



Original Article

Association between Lipid Profile Limitations and Blood Glucose Values among Obese Adult Men in South Babylon Province, Iraq

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Abstract

A main community health alarm, obesity is carefully related with metabolic syndromes for example, dyslipidemia and insulin resistance. This study aimed to assess the correlation between lipid profile limitations and fasting blood glucose values in an obese adult male in south Babylon province, Iraq. 60 adult male's individuals in a case-control investigation, including 30 obese individuals (BMI \geq 30 kg/m²) and 30 age-matched, healthy, non-obese controls. Blood glucose, total cholesterol, triglycerides (TG), high-density lipoprotein (HDL), low-density lipoprotein (LDL), and very-low-density lipoprotein (VLDL) were evaluate using conventional enzymatic colorimetric methods. The fasting blood glucose values of the obese and control groups did high significant different, according to the data (P<0.0001). Conversely, obese individual had significantly lower HDL values (P < 0.0001) and significantly higher values of total cholesterol, (TG, LDL, and VLDL). BMI was significantly positively correlated with TG, total cholesterol, and VLDL, whereas it was significantly negatively correlated with HDL. There was no correlation between fasting blood glucose and cholesterol limits or BMI. In conclusion, even in the absence of hyperglycemia, obesity in adult males is closely allied with negative changes in their lipid profiles. The consequences of this study highlight the significance of lipid profiles as early signs of metabolic risk in obese individuals in addition to the requirement for early interventions to reduce metabolic and cardiovascular problems.

Keyword: Obesity, Insulin, Lipid, Glucose, HDL

Introduction

Obesity has become a significant worldwide public health issue during the last forty years. It is caused by the accumulation of additional body fat,

which is damaging to health. A body mass index (BMI) of \geq 30 kg/m² is considered obese, while a BMI of \geq 25 kg/m² is considered overweight, according to the World Health Organization (WHO). In both developed and developing



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countries, the prevalence of overweight and obesity is rising universally through all age groups (Albuquerque *et al.*, 2017). One vital measure of metabolic health is the lipid profile, that includes total cholesterol, HDL, LDL, and TG. Insulin resistance, a threat factor for type 2 diabetes mellitus (T2DM), is intensely linked by dyslipidemia, a condition marked by elevated lipid levels that is commonly seen in obese people (Grundy, 2016). Insulin is vital for glucose metabolism since it helps cellular uptake of glucose and preserves homeostasis. However, excessive fat deposition and inflammation in obese individuals often block insulin spread, resulting in metabolic dysregulation (Le *et al.*, 2023). The relationship between changed lipid profiles and insulin resistance in obese people has been the subject of several studies. Studies reveal a clear correlation between lower HDL values and higher triglyceride values and insulin resistance (Baneu *et al.*, 2024). The metabolic health of obese individuals is further complicated by increased LDL and cholesterol values, which has been linked to the development of atherosclerosis and cardiovascular disorders (Tall *et al.*, 2022). The current study sought to determine whether there was a correlation between the lipid profiles and insulin values of obese males in south of Babylon, Iraq.

Materials and method:

Study design

The present study was aimed to examine the relationship between lipid profile and insulin values in obesity males. All patients were recruited from Marjan Teaching Hospital in Babylon. Obese individuals were diagnosed based on body mass index ($BMI \geq 30 \text{ kg/m}^2$), while non-obese controls had BMI values within the normal range (18.5–24.9 kg/m^2) (WHO, 2022). All patients were aged between [25-45] years, and gave informed consent before inclusion in the study. Individuals with a history of chronic conditions, including diabetes mellitus, cardiovascular diseases, thyroid disorders, liver or kidney diseases, or those undergoing lipid-lowering or hormonal therapies, were excluded

from the study. To reduce confounding factors, smoking and alcohol use were additional exclusion considerations. Anthropometric measures, such as body weight, height, and BMI computation, were performed on each patient.

Blood samples collection

Following an overnight fast of 10 to 12 hours, fasting blood samples were obtained using conventional venipuncture methods from a total of 60 individuals, comprising 30 samples from obese male patients and 30 samples from apparently healthy non-obese male individuals, who were matched for age. After centrifuging the blood for ten minutes at 3000 rpm, the serum was extracted and kept at -20°C until examination (Lozano *et al.*, 2023).

Biochemical analysis

This included the investigation the values of serum total cholesterol, triglycerides, HDL, LDL, and VLDL. These were measured using commercial enzymatic colorimetric kits: Total Cholesterol was determined using the Cholesterol Color Reagent Kit. Triglycerides were measured using the Triglycerides Reagent Kit. All absorbance readings were performed at 520 nm using a UV-Visible spectrophotometer. Each test was conducted in accordance with the manufacturer's instructions.

Results

Table 1 of the current study showed statistically significant differences between obese subjects and healthy controls in age distribution, residency, and BMI. Most obese participants were aged (35–40) years (23.33%), whereas the control group was predominantly younger (24–29 years), with a highly significant difference ($P = 0.001$). Residency also differed significantly ($P = 0.020$), with obesity being more prevalent among rural residents. Marital status, smoking habits, and medical history did not differ significantly between groups ($P > 0.05$). As expected, BMI classification showed a highly significant difference ($P < 0.0001$). All obese participants had a ($BMI \geq 30 \text{ kg/m}^2$).

Table (1). Socio-demographic features of patients and controls (n=60)

Variable	Category	Healthy control	Obese	Calculated P value
Age	24-29	21(70)	22(73.33)	0.001(HS)
	30-34	9(30)	1(3.33)	
	35-40	0(0)	7(23.33)	
	Total	30(100)	30(100)	
Residency	rural	0(0)	5(16.66)	0.020(S)
	urban	30(100)	25(83.33)	
	Total	30(100)	30(100)	
Marital status	single	10(33.33)	17(56.66)	0.069(NS)
	married	20(66.66)	13(43.33)	
	Total	30(100)	30(100)	
Smoking	No	8(26.66)	13(43.33)	0.176(NS)
	Yes	22(73.33)	17(56.66)	
	Total	30(100)	30(100)	
Medical history	diabetes mellitus	6(20)	4(13.33)	0.144(NS)
	diabetes mellitus and hypertension	6(20)	1(3.33)	
	hypertension	8(26.66)	9(30)	
	none	10(33.33)	16(53.33)	
	Total	30(100)	30(100)	
BMI	Less than 18	0(0)	0(0)	<0.0001(HS)
	18.5-24.9	26(86.66)	0(0)	
	25-29.9	2(6.66)	0(0)	
	30 or higher	2(6.66)	30(100)	
	Total	30(100)	30(100)	

NS: No significant difference at P<0.05, S: Significant difference at P<0.05, HS: High significant difference at p<0.01

Table 2 of the present study showed highly significant difference was observed in fasting

glucose values between obese and control groups (P<0.0001).

Table (2) Fasting glucose level in healthy and over-weight person

Variable	Healthy control		obese		
	Mean ± SE	Range	Mean ± SE	Range	
Fasting glucose	97.13±1.046	22	144.37±4.955	96	<0.0001(HS)

HS: High significant difference at p<0.01

Table 3 of the current study showed the obese participants exhibited markedly elevated total cholesterol and triglyceride (TG) values compared

with controls, with highly significant differences (P < 0.0001).

Table (3) Cholesterol values and TG level in healthy and over-weight person

Variable	Healthy control		Obese		P value
	Mean ± SE	Range	Mean ± SE	Range	
Cholesterol	103.43±3.73	94	178.2±7.19	125	<0.0001 (HS)
TG	84.7±4.85	100	232.03±10.5	307	<0.0001 (HS)

HS: high significant difference at P<0.01

Table 4 of the present study showed a highly significant reduction in HDL-cholesterol and

significant elevations in LDL and VLDL values were observed in obese subjects (P < 0.0001).

Table (4) HDL, LDL and VLDL level in healthy and over-weight person

Variable	Healthy control		obese		P value
	Mean ± SE	Range	Mean ± SE	Range	
HDL	43.26±2.34	39	30.3±0.89	20	<0.0001 (HS)
LDL	50.36±4.61	88	100.3±7.82	148	<0.0001 (HS)
VLDL	19.4±2.65	83	46.06±2.10	62	<0.0001 (HS)

HS: high significant difference at P<0.01

Table 5 of the current study revealed strong correlation and statistically significant associations between BMI and lipid parameters. BMI showed a strong positive correlation with TG (r = 0.798), cholesterol (r = 0.65), and VLDL (r = 0.663), indicating that increasing adiposity is

closely linked to worsening lipid profiles. Strong positive correlations between TG and VLDL (r = 0.871) and between cholesterol and LDL (r = 0.882) further validate the interrelated nature of lipid metabolism disturbances in obesity.

Table (5). Correlation among different studied parameters

parameter	R	BMI	Fasting	Cholest.	TG	HDL	LDL	VLDL
BMI	R	1						
	P	0						
fasting glucose	R	-0.071	1					
	P	0.592	0					
cholesterol	R	0.65	-0.097	1				
	P	0	0.463	0				
TG	R	0.798	-0.187	0.704	1			
	P	0	0.153	0	0			
HDL	R	-0.434	0.067	-0.472	-0.561	1		
	P	0.001	0.608	0	0	0		
LDL	R	0.428	0.056	0.882	0.448	-0.522	1	
	P	0.001	0.669	0	0	0	0	
VLDL	R	0.663	-0.143	0.592	0.871	-0.527	0.434	1
	P	0	0.277	0	0	0	0.001	0

Discussion

The current study is consistent with previous epidemiological data showing that decreased basal metabolic rate, hormonal changes, and cumulative lifestyle variables cause obesity prevalence to rise with age (Chooi *et al.*, 2019; WHO 2022). According to recent Middle Eastern and international research, there may be differences between rural and urban populations in terms of health responsiveness, access to preventive healthcare, dietary habits, and physical activity (Salam *et al.*, 2023). The absence of correlation with smoking is in line with research indicating that smoking status may interact with other lifestyle and metabolic variables rather than being a direct predictor of obesity (Kovács *et al.*, 2024). BMI classification verifying proper group classification. According to (Alfaris *et al.*, 2023), every obese participant had a BMI of at least 30 kg/m².

Normoglycemia in obese people may signal early-stage metabolic dysfunction without obvious hyperglycemia, despite the fact that obesity is a well-known risk factor for insulin resistance and type 2 diabetes (Islam, 2025). According to recent studies, normal fasting glucose does not rule out

underlying insulin resistance, and dyslipidemia frequently precedes glucose abnormalities in obesity (Jahdkaran and Sistanizad, 2025). As a result, fasting glucose might not be enough to identify metabolic impairment early on.

Obesity-related dyslipidemia is characterized by hypertriglyceridemia and hypercholesterolemia, which are indicative of increased hepatic lipogenesis and decreased lipid clearance (Nussbaumerova and Rosolova, 2023). Also the results of current study in line with extensive cohort studies showing a substantial correlation between obesity and higher TG and total cholesterol values, which raises the risk of cardiovascular disease (Pownall and Gotto, 2019).

Atherogenic dyslipidemia linked to obesity and metabolic syndrome is characterized by low HDL and high LDL/VLDL values (Poirier and Eckel, 2002). Impaired reverse cholesterol transport and elevated TG exchange through cholesteryl ester transfer protein (CETP) activity in obesity may be responsible for lower HDL values (Cochran *et al.*, 2025). Increased cardiovascular risk and endothelial dysfunction are further consequences of elevated LDL and VLDL.

The detrimental link between obesity and protective lipoproteins was confirmed by the negative correlation between BMI and HDL ($r = -0.434$). These results are in line with new research showing that the development of dyslipidemia is significantly influenced by visceral fat accumulation (Shetty *et al.*, 2025). Remarkably, there was no visible relationship between fasting blood glucose and lipid or BMI characteristics. This implies that in obese people, lipid abnormalities might appear before glucose dysregulation (Singla *et al.*, 2010). The results of this research demonstrated that BMI significantly negatively correlated with total cholesterol, TG, VLDL, and LDL and significantly positively correlated with HDL. This study imply that a higher BMI is associated with a more atherogenic lipid profile, which indicates a higher risk of cardiovascular disease. On the other hand, neither BMI nor lipid characteristics showed a significant correlation with fasting glucose in this patient. This results were consistent with previous research showing that dyslipidemia is associated with higher BMI in a number of populations. Dyslipidemia includes elevated total cholesterol, TG, and LDL as well as decreased HDL (e.g., significant positive correlations between BMI and lipid parameters and an inverse relationship with HDL). As an instance of the connection between obesity and dyslipidemia, a study of young people found that those who were overweight or obese had lower HDL values and higher mean values of total cholesterol, LDL, and TG than their patients who were normal weight (Rashid *et al.*, 2023). Furthermore, a different study revealed that across patients through a wide BMI range, greater BMI was negatively connected with HDL and positively correlated with total cholesterol and TG, supporting the link between obesity and unfavorable lipid profiles (Smith *et al.*, 2010).

Conclusion

In general, the current findings show that obesity is closely linked with unfavorable lipid variations, but not primarily with elevated fasting blood glucose in the early stages. This finding establish the importance of lipid profile as a primary indicator of metabolic hazard in obese patients,

even when hyperglycemia is not found. To stop the development of metabolic syndrome and cardiovascular disease, early intervention focusing on weight loss and lipid organization is recommended.

References

1. Albuquerque, D., Nóbrega, C., Manco, L., & Padez, C. (2017). The contribution of genetics and environment to obesity. *British medical bulletin*, 123(1), 159-173.
2. Alfaris, N., Alqahtani, A. M., Alamuddin, N., and Rigas, G. (2023). Global impact of obesity. *Gastroenterology Clinics*, 52(2), 277-293.
3. Baneu, P., Văcărescu, C., Drăgan, S. R., Cirin, L., Lazăr-Höcher, A. I., Cozgarea, A., ... and Cozma, D. (2024). The triglyceride/HDL ratio as a surrogate biomarker for insulin resistance. *Biomedicines*, 12(7), 1493.
4. Chooi, Y. C., Ding, C., and Magkos, F. (2019). The epidemiology of obesity. *Metabolism*, 92, 6-10.
5. Cochran, B. J., King, T. W., Chemello, K., Thomas, S. R., and Rye, K. A. (2025). HDL metabolism and function in diabetes mellitus. *Nature Reviews Endocrinology*, 1-14.
6. Grundy, S. M. (2016). Metabolic syndrome update. *Trends in cardiovascular medicine*, 26(4), 364-373.
7. Islam, M. R. (2025). *Metabolic syndrome as a nexus of diabetes and cardiovascular disease: pathophysiological insights, epidemiological evidence, and therapeutic implications* (Doctoral dissertation, Laurentian University Library and Archives).
8. Jahdkaran, M., and Sistanizad, M. (2025). From lipids to glucose: investigating the role of dyslipidemia in the risk of insulin resistance. *The Journal of Steroid*

- Biochemistry and Molecular Biology*, 106744.
9. Kovács, N., Shahin, B., Andrade, C. A. S., Mahrouseh, N., and Varga, O. (2024). Lifestyle and metabolic risk factors, and diabetes mellitus prevalence in European countries from three waves of the European Health Interview Survey. *Scientific Reports*, 14(1), 11623.
 10. Le, T. K. C., Dao, X. D., Nguyen, D. V., Luu, D. H., Bui, T. M. H., Le, T. H., and Nguyen, T. T. T. (2023). Insulin signaling and its application. *Frontiers in endocrinology*, 14, 1226655.
 11. Lozano, A. A., Malvar, W. J. D., Mana, B. A. B., Mercado, F. A. V., Queddeng, M. B., Tejano, A. S. P. P., & Florentino, C. M. (2023). Effect of Centrifugation Speed and Time on the Blood Chemistry Values of Healthy Individuals. *The Vector: International Journal of Emerging Science, Technology and Management (IJESTM)*, 32(1).
 12. Nussbaumerova, B., and Rosolova, H. (2023). Obesity and dyslipidemia. *Current atherosclerosis reports*, 25(12), 947-955.
 13. Poirier, P., and Eckel, R. H. (2002). Obesity and cardiovascular disease. *Current atherosclerosis reports*, 4(6), 448-453.
 14. Pownall, H. J., and Gotto Jr, A. M. (2019). Lipids and cardiovascular disease: putting it all together. *Methodist DeBakey cardiovascular journal*, 15(1), 5.
 15. Rashid, M., et al. (2023). Correlation of Body Mass Index with Serum Lipid Profile Level in Adolescent Students of Bangladesh. *Journal of Clinical & Diagnostic Research*. PMID:38944735.
 16. Salam, M. M., Yousuf, R., Salam, M. W., and Haque, M. (2023). Obesity and overweight: A global public health issue. *Advances in Human Biology*, 13(1), 154-156.
 17. Shetty, S., Suvarna, R., Bhattacharya, S., and Seetharaman, K. (2025). Visceral adiposity and Cardiometabolic risk: clinical insights and assessment. *Cardiology in Review*, 10-1097.
 18. Singla, P., Bardoloi, A., and Parkash, A. A. (2010). Metabolic effects of obesity: a review. *World journal of diabetes*, 1(3), 76.
 19. Smith, J. D., et al. (2010). Association of body mass index and lipid profiles: evaluation of a broad spectrum of body mass index patients including the morbidly obese. *Obesity Research & Clinical Practice*. PMID:20563664.
 20. Tall, A. R., Thomas, D. G., Gonzalez-Cabodevilla, A. G., and Goldberg, I. J. (2022). Addressing dyslipidemic risk beyond LDL-cholesterol. *The Journal of clinical investigation*, 132(1).
 21. World Health Organization (WHO) (2022). Obesity and Overweight. <http://www.who.int/mediacentre/factsheets/fs311/en/>.