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ORIGINAL RESEARCH

Association of Adiponectin & Leptin with Insulin Resistance in Type-2 Diabetes

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ABSTRACT

Introduction: Type 2 diabetes is a growing health concern, and early detection is crucial for effective management.It's been demonstrated in few recent studies that the abnormal levels of adipocytokines (Adiponectin, leptin) can contribute to IR and type-2 diabetes. **Objective:** This study aimed to investigate the relationship between insulin resistance, adiponectin, and leptin in type 2 diabetes patients and healthy controls. **Material and Method:** A total of 180 participants (120 patients and 60 controls) were enrolled in this study. Demographic and clinical profiles were compared between the two groups. Correlations between insulin resistance, adiponectin, and leptin were analysed, and optimal cutoff values were determined. **Result:** Patients had higher mean BMI, fasting blood sugar, postprandial blood sugar, total cholesterol, triglycerides, LDL, and VLDL, and lower mean HDL. Insulin resistance was positively correlated with adiponectin and leptin. The optimal cutoff values for insulin resistance, adiponectin, and leptin were 2.40, 17.22, and 18.75, respectively.At these cutoff values, insulin resistance and adiponectin showed perfect sensitivity and specificity (100% each), resulting in an accuracy of 100%. Leptin, on the other hand, showed a sensitivity of 80%, specificity of 55%, and accuracy of 74.9%. **Conclusion:** This study highlights the importance of insulin resistance, adiponectin, and leptin as biomarkers for type 2 diabetes. Early detection and management of insulin resistance and related metabolic abnormalities are crucial for preventing the development of type 2 diabetes and its complications. This study provides valuable insights into the pathophysiology of type 2 diabetes and has implications for the development of novel therapeutic strategies.

Keywords: Adiponectin, leptin, type 2 diabetes mellitus, adipocytokines, insulin resistance.

INTRODUCTION

Diabetes mellitus is the leading cause of mortality and disability worldwide. Type-2 diabetes mellitus (T2DM) is most common form of diabetes occurring in 90% of the diabetic population[1].India has earned distinction of being a diabetes capital ofthe world.[2]The patients with diabetes in India are nearly over 62 million.[3]Insulin Resistance is themain characteristic of T2DM and it is developed in multiple organs including the skeletal muscle, liver, adipose tissue & the heart. Onset of hyperglycemia and diabetes is often leads by several years of IR.Obesity plays an importantpart in this phenomenon & provides a major link between T2DM and accumulation of fat.[4] Adipocytokines are cytokines secreted by adipose tissue. Among other, they include adiponectin and leptin.[5]Adiponectin generally modulates glucose regulation andfatty acid catabolism.[6]Despite being generated in adipose tissue, adiponectin is reduced in obesity and the circulating levels of the adiponectin are correlated inversely with the body fatpercentage in adults showing significant increase after weight reduction.[7]Overt diabetes takes place when beta cells of the pancreas can no longer afford excess insulin secretion in compensation of insulin resistance.[8] But exact mechanism that can lead to insulin resistance is still unknown/unclear.Adiponectin & leptin are the abundant adipocytokines generated by adipocytes, and the best studiedmolecules till date. Their role has been researched in relation to the various pathological conditions like the deranged energy homeostasis, the abnormal leukocyte migration, the polycystic ovary etc.[9]But adequate evidences are still lacking regarding the effect of the above molecules in pathogenesis of IRLeptin, the product of ob gene.[10] is almost expressed exclusively and produced by the white adipose tissue specifically, by differentiated adipocytes. It regulates the body weight, modulates the insulin activity, sensitivity, metabolism and the reproductive function.[11]Not enough data is present on the Indian population.

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The current study was performed to find if the T2DM in this phenotype, with/without obesity, displays any correlation that is significant with circulating levels of the adiponectin and leptin. So, with this basic idea the present study was done in north Indian population with the aim to overview the part of obesity, adiponectin, leptin and IR which lead to type-2 diabetes.

MATERIAL AND METHOD

This case control hospital-based study was performed in Department of General Medicine and Department of Biochemistry at Gautam Buddha Chikitsa, Mahavidyalaya, RBB Subharti University, Dehradun, Uttarakhand;on type 2 diabetes cases & age-sex matched healthy adult population. A total 120 recent onset diabetic mellitus patients and 60 healthy subjects who were non-diabetic, non-hypertensive and having no family history of hypertension were enrolled in our study during the study period.

Study population: A total 120 recent onset diabetic mellitus patients and 60 healthy subjects who were non diabetic, non-hypertensive and having no family history of hypertension who were visited to OPD hospital.

Study Design: Case control study

Study location: Department of General Medicine and Department of Biochemistry at Gautam Buddha Chikitsa, Mahavidyalaya, RBB Subharti University, Dehradun,Uttarakhand

Sample Size: 180 patients

Sample size calculation: The sample was calculated on the basis of prevalence using the formula: -

$$n = \frac{Z^2 P(1-P)}{d^2}$$

Z (Confidence Level) is 1.96, d (error) is 4.36, **n = 180.**

Inclusion Criteria

- 120 recent onset (<2 years of disease duration) type-2 diabetic patients
- Age 30- 60 years
- Both male and female

Exclusion criteria

- Subjects with any other chronic illness were not included
- Patient on insulin therapy or on oral hypoglycemics other than short acting 2nd generation sulfonylurea.

Investigation

- After giving the informed consent, the anthropometric measurements like height, and weight, were taken. Height was calculated to the closest 0.1 cm with subject standing barefoot. Body-weight was measured to nearest 0.1 kg on the balanced scale. BMI was calculated by weight (kg) divided by height (m) square.

Blood collection

- Fasting blood samples (8 ml) from the participants were collected by venipuncture & collected in plain and EDTA vacutainers.

Biochemical investigations

- Serum glucose and cholesterol, triglyceride, high density lipoproteins (HDL), and low-density lipoproteins (LDL) were estimated using commercially available kits. The serum-levels of circulating adiponectin & leptin were calculated by available ELISA kits (Invitrogen, USA).It was calculated using HOMA-IR (Homeostasis model assessment of insulin resistance) calculation.
- HOMA-IR = Fasting plasma insulin (µU/L) × fasting glucose (mg)dl) / 22.5.

Statistical analysis

- Data were analyzed using the Statistical Package of the Social Sciences (SPSS version 20.0). Data are presented as mean ± standard deviation. Independent sample t-test, Chi-square test, Pearson correlations were used to compare different parameters. ROC curve analysis was done for diagnostic efficacy of the markers. The differences among the means (Mean ± SD) were considered significant if $P < 0.01$ & 0.05.

OBSERVATION & RESULTS

The patient group had a lower proportion of individuals ≤40 years old (40.8% vs 18.3%, p=0.002) and a higher proportion of individuals aged 41-50 years (33.3% vs 58.3%). The control group had a higher proportion of males (66.7% vs 52.5%, p=0.070), although the difference was not statistically significant. Notably, all patients had a BMI >25 kg/m2, while all controls had a BMI ≤25 kg/m2, indicating a significant difference in BMI between the two groups (p<0.001). [Table No. 1]

The clinical and biochemical profile of the cases (n=120) and the controls (n=60) and the association of all the parameters were found to be highly statistically significant (p<0.001) among the cases and the controls. [Table No. 2]

Insulin resistance was positively correlated with adiponectin (r=0.813, p<0.001) and leptin (r=0.280, p<0.001), indicating a strong association between these biomarkers. Adiponectin was also positively correlated with leptin (r=0.391, p<0.001). Additionally, insulin resistance was significantly correlated with fasting insulin (r=0.834, p<0.001),

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fasting blood sugar (r=0.838, p<0.001), postprandial blood sugar (r=0.790, p<0.001), BMI (r=0.814, p<0.001), total cholesterol (r=0.825, p<0.001), triglycerides (r=0.693, p<0.001), LDL (r=0.830, p<0.001), and VLDL (r=-0.794, p<0.001). In contrast, insulin resistance was negatively correlated with HDL (r=-0.857, p<0.001). [Table No. 3]

The receiver operating curve (ROC) which shows that IR and Adiponectin and also leptinrebelled that the optimal cutoff values for insulin resistance, adiponectin, and leptin were 2.40,17.22, and 18.75, respectively. At these cutoff values, insulin resistance and adiponectin showed perfect sensitivity and specificity (100% each), resulting in an accuracy of 100%. Leptin, on the other hand, showed a sensitivity of 80%, specificity of 55%, and accuracy of 74.9%.[Table No. 4 & 5]

Table No. 1: Demographic variables in Studied Patients and Healthy control

		Case (n=120)	Control (n=60)	Total		P value
Age (Years)	**≤40**	49 (40.8%)	11 (18.3%)	60 (33.3)	**12.175**	**0.002**
	41-50	40 (33.3%)	35 (58.3%)	75 (41.7)		
	>50	31 (25.8%)	14 (23.3%)	45 (25.0)		
Sex	**Male**	63 (52.5%)	40 (66.7%)	103 (57.3)	3.280	0.070
	Female	57 (47.5%)	20 (33.3%)	77 (42.7)		
BMI (kg/m^2)	**≤25**	0 (0.0%)	60 10(0.0%)	60	**180.000**	**<0.001**
	>25	120 (100.0%)	\0 (0.0%)	120		

Table No. 2: Clinical Profile of the Studied Patients and Healthy control.

	Case (n=120)	Control (n=60)	t-value	p-Value
Age	42.98±8.165	46.18±5.413	2.747	0.007
BMI	32.97±4.03	21.29±1.96	21.209	<0.001
Fasting Blood Sugar	154.46±35.91	90.88±12.94	13.274	<0.001
Post Prandial Blood Sugar	245.67±52.98	80.55±10.95	23.856	<0.001
Total Cholesterol	229.47±33.31	115.17±10.80	25.877	<0.001
Triglycerides	243.06±40.18	184.63±19.10	10.666	<0.001
HDL	34.34±6.91	121.45±21.13	41.071	<0.001
LDL	146.67±32.67	24.27±4.23	28.859	<0.001
VLDL	48.72±7.74	113.50±19.72	31.513	<0.001
Fasting Insulin	13.23±2.10	6.41±1.02	23.796	<0.001
Insulin Resistance	5.02±1.07	1.29±0.29	26.519	<0.001
Adiponectin	27.19±5.02	7.80±2.91	27.645	<0.001
Leptin	36.35±25.86	19.85±8.41	4.810	<0.001

Table No. 3: Interrelationship of adiponectin and Leptin with Insulin resistance in Type-2 Diabetes of study area

		Insulin Resistance	Adiponectin	Leptin
Insulin Resistance	Pearson Correlation	1	0.813^{**}	$.280^{**}$
	p-value	--	0.000	0.000
Adiponectin(ng/ml)	Pearson Correlation	0.813^{**}	1	0.391^{**}
	p-value	0.000	--	0.000
Liptin(ng/ml)	Pearson Correlation	0.280^{**}	0.391^{**}	1
	p-value	0.000	0.000	--
Fasting Insulin(IU/ml)	Pearson Correlation	0.834^{**}	0.791^{**}	0.324^{**}
	p-value	0.000	0.000	0.000
Fasting Blood Sugar (mg/dl)	Pearson Correlation	0.838^{**}	0.629^{**}	0.171^{*}
	p-value	0.000	0.000	0.022
Post Prandial Blood Sugar (mg/dl)	Pearson Correlation	0.790^{**}	0.781^{**}	0.295^{**}
	p-value	0.000	0.000	0.000
BMI (Kg/m^2)	Pearson Correlation	0.814^{**}	0.779^{**}	0.212^{**}
	p-value	0.000	0.000	0.004
Total Cholesterol (mg/dl)	Pearson Correlation	0.825^{**}	0.776^{**}	0.225^{**}
	p-value	0.000	0.000	0.002
Triglycerides (mg/dl)	Pearson Correlation	0.693^{**}	0.576^{**}	0.183^{*}
	p-value	0.000	0.000	0.014
HDL (mg/dl)	Pearson Correlation	-0.857^{**}	-0.851^{**}	-0.337^{**}

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	p-value	.000	0.000	0.000
LDL (mg/dl)	Pearson Correlation	0.830**	0.803**	0.253**
	p-value	0.000	0.000	0.001
VLDL (mg/dl)	Pearson Correlation	-0.794**	-0.827**	-0.320**
	p-value	0.000	0.000	0.000
**. Correlation is significant at the 0.01 level (2-tailed).				
*. Correlation is significant at the 0.05 level (2-tailed).				

Table No. 4: ROC Curve analysis of Insulin Resistance, Adiponectin and leptin for detection of diabetes.

Test Result Variable(s)	Area	Std. Error[a]	Asymptotic Sig.[b]	Asymptotic 95% Confidence Interval	
				Lower Bound	Upper Bound
Insulin Resistance	1.000	0.000	0.000	1.000	1.000
Adiponectin	1.000	0.000	0.000	1.000	1.000
Liptin	0.749	0.036	0.000	0.678	0.821
The test result variable(s): Liptin has at least one tie between the positive actual state group and the negative actual state group. Statistics may be biased.					
a. Under the nonparametric assumption.					
b. Null hypothesis: true area = 0.5					

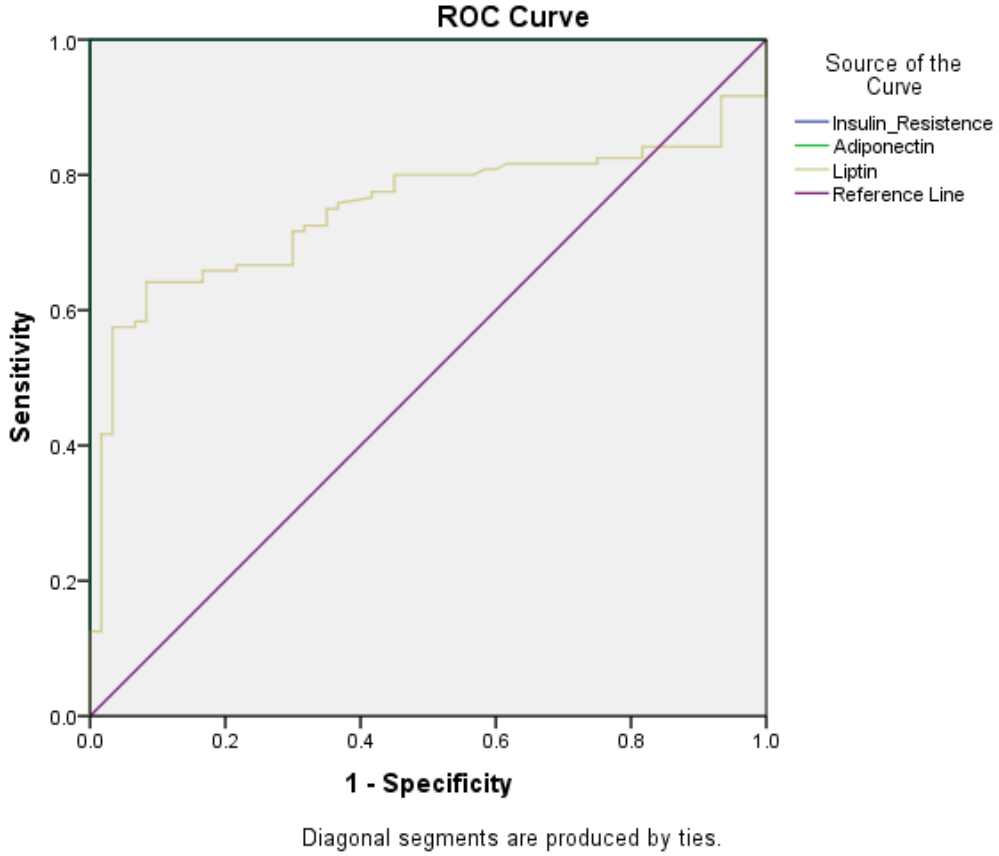

Table No. 5: Sensitive, Specificity and Accuracy of the adiponectin & Leptin with Insulin resistance in Type-2 Diabetes of study area

	Cutoff Value	Sensitive	Specificity	Accuracy
Insulin Resistance	2.40	100.0%	100.0%	100.0%
Adiponectin	17.22	100.0%	100.0%	100.0%
Liptin	18.75	80.0%	55.0%	74.9%

DISCUSSION

Leptin is a type of adipokinethat under the normal physiological condition functions to reduce the appetite, increase the energy expenditure, increase the sympathetic activity, facilitate the glucose utilization, and improve the insulin sensitivity.[12]It is expressed in the levels proportionate to the adipose mass, and although it is generally produced by adipocytes, it is also produced by the vascular smooth muscle cells, cardiomyocytes, & placenta in the pregnant women.

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This was a case-control observational study performed in a tertiary care teaching hospital in the north India.The present study was carried out at department of Medicine, Subharti Medical College and hospital; Meerut. Search terms included "Leptin" or "Adiponectin" and "type-2 Diabetes" or "Insulin resistance", and combinations thereof. Few case-control studies by**Das P et al.[13]and Aleidi S et al**.[14]have directly correlates the diponectin serum levels correlate with insulin resistance (IR) in type-2 diabetic patients, interrelationship of adiponectin & Leptin with IR in Type-2 Diabetes. The advantages of the cross-sectional study is that it is used to prove and or disprove the assumptions, inexpensive, captures the specific point in time, contains multiple variables at time of data snapshot. Other than the above-mentioned studies mostly were the case reports in which one or two patients were analyzed.

Serum insulin was measured by sandwich ELISA method using the Bioline insulin assay kit for the research purpose only. Adiponectin Estimation wasmeasured using the Bio Vendor's Human ELISA (sandwich) kit while leptin estimation was performed was measured through DRG kit (sandwich ELISA). Similar tools were used by **Das P et al, Aleidi S et al.[5]**and **Yadav.[15]**in their respective studies that show the above-mentioned tools were used as a gold standard for evaluating serum Adiponectin, leptin & Insulin resistance in type 2 Diabetes patients.

In our study the mean age of the cases was found to be42.98±8.16 years while that of controls it was 46.18±5.41 years and the association among them was found to be statistically significant ($p<0.05$). Similar observations were reported by **Das P et al.[4],Diwan AG et al[8] Shebla TH et al[9]**and **Aleidi S et al.[5]**in their respective studies which show that the problem of T2Dm was mainly associated in the 5th decade of the life and as the age increases the prevalence of diabetes also increases.

In present study the majority of patients were found to be males (52.5%) in cases and 47.5% were females while 66.7% were male and 33.3% were females in controls. Our study was in accordance to the study performed by **Yadav A et al.[6], and Diwan AG et al [8]Shebla TH et al [9]** and **Aleidi S et al[5]**who also reported the prevalence of males over females in their respective studies. According to the above studies it can be interpret that males are more exposed to the unhealthy environment than females which causes the diabetic easily.

In the present study there was a statistically significant association among the cases and the controls in respect to their BMI measurement ($P<0.05$). **Das P et al[4]**reported the similar finding in their study ($p<0.05$). Also, **Aleidi S et al[5]**depicted the significant association among the cases with respect to BMI ($p<0.05$). The above discussion shows that BMI increase with diabetes. In our study the Interrelationship of adiponectin, Leptin and Insulin resistance with the clinical parameters of the studied patients and there was a positive and statistically significant ($p<0.05$) correlation among them.**Das P et al[4]**reported positive correlation of leptin and negative correlation of adiponectin with insulin resistance. Our finding is also supported by some other workers.[16,17] Corroborative with our study finding, recently some studies have shown that leptin prevents insulin secretion and antagonises insulin effects on liver and adipose tissue.It is known that insulin resistance increases with age, which would predict lower adiponectin levels in the elderly.[18] Interestingly, our study has shown the adiponectin levels were related positively with age andthe increase in the adiponectin levels with the age can be explained by the facts that the decline in the sex steroidal hormones with the age may rise adiponectin levels in the elderly.[19,20] Moreover, the downfall in renal function with the aging might decrease adiponectin clearance by kidney.[21] Generally, obesity is related with insulin resistance.[13] Abdominal obesity, where fat is distributedcentrally, is particularly the major determinant of IR.[13,22] It's been reported that the abdominal obesity, measured by WC, is strongly correlated with the lower levels of the adiponectin and decreasesthe insulin sensitivity among the diabetic patients.[13,23] The high serum-leptin was recorded in the diabetics, compared to the incidence of high-serum leptin in non-diabetics. The leptin levels were found lower in diabetic population & were higher in females of diabetic & non-diabetic groups.[24] and **McNeely *et al.*[25]**showed that among the Japanese Americans, raised baseline leptin-levels were associated with the increased risk of the developing T2DM in men as compared to women. In accordance with such studies, the incidence of high serum-leptin was raised in diabetics than non-diabetics.In the present study the receiver operating curve (ROC) which shows that IR and Adiponectin and also leptin above 2.40, 17.22 and 18.75 can be used as cut off to predict the occurrence of T2DM in patients with sensitivity 100.0 and specificity 100.0 and 80.0% respectively whereas the accuracy was 100.0%, 100.0% and 74.9%. **Bozkurt L et al.[26]**reported that during pregnancy progress, GDM women displayed constantly lower concentrations of the adiponectin in contrast to the unaffected pregnancies as such women were analyzed by the decreasing trajectories towards the end of the gestation of initially raised levels. Moreover, the adiponectin remained lower in those with downgrade glucose tolerance at the early postpartum after the pregnancy with GDM.

Strengths

The study was carried out on the basis of consent data our study was very much usefulfor generating the hypothesis for public health planning and can measure the association among the variables.

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Limitations

The cross-sectional study design might have led to a selection bias.The self-reported history of diabetes mellitus in this low-education population might have underrated the number of subjects with diabetes mellitus

Recommendations

Larger epidemiological studies are needed to clarify the diagnostic value to evaluate serum Adiponectin, leptin &IR in type-2 Diabetes patients in study population

CONCLUSION

In conclusion, this study demonstrated diabetic patients had higher mean BMI, fasting blood sugar, postprandial blood sugar, total cholesterol, triglycerides, LDL, and VLDL, and lower mean HDL. Additionally, patients had higher mean levels of fasting insulin, insulin resistance, adiponectin, and leptin.The study also revealed strong correlations between insulin resistance, adiponectin, and leptin, indicating their interrelated roles in the pathophysiology of type 2 diabetes. The optimal cutoff values for insulin resistance, adiponectin, and leptin were identified, and their sensitivity, specificity, and accuracy were determined.The findings of this study highlight the importance of insulin resistance, adiponectin, and leptin as biomarkers for type 2 diabetes. They also emphasize the need for early detection and management of insulin resistance and related metabolic abnormalities to prevent the development of type 2 diabetes and its complications. Further studies are warranted to explore the potential therapeutic targets and interventions aimed at modulating these biomarkers to improve glycaemic control and reduce cardiovascular risk in patients with type 2 diabetes.

REFERENCES

1. Shaw JE, Sicree RA, Zimmet PZ. Global estimates of the prevalence of diabetes for 2010 and 2030. Diabetes Res Clin Pract 2010; 87:4-14.
2. Mohan V, Sandeep S, Deepa R, Shah B, Varghese C. Epidemiology of type 2 diabetes: Indian Scenario. Indian J Med Res 2007;125:217-30
3. World Health Organization. Fact Sheet No. 312: What is Diabetes? Available from: http://www.who.iny/mediacentre/factsheets/fs312/en 2014. [Last accessed on 2015 Jul 22].
4. Bhatia LS, Curzen NP, Calder PC, Byrne CD. Non-alcoholic fatty liver disease: a new and important cardiovascular risk factor? Eur Heart J, 2012;33:1190–1200.
5. Waki H, Yamauchi T, Kamon J, Ito Y, Uchida S, Kita S, et al. Impaired multimerization of human adiponectin mutants associated with diabetes. Molecular structure and multimer formation of adiponectin. J Biol Chem 2003;278:40352-63.
6. Liu Q, Yuan B, Lo KA, Patterson HC, Sun Y, Lodish HF, et al. Adiponectin regulates expression of hepatic genes critical for glucose and lipid metabolism. Proc Natl Acad Sci U S A 2012;109:14568-73.
7. Díez JJ, Iglesias P. The role of the novel adipocyte-derived hormone adiponectin in human disease. Eur J Endocrinol 2003;148:293-300.
8. Ceriello A, Mokdad AH, Bowman BA, et al. The continuing epidemics of obesity and diabetes in the United States. JAMA 2001; 286: 1195–1200.
9. Lang K, Ratke J. Leptin and Adiponectin: new players in the field of tumor cell and leukocyte migration. Cell Commun Signal 2009; 7: 27.
10. Ling, C. and L. Groop, Epigenetics: a molecular link between environmental factors and type 2 diabetes. Diabetes, 2009. 58(12): p. 2718-25.
11. McNeely MJ, Boyko EJ, Weigle DS, Shofer JB, Chessler SD, Leonnetti DL, et al. Association between baseline plasma leptin levels and subsequent development of diabetes in Japanese Americans. Diabetes Care 1999;22:65-70
12. Dong M, Ren J. What fans the fire: insights into mechanisms of leptin in metabolic syndrome-associated heart diseases. Curr Pharm Des 2014;20:652–658.
13. Piyali Das1, Subhradev Biswas2, Subir Kumar Bandyopadhyay3, Madhumita Manna4, Gorachand Bhattacharya. Correlation of adipocytokines and oxidative stress in type 2 diabetes mellitus and their association with degree of insulin resistance.Int. J Basic Appl. Physiol.,5(1),2016
14. Shereen Aleidi a, Ala Issa a, Haidar Bustanji b, Mohammad Khalil a, Yasser Bustanji. Adiponectin serum levels correlate with **Error! Bookmark not defined.**insulin resistance in type 2 diabetic patients. Saudi Pharmaceutical Journal (2015) 23, 250–256
15. Amita Yadav • Pramila jyoti • S. K. Jain, Jayashree Bhattacharjee. Correlation of Adiponectin and Leptin with Insulin Resistance: A Pilot Study in Healthy North Indian Population. Ind J Clin Biochem (Apr-June 2011) 26(2):193–196
16. Girard J. Is leptin the link between obesity and insulin resistance? Diabetes Metab. 1997 ; 23(3):16-24.
17. Soodini GR, Hamdy O. Adiponectin and Leptin in Relation to Insulin Sensitivity. Metabolic syndrome and related disorders 2004; 2(2): 114-123
18. Cnop, M., Havel, P.J., Utzschneider, K.M., Carr, D.B., Sinha, M.K., Boyko, E.J., Kahn, S.E., 2003. Relationship of adiponectin to body fat distribution, insulin sensitivity and plasma lipoproteins: evidence for independent roles of age and sex. Diabetologia 46, 459–469.
19. Bottner, A., Kratzsch, J., Muller, G., Kapellen, T.M., Bluher, S., Keller, E., Kiess, W., 2004. Gender differences of adiponectin levels develop during the progression of puberty and are related to serum androgen levels. J. Clin. Endocrinol. Metab. 89, 4053–4061.
20. Nishizawa, H., Shimomura, I., Kishida, K., Maeda, N., Kuriyama, H., Nagaretani, H., Matsuzawa, Y., 2002. Androgens decrease plasma adiponectin, an insulin-sensitizing adipocyte-derived protein. Diabetes 51, 2734–2741
21. Isobe, T., Saitoh, S., Takagi, S., Takeuchi, H., Chiba, Y., Katoh, N., Shimamoto, K., 2005. Influence of gender, age and renal function on plasma adiponectin level: the Tanno and Sobetsu study. Eur. J. Endocrinol. 153, 91–98.

DOI: 10.69605/ijlbpr_14.3.2025.82

22. Gierach, M., Gierach, J., Ewertowska, M., Arndt, A., Junik, R., 2014. Correlation between body mass index and waist circumference in patients with metabolic syndrome. ISRN Endocrinol. 2014, 514589.
23. Mohammadzadeh, G., Ghaffari, M.A., 2014. Additional effect of diabetes mellitus type 2 on the risk of coronary artery disease: role of serum adiponectin. Iran Red. Crescent Med. J. 16, e8742
24. Tatti P, Masselli L, Buonanno A, Di Mauro P, Strollo F. Leptin levels in diabetic and nondiabetic subjects. Endocrine 2001;15:305-8.
25. McNeely MJ, Boyko EJ, Weigle DS, Shofer JB, Chessler SD, Leonnetti DL, et al. Association between baseline plasma leptin levels and subsequent development of diabetes in Japanese Americans. Diabetes Care 1999;22:65-70.
26. Latife Bozkurt ,1 Christian S. Göbl ,2 Sabina Baumgartner-Parzer,1 Anton Luger, Giovanni Pacini,3 and Alexandra Kautzky-Willer. Adiponectin and Leptin at Early Pregnancy: Association to Actual Glucose Disposal and Risk for GDM—A Prospective Cohort Study. International Journal of Endocrinology. Volume 2018, Article ID 5463762, 8 pages