase in the prevalence of type 2 diabetes has so paralleled the worldwide trend toward increasing rates of obesity.

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