Chemical Methodologies 6(2022) 357-365

Chemical Methodologies

Journal homepage: http://chemmethod.com

Original Research Article

Correlation of Serum Adiponectin and C-reactive protein with Other Biochemical Parameters in Iraqi Pregnant Women

Israa Talib Ali[1], Namir Ibrahim A. Haddad[1,*], Eklas Ali Hussein[2]

[1]*Department of Chemistry, College of Science, University of Baghdad, Baghdad, Iraq*
[2]*Department of Obstetrics and Gynecology, College of Medicine, Al-Iraqia University, Baghdad, Iraq*

ARTICLE INFO

Article history

Submitted: 2022-02-05
Revised: 2022-03-02
Accepted: 2022-03-16
Manuscript ID: CHEMM-2202-1435
Checked for Plagiarism: **Yes**
Language Editor:
Dr. Behrouz Jamalvandi
Editor who approved publication:
Dr. Zeinab Arzehgar

DOI:10.22034/CHEMM.2022.328177.1435

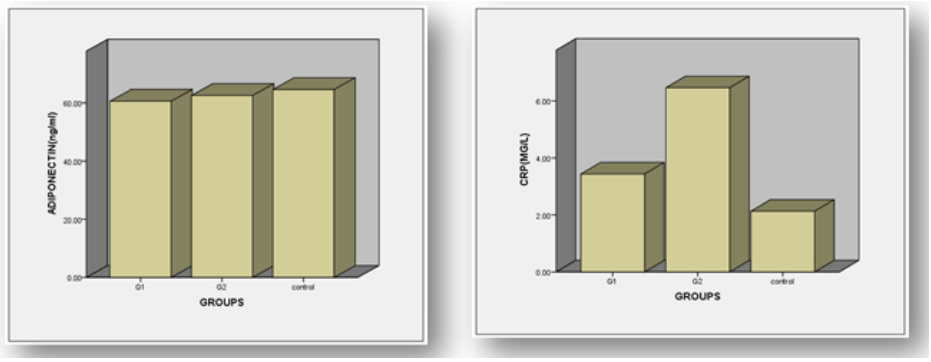
KEYWORDS

Adiponectin
CRP
Insulin resistance
Hyperlipidemia
Maternal adaptions

ABSTRACT

Adiponectin has anti-inflammatory, insulin-sensitizing and anti-atherogenic properties, while C-reactive protein (CRP) is a systemic inflammation marker. This study was conducted to investigate the relation between adiponectin and CRP with glycemic, lipid profile, liver enzymes, and blood pressure parameters in gestation. Ninety pregnant women were divided into three groups including: Thirty pregnant women in their first trimester (G1 group), another thirty pregnant women in their third trimester (G2 group) and thirty non-pregnant women (C or control group). All subjects had been fasting for ten hours before blood samples were drawn. The concentrations of serum adiponectin, serum CRP, fasting plasma glucose, serum insulin, glycated hemoglobin, alanine amine-transferase, aspartate amine-transferase, and blood pressure parameters were assessed. The results showed that serum adiponectin significantly decreased in gestation in G1 group (p<0.01) and non- significantly in G2 group in comparison to the C group. CRP continuously increased from G1 to G2 groups (p<0.01) in comparison to C group. In G1 group, there was a significant increase in fasting serum insulin. The homeostasis model assessment of insulin resistance (HOMA2-IR), systolic blood pressure and alanine amine-transferase significantly increased (p<0.05) in G1 group compared with C group. The homeostasis model assessment for β cell function (HOMA2-β) and diastolic blood pressure significantly increased (p<0.01) in G1 group in comparison with C group. In G2 group, BMI, BF %, FSI, HOMA2-β, HOMA2-IR, systolic, diastolic blood pressure, and lipid parameters were significantly increased (p<0.01) in comparison to C group, while aspartate amine-transferase significantly changed (p<0.05) when compared with C group. This study showed that the early decrease in serum adiponectin and its late correlation with high density lipoprotein might be an adaption mechanism to protect maternal system against high triglycerides level in third trimester.

GRAPHICAL ABSTRACT



* Corresponding author: Namir Ibrahim A. Haddad
✉ E-mail: namir.haddad@gmail.com

Introduction

Adiponectin hormone is a bioactive adipokine produced mainly by white adipose tissue [1]. Adiponectin consists of 224 amino acids [2]. It has an anti- inflammatory action, insulin sensitizing, anti-atherogenic [3] and immune-response regulating properties [4]. It circulates in the blood in three main isoforms: Low molecular weight (trimer), middle molecular weight (hexamer) and high molecular weight (4-6 trimers) [5]. Each adiponectin isoform activates definite signaling pathways, controlling distinct biological function [6]. C-reactive protein (CRP) is a systemic biomarker of inflammation, mainly produced in liver [7], adipocytes, endothelial cells, macrophages and lymphocytes. Its secretion increases rapidly in inflammation state [8]. CRP has two isoforms one of which is created in inflammation state at site of damaged or inflamed tissue and it has also more pro-inflammatory actions, while the other is created in absence of inflammation with anti-inflammatory action [9]. It is well known that pregnancy is a state of alterations in immune response, metabolic phase and endocrine system [10], which are obligatory for fetal development [11]. The success of pregnancy needs high-controlled adaption mechanisms [12], and the impairment in these mechanisms results in complications and pathologic states [13]. In this article, we aimed to investigate the behavior of adiponectin, and CRP, and their associations with other biochemical factors in gestation.

Materials and methods

Serum adiponectin and fasting serum insulin concentrations were determined by enzyme-linked immunoassay (ELISA) using commercial kits produced by Mybiosource Company (San Diego, USA) and Demeditec company (Lise Meitner, Germany), respectively. Serum CRP was analyzed by a latex turbidimetry method using kit produced by Linear Chemicals S.L. Company (Barcelona, Spain). Fasting plasma glucose (FPG), total cholesterol (TC), triglyceride (TG) were analyzed by enzymetic colorimetric method end point, alanine aminotransferase (ALT/GPT) and aspartate aminotransferase (AST/GOT) were analyzed by enzymetic colorimetric method kinetic using kits produced by Linear Chemicals, S.L. company (Barcelona, Spain). A glycated hemoglobin was analyzed by latex turbidmetry using kit manufactured by Spinreact, S. A. company (Santa Coloma, Spain). The high-density lipoprotein (HDL) was analyzed by enzymatic colorimetric end point method using kit made by Biosystem S.A. (Barcelona, Spain). The ELISA plate washer and reader used were made by Thermo, Germany. The spectrophotometer used was made by Hook and Tucker, Germany. The centrifuge and the water bath were made by Universal, Germany. And, Water bath was made by Memmert, Germany.

Homeostasis model assessment of insulin resistance (HOMA2-IR), insulin sensitivity (HOMA2-S) and β functions (HOMA2-β) equations were driven by the oxford center of diabetes [14]. The body mass index (BMI) and the body fat percentage (BF %) were calculated by Deurenberg's and Friedwald's equations [15,16].

Subjects

Ninety Iraqi women who had been visiting al-Yarmouk teaching hospital/ obstetrics and gynecology clinic from November 2021 to February 2021 were grouped into three groups (n=30); the first group included pregnant women in their first trimester (the G1 group), second group included pregnant women in their third trimester (G2 group) and third group included non-pregnant women (C group). All the subjects had been fasting for ten hours before blood samples were drawn at morning. A ten ml vein blood was drawn from each fasting subject then divided into parts: Two ml was poured into tubes coated with ethylenediaminetetraacetic acid (EDTA) for fasting plasma glucose (FPG) assay and eight ml poured into gel tubes. Biochemical test tubes were left for thirty minutes incubation then underwent a ten-minute centrifuge at 3000 xg. The serum was aspirated, poured in Eppindorff tube of (250 µl), and frozen under -20 °C till they were analyzed.

Data analysis

Data analysis was done by Statistical Package for the Social Sciences Software (SPSS), version 24.

Analysis of variance (ANOVA) was used to match between the means of different groups of a study. Post hoc test was used to estimate the significance of means difference between two groups. Pearson correlation was applied to evaluate the strength of relation serum adiponectin and other parameters. Also, multiple regression analysis was done to find a significant independent predictor for serum adiponectin. The *p*-value less than 0.05 refers to statically significant difference and less than 0.01 refers to statically high significant difference.

Results and Discussion

The main energy source for fetus is circulating maternal glucose which is shifted to fetus through placenta, based on which the blood glucose level must be maintained to meet the needs of the fetus, which is accomplished through a homeostatic increased in insulin resistance [17,18]. The previous fact is in alignment with our results in Table 1 that shows a non-significant decrease in Fasting Plasma Glucose (FPG) in G1 and G2 groups in comparison with C group and a significant and a highly significant increase in homeostasis model assessment of insulin resistance (HOMA2-IR) in G1, G2 groups in comparison to C group at (p<0.01). The current results reveal a significant decline in Homeostasis Model Assessment 2 insulin sensitivity (HOMA2-S) in G2 group (p<0.05) in comparison to C group. This result is in alignment with previous research [19, 20]. As shown in Table 1, adiponectin significantly declined in the G1group (p<0.01) and non-significantly declined in G2 group in comparison to C group (as shown in Figure 1), and this finding is in agreement with previous studies having reported a decline in adiponectin in normal and in complicated pregnancy by hypertension [21,22]. In Table 1, the Fasting Serum Insulin (FSI) and HOMA2-β continuously increased from G1 to G2 groups in comparison to C group. This increase is a compensatory mechanism for insulin resistance; this result is in agreement with previous studies [23,24]. The current results declare continuous elevation in CRP (p<0.01) from G1 to G2 groups in comparison to control group (as shown in Figure 2). This result is also consistent with previous study [25]. The glycated hemoglobin percentage (HbA1c %) declined in gestation due to short life span of erythrocytes in pregnancy [26,27]. Recently published studies are in agreement with our results displayed in Table 3, reporting that HbA1c % decreased in non-significant manner in G1 and G2 groups in comparison to C group. Further, Table 1 shows that the total cholesterol, triglyceride, low density lipoprotein (LDL) and very low-density lipoprotein (VLDL) significantly increased in G2 group (p<0.01), which is in line with another previous study [28]. The insulin resistance in late pregnancy attenuated insulin-lipolysis suppression [29] and decreased adipose tissue lipoprotein lipase activity leading to high level of circulating triglycerides are associated with lipoproteins that will be lipolysis by placental lipoprotein lipase into free fatty acids and shifted to fetus [30]. The results of the current study showed that HDL significantly decreased in G2 group with 56 % parous pregnant women (p<0.01) in comparison to C group, being parallel to the results of previous studies reporting that HDL in parous pregnant women was lower than that in nil-parous pregnant women [31,32,33]. Table 1 illustrates that although ALT significantly increased in G1 group (p<0.05) in comparison to C group and AST significantly decreased in G2 group (p<0.05) in comparison to C group; they both remain within normal limits in spite of hemo-dilution (increased plasma volume expansion in gestation more than volume of erythrocytes) [34-38].

Table 1 shows that both systolic blood pressure (SBP) and diastolic blood pressure (DBP) increased significantly (p<0.05) and significantly (p<0.01) from G1 to G2 groups, respectively in comparison to C group; this result is similar to those of previous studies [39, 40]. According to American Heart Association, blood pressure is classified as elevated when SBP is (120-129 mm Hg) and DBP is (< 80 mm Hg) [39]. Table 1 shows that BMI and BF % significantly increased in G2 group (p<0.01) in comparison to C group. This result is consistent with previous studies [41- 43].

Table 1: ANOVA analysis and post hoc test for parameters of G1, G2, C groups

Parameter	Group C	Group G1	Group G2	P-value
FPG(mg/dl)	85.61±7.26	81.88±11.17	79.59±12.56	0.09
HOMA2–IR (%)	1.23±0.31	1.75±0.8*a	1.85±0.9**b	<0.01
HOMA2–β (%)	125.80±24.62	169.68±36.97**a	191.52±51.54**b	<0.01
HOMA2-S (%)	85.31±17.60	72.21±24.19	65.58±31.3*b	<0.05
Adiponectin(ng/ml)	64.58±5.26	60.57±5.27**a	62.53±1.84	<0.01
FSI(micro(μ)IU/ml)	9.61±2.49	13.97±6.17*a	15.08±7.14** b	<0.01
CRP (mg/l)	2.13±0.27	3.43±0.97**a	6.46±2.44**b,**c	<0.01
HbA1c %	5.86±0.3	5.46±0.72	5.41±1.07	0.05
TC (mg/dl)	165.13±27.7	167.56±28.4	234.79±43.06**c,**b	<0.01
TG (mg/dl)	147.48±17.89	154.18±56.15	218.79±70.27**b,**c	<0.01
HDL(mg/dl)	46.62±11.46	44.85±8.33	38.27±6.77*b,*c	<0.01
LDL (mg/dl)	89.02±26.94	93.13±30.11	154.11±40.15**b,**c	<0.01
VLDL (mg/dl)	29.50±3.58	30.77±11.31	43.73±14.07**b,**c	<0.01
ALT (mg/dl)	15.07±5.25	19.71±7.58*a	17.15±6.51	<0.05
AST (mg/dl)	18.28±3.54	17.56±7.09	14.46±4.02*b*c	<0.05
SBP(mm Hg)	122.7±6.78	127.2±7.15*a	128.23±6.94*b	<0.01
DBP(mm Hg)	68.03±3.19	73.27±5.78**a	76.43±6.4**b	<0.01
BMI (kg/m^2)	25.01±3.97	25.03±2.80	29.93±6.46**b,**c	<0.01
BF %	31.46±5.26	31.44±4.44	36.58±8.21**b,**c	<0.01

a: refers to significant differences between G1 and C groups; b: refers to significant differences between G2 and C groups; c: refers to significant differences between G1and G2 groups; *: refers to significant difference p<0.05; **: refers to high significant difference p<0.01

Table 2 shows a Pearson correlation between serum adiponectin and other biochemical factors. The coefficient of correlation was represented by (r), and the significance of that correlation was represented by (*p*). Table 2 also shows a negative significant correlation between serum adiponectin with BMI and BF % (r=_0.434, *p*<0.05) and (r=_0.421, *p*<0.05) in the G2 group, which is in line with previous studies reporting that the high degree of obesity is parallel with a low degree of adiponectin level and that BF % increased by 25 % with 2.5 fold decrease in adiponectin level [44-46]. In obesity or gestational diabetes, insulin resistance induces ubiquitination or degradation of adiponectin [47]. Table 2 displays a positive significant correlation between adiponectin and HDL (r=0.376, p<0.05) in G2 group. This result is in tandem with preceding studies finding that incubation (50 μgm/ml HDL) with partially differentiated adipose tissue increased adiponectin expression by (1.5 fold).

Adiponectin correlates with HDL to minimize adverse effects of HDL decline in late gestation [48, 49]. Table 2 illustrates that there is no significant correlation between serum adiponectin and HOMA2-β, which is consistent with that of a prior study which reported that glucose-stimulated insulin secretion was not affected by serum adiponectin [50]. Another previous study reported that deficient adiponectin did not decrease insulin sensitivity in rats [51]; this finding is parallel with the current results which state that there is no significant correlation between serum adiponectin and HOMA2-S. Table 2 shows no significant correlation between adiponectin and TC, TG, LDL, and VLDL, which is partially consistent with a prior study [52]. The current results show that there is no significant correlation between adiponectin and SBP and DBP. Unlike this finding, a previous study has stated that decreased adiponectin due to elevation in pro-inflammatory tumor necrosis factor (TNF) leads to hypertension induced by vessels-constriction [53].

Table 2: Pearson correlation of serum adiponectin with other parameters in G1, G2 groups

Parameters	Maternal serum adiponectin					
	G1 (N=30)			G2(N=30)		
	r	P	Sig.	r	P	Sig.
BMI (kg/m^2)	0.122	0.522	N.S.	-0.434	<0.05	S.
BF %	0.098	0.606	N.S.	-0.421	<0.05	S.
FPG (mg/dl)	-0.018	0.927	N.S.	-0.285	0.127	N.S.
HbA1c (%)	-0.105	0.579	N.S.	-0.339	0.067	N.S.
FSI (µIU/ml)	-0.202	0.285	N.S.	-0.181	0.338	N.S.
HOMA2-IR	-0.197	0.309	N.S.	-0.145	0.446	N.S.
HOMA2-β (%)	-0.216	0.251	N.S.	0.158	0.405	N.S.
HOMA2-S(%)	0.156	0.409	N.S.	0.202	0.285	N.S.
TC (mg/dl)	0.241	0.200	N.S.	0.335	0.07	N.S.
TG (mg/dl)	-0.069	0.719	N.S.	-0.025	0.897	N.S.
HDL (mg/dl)	0.276	0.253	N.S.	0.376	<0.05	S.
LDL (mg/dl)	-0.04	0.832	N.S.	0.313	0.092	N.S.
VLDL (mg/dl)	-0.071	0.708	N.S.	-0.025	0.898	N.S.
AST (u/l)	0.229	0.223	N.S.	0.232	0.218	N.S.
ALT (u/l)	0.002	0.991	N.S.	0.345	0.062	N.S.
SBP (mmHg)	0.072	0.706	N.S.	0.02	0.917	N.S.
DBP (mmHg)	0.013	0.946	N.S.	-0.05	0.981	N.S.
CRP (mg/l)	-0.588	<0.01	H.S.	-0.175	0.354	N.S.

N.S.: for p≥0.05, S: for p<0.05, H.S.: for p< 0.01

As Table 3 reveals, serum CRP had a positive significant correlation with BMI, BF % (r=0.388, *p*=0.034) and (r=0.379, *p*=0.039), respectively, in G2 group. This result is consistent with previous studies which have reported a strong correlation between CRP and BMI in spite of gestational hemodilution [54,55]. In excessive fat accumulation as in obesity state, CRP significantly elevated in comparison to lean [55]. Table 3 displays a highly significant positive correlation between CRP and SBP (*P*<0.01) in G2 group; this finding is parallel to those of prior results [57]. The current results reported that there was no significant correlation between CRP and maternal glycemic parameters. This finding is in partial agreement with a previous study [58].

Table 3: Pearson correlation of serum CRP with other parameters in G1, G2 groups

parameter	Maternal serum CRP					
	G1 (N=30)			G2(N=30)		
	r	*P*	Sig	r	*p*	Sig
BMI(kg/m²)	0.296	0.112	N.S.	0.388	0.034	S.
BF%	0.264	0.159	N.S.	0.379	0.039	S.
FPG(mg/dl)	-0.168	0.347	N.S.	0.164	0.386	N.S.
HbA1c(%)	-0.012	0.949	N.S.	0.112	0.555	N.S.
FSI(µIU/ml)	-0.044	0.819	N.S.	0.045	0.811	N.S.
HOMA2-IR(%)	-0.057	0.765	N.S.	0.041	0.828	N.S.
HOMA2-β(%)	0.159	0.400	N.S.	-0.149	0.433	N.S.
HOMA2-S(%)	-0.102	0.590	N.S.	-0.008	0.968	N.S.
TC(mg/dl)	-0.173	0.347	N.S.	0.199	0.291	N.S.
TG(mg/dl)	0.025	0.894	N.S.	0.216	0.252	N.S.
HDL(mg/dl)	-0.131	0.491	N.S.	-0.171	0.367	N.S.
LDL(mg/dl)	-0.034	0.859	N.S.	0.144	0.449	N.S.
VLDL(mg/dl)	0.031	0.872	N.S.	0.215	0.255	N.S.
AST(u/l)	-0.12	0.529	N.S.	-0.079	0.680	N.S.
ALT(u/l)	0.107	0.575	N.S.	-0.261	0.164	N.S.
SBP(mmHg)	-0.013	0.947	N.S.	0.494	0.006	H.S.
DBP(mmHg)	-0.048	0.799	N.S.	0.147	0.437	N.S.

N.S.: for p≥0.05, S: for p<0.05, H.S.: for p< 0.01

Table 4 summarizes the regression analysis between serum adiponectin and studied parameters, stating that HDL with BMI form an independent-significant predictor model for serum adiponectin in late pregnancy (p<0.01).

Table 4: The regression analysis of studied parameters with serum adiponectin

Model 1	B	B	Coefficient of standard error	*p*-value
BMI	-0.144	-0.506	0.043	0.01
HDL	0.124	0.457	0.041	0.01

Adiponectin R^2 =0.391, Adjusted R^2 =0.346

*p<0.05, **p<0.01, no asterisk: p≥0.05

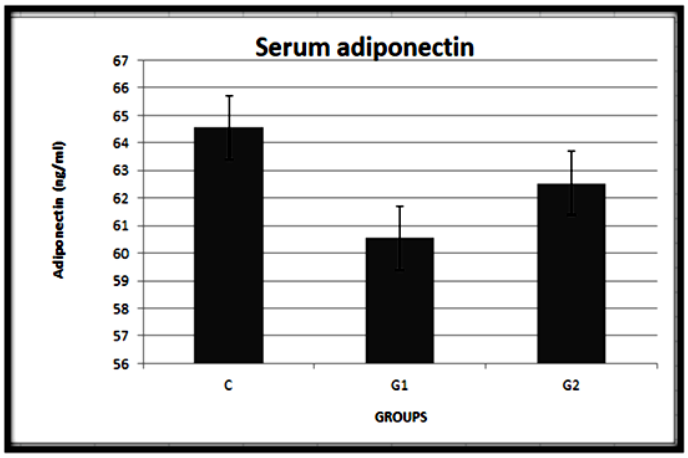


Figure 1: Maternal serum adiponectin of three groups

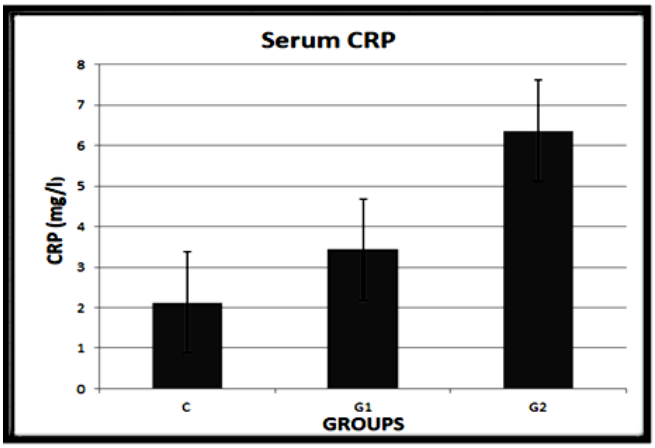


Figure 2: Maternal serum CRP of three groups

Conclusion

During pregnancy, the level of maternal serum adiponectin decreases. It has a substantially negative relationship with BMI, BF percent in late pregnancy, and CRP in early pregnancy. In late pregnancy, it has a favorable and significant relationship with HDL, which appears to be a mechanism for balancing the impacts of physiologically growing TC, TG, and CRP, which occurred naturally in the third trimester. In the third trimester, HDL with BMI was found to be a significant independent prediction model for serum adiponectin.

Funding

This research did not receive any specific grant from fundig agencies in the public, commercial, or not-for-profit sectors.

Authors' contributions

All authors contributed toward data analysis, drafting and revising the paper and agreed to responsible for all the aspects of this work.

Conflict of Interest

We have no conflicts of interest to disclose.

References

[1]. Naimo G.D., Gelsomino L., Catalano S., Mauro L., Andò S., *Front. Endocrinol.*, 2020, **11**:66 [Crossref], [Google Scholar], [Publisher]

[2]. Khoramipour K., Chamari K., Hekmatikar A., Ziyaiyan A., Taherkhani S., Elguindy N., Bragazzi N., *Nutrients*, 2021, **13**:1180 [Crossref], [Google Scholar], [Publisher]

[3]. Choi H.M., Doss H.M., Kim K.S., *Int. J. Mol. Sci.*, 2020, **21**:1219 [Crossref], [Google Scholar], [Publisher]

[4]. Szumilas K., Szumilas P., Słuczanowska-Głąbowska S., Zgutka K., Pawlik A., *Int. J. Mol. Sci.*, 2020, **21**:8265 [Crossref], [Google Scholar], [Publisher]

[5]. Nguyen T.M.D., Int. J. Prev. Med., 2020, **11**:136 [Crossref], [Google Scholar], [Publisher]

[6]. Bandini E., Rossi T., Gallerani G., Fabbri F., *Cancers*, 2019, **11**:1451 [Crossref], [Google Scholar], [Publisher]

[7]. Hosseini H., Koushki M., Khodabandehloo H., Fathi M., Panahi G., Teimouri M., Meshkani R., *Complement. Ther. Med.*, 2020, **49**:102251 [Crossref], [Google Scholar], [Publisher]

[8]. Huerta-Delgado A.S., Roffe-Vazquez D.N., Gonzalez-Gil A.M., Villarreal-Calderón J.R., Tamez-Rivera O., Rodriguez-Gutierrez N.A., Castillo E.C., Silva-Platas C., Garcia-Rivas G., Elizondo-Montemayor L., *J. Diabetes Res.*, 2020, 1949415 [Crossref], [Google Scholar], [Publisher].

[9]. Del Giudice M., Gangestad S., *Brain, Behav. Immun.*, 2018, **70**:61 [Crossref], [Google Scholar], [Publisher]

[10]. Liu H., Hou C., Li N., Zhang X., Zhang G., Yang F., Qiao S., *FASEB J.*, 2019, **33**:4490 [Crossref], [Google Scholar], [Publisher]

[11]. Most J., Marlatt K. Altazan A., Redman L., *Eur. J. Clin. Nutr.*, 2018, **72**:645 [Crossref], [Google Scholar], [Publisher]

[12]. Wu S.P., Li R., DeMayo F.J., *Trends Endocrinol. Metab.*, 2018, **29**:481 [Crossref], [Google Scholar], [Publisher]

[13]. P. Hofstee, D.R. McKeating, A.V. Perkins, Cuffe J.S., *Clin. Exp. Pharmacol. Physiol.*, 2018, **45**:871 [Crossref], [Google Scholar], [Publisher]

[14]. Calculator H.O.M.A., *The Oxford Center for Diabetes*. Endocrinology & Metabolism, Diabetes Trial Unit. 2009 [Google Scholar]

[15]. Deurenberg P., Weststrate J.A., Seidell J.C., *Br. J. Nutr.*, 1991, **65**:105 [Crossref], [Google Scholar], [Publisher]

[16]. Friedewald W.T., Levy R.I., Fredrickson D.S., *Clin. Chem.*, 1972, **18**:499 [Crossref], [Google Scholar], [Publisher]

[17]. Francis E.C., Dabelea D., Ringham B.M., Sauder K.A., Perng W., *Diabetologia*, 2021, **64**:83 [Crossref], [Google Scholar], [Publisher]

[18]. Huang Y., Chen F., Zhimin M., Heming G., Ruihua W., Hu J., *Rev. Bras. Ginecol. Obstet.*, 2016, **38**:287 [Crossref], [Google Scholar], [Publisher]

[19]. Kampmann U., Knorr S., Fuglsang J., Ovesen P., *J. Diabetes Res.*, 2019, 5320156 [Crossref], [Google Scholar], [Publisher]

[20]. Powe C.E., Huston Presley L., Locascio J.,. *Diabetologia*, 2019, **62**:1445 [Crossref], [Google Scholar], [Publisher]

[21]. Kadhum S., Al-Taha T., Al-assady A., *J. Thi-qar Univ.*, 2017, **12**:1 [PDF]

[22]. Naruse K., Noguchi T., Sado T., Tsunemi T., Shigetomi H., Kanayama S., Akasaka J. Koike N., Oi H., Kobayashi H., *Mediat. Inflamm.*, 2012, 432575 [Crossref], [Google Scholar], [Publisher]

[23]. Harlev A., Wiznitzer A., *Curr. Diab. Rep.*, 2010, **10**:242 [Crossref], [Google Scholar], [Publisher]

[24]. Huang Y., Chen F., Zhimin M., Heming G., Ruihua W., Hu J., *Rev. Bras. Ginecol. Obstet.*, 2016, **38**:287 [Crossref], [Google Scholar], [Publisher]

[25]. de Oliveira L.C., Franco-Sena A.B., Farias D.R., Rebelo F., Lepsch J., Lima N., Kac G., *J. Matern.Fetal Neonatal Med.*, 2017, **30**:2346 [Crossref], [Google Scholar], [Publisher]

[26]. Hashimoto K., Koga M., *J. Clin. Med.*, 2018, **7**:34 [Crossref], [Google Scholar], [Publisher]

[27]. Siddig A., Khalid A., Ali A., Musa O., *Saudi J. Med.*, 2018, **3**:40 [Crossref], [Google Scholar], [PDF]

[28]. Herrera E., Ortega-Senovilla H., *Curr. Pharm. Biotechnol.*, 2014, **15**:24 [Google Scholar], [Publisher]

[29]. Hernande T.L., Van Pelt R.E., Anderson M.A., Daniels L.J., West N.A., Donahoo W.T., Friedman,

J.E., Barbour L.A., *Diabetes Care*, 2014, **37**:1254 [Crossref], [Google Scholar], [Publisher]
[30]. Herrera E., Desoye G., *Horm. Mol. Biol. Clin. Investig.,* 2016, **26**:109 [Crossref], [Google Scholar], [Publisher]
[31]. Markovitz A.R., Haug E.B., Horn J., Fraser A., Macdonald-Wallis C., Tilling, K., Rimm E.B., Missmer S.A., Williams P.L., Romundstad P.R., Åsvold B.O., Rich-Edwards J.W., *J. Lipid Res.*, 2018, **59**:2403 [Crossref], [Google Scholar], [Publisher]
[32]. Pusukuru R., Shenoi A., Kyada P., Ghodke B., Mehta V., Bhuta K., Bhatia A., *J. Clin. Diagn. Res.*, 2016, **10**:QC12 [Crossref], [Google Scholar], [Publisher]
[33]. Wang H., Dang Q., Zhu H., *Lipids Health Dis.*, 2020, **19**:93 [Crossref], [Google Scholar], [Publisher]
[34]. Lee S.M., Park J.S., Han Y.J., Kim W., Bang S.H., Kim B.J., Park C., Kim M.Y., *J. Korean Med. Sci.*, 2020, **35**:e198 [Crossref], [Google Scholar], [Publisher]
[35]. Loganathan G., George R., Eapen C.E., Mathai M., Jasper P., Seshadri L., Shankar V., Paul S., Joseph G., Balasubramanian K., Chandy G. *Indian J. Gastroenterol.*, 2005, **24**:268 [Google Scholar], [Publisher]
[36]. Vricella L.K., *Am. J. Clin. Nutr.*, 2017, **106**:1620S [Crossref], [Google Scholar], [Publisher]
[37]. Shekhar S., Diddi G., *Taiwanese J. Obstet. Gynecol.*, 2015, **54**:475. [Crossref], [Google Scholar], [Publisher]
[38]. Hill C.C., Pickinpaugh J., *Surg. Clin. N. Am.*, 2008, **88**:391 [Crossref], [Google Scholar], [Publisher]
[39]. Gaillard R., akker R., Steegers E.A.P., Hofman A., Hofman A., Jaddoe V.W.V., *Am. J. Hypertens.*, 2011, **24**:1046 [Crossref], [Google Scholar], [Publisher]
[40]. Jahromi A.S., Shojaei M., Ghobadifa M.A., *Immune. Netw.,* 2014, **14**:149 [Crossref], [Google Scholar], [Publisher]
[41]. Hu J., Li Y., Zhang B., Zheng T., Li J., Peng J., Zhou A., Buka S., Zhang S., Shi Y., Xia K., Rexrode S., Xu S., *Circ. Res.*, 2019, **125**:184 [Crossref], [Google Scholar], [Publisher]
[42]. Prakash S., Pandeya D. R., *World J. Gynecol. Women's Health*, 2019, 3 [Google Scholar], [Publisher]
[43]. Most J., Marlatt K.L., Altazan A.D., Redman L.M., *Eur. J. Clin. Nutr.*, 2018, **72**:645 [Crossref], [Google Scholar], [Publisher]
[44]. Vernini J.M., Moreli J.B., Costa R.A.A., Negrato C.A., Rudge M.V.C., Calderon I.M.P., *Diabetol. Metab. Syndr.*, 2016, **8**:68 [Crossref], [Google Scholar], [Publisher]
[45]. Kapustin R.V., Chepanov S.V., Babakov V.N., Rogovskaya N.Y., Kopteeva E.V., Alekseenkova E.N., Arzhanova O.N., *Eur. J. Obstet., Gynecol. Reprod. Biol.*, 2020, **254**:284 [Crossref], [Google Scholar], [Publisher]
[46]. Catalano P.M., Hoegh M., Minium J., Huston-Presley L., Bernard S., Kalhan S., Hauguel-De Mouzon S., *Diabetologia*, 2006, **49**:1677 [Crossref], [Google Scholar], [Publisher]
[47]. Aye I.L.M.H., Rosario F.J., Kramer A., Kristiansen O., Michelsen T.M., Powell T.L., Jansson T., *J. Clin. Endocrinol. Metab.*, 2022, **107**:53 [Crossref], [Google Scholar], [Publisher]
[48]. Van Linthout S., Foryst-Ludwig A., Spillmann F., Peng J., Feng Y., Meloni M., Van Craeyveld E., Kintscher, U., Schultheiss, H., De Geest B., Tschöpe C., *Atherosclerosis*, 2010, **210**:438 [Crossref], [Google Scholar], [Publisher]
[49]. Pijanović M., Stefanović A., Miljković M., Marić-Krejović S., S. Spasić S., *LaboratoriumsMedizin*, 2017, **41**:129 [Crossref], [Google Scholar], [Publisher]
[50]. Staiger K., Stefan N., Staiger H., Brendle M., Brandharst D., Machicaa, F., Killerer M., Stumvall M., Fritche A., Harins H., *J. Endocrinol. Metab.*, 2005, **90**:6707 [Crossref], [Google Scholar], [Publisher]
[51]. Qiao L., Wattez J., Lee S., Nguyen A., Schaack J., Hay W., Jr W., Shao J., *Diabetes*, 2017, **66**:1126 [Crossref], [Google Scholar], [Publisher]
[52]. Makedou K., Kourtis A., Gkiomisi A., Toulis K., Mouzaki M., Anastasilakis A., Gerou S., Gavana E., Agorastos T., *Gynecol. Endocrinol.*, 2011, **27**:1070 [Crossref], [Google Scholar], [Publisher]
[53]. Craici I., Wagner S., Weissgerber T., Grande J., Garovic V., *Kidney Int.*, 2014, **86**:275 [Crossref], [Google Scholar], [Publisher]

[54]. Fink N.R., Chawes B., Bønnelykke K., Thorsen J., Stokholm J., Rasmussen M.A., Brix S., Bisgaard H., *Sci. Rep.*, 2019, **9**:3043 [Crossref], [Google Scholar], [Publisher]

[55]. Dockree S., Brook J., James T., Shine B., Impey L., Vatish M., *Clin. Chim. Acta*, 2021, **517**:81 [Crossref], [Google Scholar], [Publisher]

[56]. Tinius R.A., Cahill A., Strand E., Cade W., *Integr. Obes. Diabetes*, 2015, **2**:168 [Google Scholar], [Publisher]

[57]. de Jonge L., Steegers E., Ernst G., *J. Hypertens.*, 2011, **29**:2413 [Crossref], [Google Scholar], [Publisher]

[58]. Kianpour M., Saadatmand F., Nematbakhsh M., Fahami F., *Iran. J. Nurs. Midwifery Res.*, 2019, **24**:360 [Crossref], [Google Scholar], [Publisher]

HOW TO CITE THIS ARTICLE

Israa Talib Ali, Namir Ibrahim A. Haddad, Eklas Ali Hussein. Correlation of Serum Adiponectin and C - Reactive protein with Other Biochemical Parameters in Iraqi Pregnant Women. *Chem. Methodol.*, 2022, 6(5) 357-365
https://doi.org/10.22034/CHEMM.2022.328177.1435
URL: http://www.chemmethod.com/article_147221.html