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Changes in Blood Glucose and Lipid Profile in Obese vs. Non-Obese Individuals

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KEYWORDS

Blood Glucose, Lipid Profile, Obesity.

ABSTRACT:

Background: Obesity has become a major public health concern worldwide due to its strong association with metabolic disorders, including insulin resistance, type 2 diabetes, and dyslipidemia. Therefore, the present study was designed to evaluate and compare blood glucose and lipid profile parameters between obese and non-obese individuals. ***Aim of the study:*** The aim of the study was to evaluate and compare blood glucose and lipid profile parameters between obese and non-obese individuals. ***Methods:*** This cross-sectional comparative study at the Department of Physiology, Rajshahi Medical College, Rajshahi, Bangladesh (July 2014–June 2015) included 82 adults (42 obese, 40 non-obese). BMI, fasting and 2-hour postprandial glucose, and lipid profile (TC, TG, HDL-C, LDL-C, VLDL-C) were measured. Data were analyzed using unpaired t-test and Chi-square test ($p < 0.05$) in SPSS 20.0. ***Results:*** Among 82 participants (42 obese, 40 non-obese), BMI was higher in the obese group (33.1 ± 2.1 vs 22.8 ± 1.5 kg/m^2; $p < 0.001$). Obese individuals had elevated fasting (107.5 ± 9.2 vs 88.9 ± 5.8 mg/dL) and 2-hour glucose (145.8 ± 18.3 vs 112.4 ± 12.5 mg/dL), and showed higher total cholesterol, triglycerides, LDL-C, VLDL-C, with lower HDL-C (all $p < 0.001$). ***Conclusion:*** Obesity is associated with impaired glucose regulation and an atherogenic lipid profile, increasing cardiometabolic risk.

INTRODUCTION

In recent years, obesity has emerged as a significant public health issue due to its strong association with chronic conditions such as Cardiovascular Disease (CVD) and Type 2 Diabetes (T2D) [1]. Approximately two billion adults worldwide meet the World Health Organization's criteria for overweight or obesity [2]. This condition has become a major global health challenge, contributing substantially to the prevalence of non-communicable chronic diseases and ranking among the leading causes of illness and death in both high- and low-income countries [3]. Obesity now poses serious threats to population health, well-being, and economic stability in nearly every nation [4,5]. Current estimates suggest that over 300 million individuals are obese, placing nearly 1.7 billion people at elevated risk for severe conditions such as heart attacks and strokes [6,7].

Research indicates that obesity is closely linked with non-communicable disorders, including insulin resistance, type 2 diabetes mellitus, strokes, and certain types of cancer [3]. It represents a significant contributor to future metabolic complications. One of the primary indicators of obesity's detrimental impact is elevated blood glucose levels [8]. The excessive accumulation of adipose tissue, particularly in visceral and ectopic regions such as the liver and skeletal muscles, promotes the release of triglycerides and free fatty acids, triggering a range of metabolic and hormonal alterations. These changes progressively impair insulin signaling [9], leading to increased insulin resistance in

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adipose tissue, liver, skeletal muscle, and vascular endothelium, which may ultimately result in glucose intolerance [10]. Obesity is also a well-established risk factor for dyslipidemia, with 60–70% of obese individuals exhibiting abnormal lipid profiles [11,12], including increased triglycerides (TGs), very low-density lipoprotein (VLDL), and apolipoprotein B (Apo B), along with reduced levels of HDL and apolipoprotein A-I (Apo A-I), while LDL levels are typically normal or slightly elevated [13-15]. Although insulin resistance in obesity is closely linked with lipid metabolism disturbances, not all obese individuals progress to prediabetes or T2D [16,17].

The impact of obesity on metabolic health is frequently evaluated through measurements of blood glucose [8]. Elevated glucose levels indicate a higher risk of developing type 2 diabetes mellitus. Glycated hemoglobin (HbA1c) is the primary form of hemoglobin used to assess long-term glycemic control over the previous three months, providing a more stable indicator than short-term blood glucose measurements. HbA1c can also help predict the risk of diabetic complications and may serve as a more sensitive diagnostic tool for detecting diabetes and prediabetes compared to fasting glucose or oral glucose tolerance tests (OGTT) [18]. Lipid profiles, including LDL (low-density lipoprotein cholesterol, "bad" cholesterol), HDL (high-density lipoprotein cholesterol, "good" cholesterol), triglycerides, and total cholesterol, are key markers of cardiovascular and metabolic risk. LDL cholesterol can accumulate within arterial walls, increasing the likelihood of heart disease, whereas HDL cholesterol provides protection by removing excess cholesterol from circulation. Triglyceride levels are classified as normal (<150 mg/dL), mildly high (150–199 mg/dL), high (200–499 mg/dL), and very high (>500 mg/dL), while total cholesterol reflects a combination of LDL, HDL, and other lipid components, all of which are critical for assessing cardiovascular and metabolic health [19].

Despite extensive research on obesity and its metabolic consequences, data on the comparative alterations in blood glucose and lipid profile among obese and non-obese individuals remain limited, particularly in the South Asian population. Most studies have focused either on obesity-related insulin resistance or dyslipidemia independently, while comprehensive evaluations comparing both glucose and lipid parameters across different BMI categories are relatively scarce. Moreover, regional variations in lifestyle, diet, and genetic predisposition may influence these metabolic profiles, highlighting the need for population-specific evidence. Therefore, to address this gap, the present study was designed to evaluate and compare blood glucose and lipid profile parameters between obese and non-obese individuals.

Objective

- To evaluate and compare blood glucose and lipid profile parameters between obese and non-obese individuals.

METHODOLOGY & MATERIALS

This cross-sectional comparative study was conducted at the Department of Physiology, Rajshahi Medical College, Rajshahi, Bangladesh, from July 2014 to June 2015. A total of 82 adult participants were included, comprising 42 obese and 40 non-obese individuals, selected based on predefined inclusion and exclusion criteria. Data on anthropometric measurements, blood glucose, and lipid profile were collected to evaluate and compare metabolic parameters between the obese and non-obese groups.

Inclusion Criteria:

- Adults aged 18–60 years
- Willing to participate and provide informed consent

Exclusion Criteria:

- Known diabetes mellitus
- Hypertension or cardiovascular disease
- Chronic kidney or liver disease
- Use of medications affecting glucose or lipid metabolism

Demographic data including age, sex, and anthropometric measurements were recorded. Weight and height were measured using a standard weighing scale and stadiometer, and BMI was calculated as weight (kg) divided by height squared (m^2). After an overnight fast of 8–10 hours, venous blood samples were collected to measure fasting blood glucose, followed by a 2-hour postprandial glucose test after ingestion of 75 g oral glucose. Lipid profile parameters, including total cholesterol, triglycerides, HDL-C, LDL-C, and VLDL-C, were analyzed using standard enzymatic colorimetric methods. Data were expressed as mean ± standard deviation, and comparisons between obese and non-obese groups were performed using the unpaired Student's t-test for continuous variables and Chi-square test for categorical variables, with $p < 0.05$ considered statistically significant. All analyses were conducted using SPSS version 20.0.

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RESULTS

Table 1: Baseline Characteristics of Study Participants (n=82)

Parameter	Obese (n=42)	Non-Obese (n=40)	p-value
Age (years)	41.3 ± 9.5	39.8 ± 10.1	0.491
Gender (M/F)	22 / 20	19 / 21	0.825
BMI (kg/m²)	33.1 ± 2.1	22.8 ± 1.5	<0.001

Table 1 presents the baseline characteristics of 82 study participants. Among them, 42 were classified as obese and 40 as non-obese. The mean age was 41.3 ± 9.5 years in the obese group and 39.8 ± 10.1 years in the non-obese group. Gender distribution in the obese group included 22 males (52.4%) and 20 females (47.6%), while the non-obese group had 19 males (47.5%) and 21 females (52.5%). Body mass index (BMI) was significantly higher in the obese group (33.1 ± 2.1 kg/m²) compared to the non-obese group (22.8 ± 1.5 kg/m²; $p < 0.001$). No significant differences were observed in age or gender between the groups.

Table 2: Blood Glucose Profile of Study Participants (n = 82)

Parameter	Obese (n = 42)	Non-Obese (n = 40)	p-value
Fasting Blood Glucose (mg/dL)	107.5 ± 9.2	88.9 ± 5.8	< 0.001
2-Hour Postprandial Glucose (mg/dL)	145.8 ± 18.3	112.4 ± 12.5	< 0.001

Table 2 presents the comparison of fasting and postprandial blood glucose levels between obese (n = 42) and non-obese (n = 40) participants. Both fasting and 2-hour postprandial glucose values were significantly higher in the obese group ($p < 0.001$).

Table 3: Lipid Profile of Study Participants (n = 82)

Parameter	Obese (n = 42)	Non-Obese (n = 40)	p-value
Total Cholesterol (mg/dL)	215.6 ± 25.7	184.3 ± 20.4	< 0.001
Triglycerides (mg/dL)	168.2 ± 32.5	96.7 ± 22.1	< 0.001
HDL-C (mg/dL)	37.8 ± 4.9	52.3 ± 5.5	< 0.001
LDL-C (mg/dL)	136.1 ± 22.8	113.5 ± 18.2	< 0.001
VLDL-C (mg/dL)	33.6 ± 6.5	19.3 ± 4.4	< 0.001

Table 3 compares the lipid profile between obese (n = 42) and non-obese (n = 40) participants. The obese group showed significantly higher levels of total cholesterol, triglycerides, LDL-C, and VLDL-C, along with significantly lower HDL-C compared to the non-obese group (all $p < 0.001$).

DISCUSSION

Obesity is a major public health concern worldwide, closely associated with metabolic disorders such as insulin resistance, type 2 diabetes, and dyslipidemia. These metabolic alterations significantly increase the risk of cardiovascular disease and other long-term complications, affecting overall health and quality of life. Assessment of blood glucose and lipid profile parameters provides crucial insights into the metabolic status of individuals across different BMI categories. The findings of this study demonstrate that obese individuals exhibit significantly higher fasting and postprandial glucose levels, along with adverse lipid profiles, compared to non-obese participants, highlighting the pronounced metabolic impact of obesity and the importance of early monitoring and intervention.

In the present study, the mean age and gender distribution were comparable between obese and non-obese participants, indicating that differences observed in subsequent biochemical parameters are unlikely to be influenced by these factors. However, body mass index (BMI) was significantly higher in the obese group (33.1 ± 2.1 kg/m²) compared to the non-obese group (22.8 ± 1.5 kg/m²; $p < 0.001$), confirming appropriate group classification. These results are consistent with the findings of Al-Jawadi *et al.*[20], who also reported a significant difference in BMI between obese and non-obese adults (median = 31.86 vs 20.86; $p < 0.001$), while age and dietary parameters remained comparable. Additionally, national data from Gupta *et al.*[21] demonstrated a higher prevalence of overweight and obesity among Bangladeshi females (45.6%) than males (32.7%), supporting the importance of documenting gender distribution in obesity-related studies. Collectively, these findings establish a comparable demographic baseline and reinforce BMI as the primary distinguishing characteristic between obese and non-obese groups, providing a solid foundation for

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evaluating subsequent differences in blood glucose and lipid profile parameters.

In the present study, both fasting and postprandial blood glucose levels were significantly higher in obese individuals compared to non-obese participants, indicating a clear alteration in glucose metabolism associated with increased adiposity. The mean fasting glucose in the obese group (107.5 ± 9.2 mg/dL) was notably higher than that of the non-obese group (88.9 ± 5.8 mg/dL; $p < 0.001$), while the 2-hour postprandial glucose also showed a significant elevation (145.8 ± 18.3 mg/dL vs. 112.4 ± 12.5 mg/dL; $p < 0.001$). These findings are in agreement with Saraswati *et al*.[22], who reported that blood glucose levels at all time points following an oral glucose load were consistently higher among obese subjects than non-obese, reflecting impaired glucose tolerance. Similarly, Akter *et al*.[23] observed significantly elevated fasting serum glucose levels in obese individuals, supporting the present results. Collectively, these observations suggest that obesity contributes to dysregulation of glucose homeostasis, likely due to increased insulin resistance and reduced glucose utilization, thereby predisposing obese individuals to early metabolic disturbances.

In the present study, obese individuals demonstrated a distinctly altered lipid profile characterized by significantly higher total cholesterol, triglycerides, LDL-C, and VLDL-C levels, accompanied by a lower HDL-C concentration compared to non-obese participants. This dyslipidemic pattern is consistent with the findings of Mahaur *et al*.[24], who reported significantly elevated total cholesterol among obese individuals (189.2 mg/dL) compared to non-obese controls (159.8 mg/dL), supporting the current trend of higher cholesterol levels with increasing adiposity. Similarly, Taqiuddin *et al*.[25] observed that obese subjects had substantially higher triglycerides (≈ 318 mg/dL) and LDL-C (≈ 137 mg/dL), with reduced HDL-C (≈ 44 mg/dL) compared to non-obese participants, mirroring the present study's results. Furthermore, Alshuweishi *et al*.[26] found that obese adults exhibited more atherogenic lipid patterns, with lower HDL-C and elevated atherogenic fractions, in agreement with the dyslipidemic shifts observed here. Collectively, these findings reinforce that obesity is strongly associated with adverse lipid alterations, likely mediated by insulin resistance, increased hepatic VLDL synthesis, and reduced HDL-mediated reverse cholesterol transport, thereby predisposing obese individuals to atherogenic cardiovascular risk.

Limitations of the study

This study had some limitations:

- The study population was relatively small, limiting generalizability.
- The sample was not randomly selected.
- The study's limited geographic scope may introduce sample bias, potentially affecting the broader applicability of the findings.

CONCLUSION

Obesity is associated with significant metabolic alterations, including elevated fasting and postprandial glucose levels and an atherogenic lipid profile characterized by higher total cholesterol, triglycerides, LDL-C, VLDL-C, and lower HDL-C. These findings underscore the importance of monitoring and managing glucose and lipid parameters in obese individuals to reduce future cardiometabolic risk.

REFERENCES

1. Forse RA, Betancourt-Garcia MM, Kissee MC. Epidemiology and discrimination in obesity. The ASMBS textbook of bariatric surgery. 2019 Oct 17:3-14.
2. Kahaer M, Zhang B, Chen W, Liang M, He Y, Chen M, Li R, Tian T, Hu C, Sun Y. Triglyceride glucose index is more closely related to hyperuricemia than obesity indices in the medical checkup population in Xinjiang, China. Frontiers in Endocrinology. 2022 Mar 2;13:861760.
3. National Institute for Health and Care Excellence (NICE). Obesity. Clinical Knowledge Summary. London: NICE; 2012 Oct [cited 2025 Nov 4]. Available from: https://cks.nice.org.uk/topics/obesity/
4. Egusa G, Beltz WF, Grundy SM, Howard BV. Influence of obesity on the metabolism of apolipoprotein B in humans. The Journal of clinical investigation. 1985 Aug 1;76(2):596-603.
5. Fujioka S, Matsuzawa Y, Tokunaga K, Kawamoto T, Kobatake T, Keno Y, Kotani K, Yoshida S, Tarui S. Improvement of glucose and lipid metabolism associated with selective reduction of intra-abdominal visceral fat in premenopausal women with visceral fat obesity. International journal of obesity. 1991 Dec 1;15(12):853-9.
6. Yadav NK, Thanpari C, Shrewastwa MK, Mittal RK. Comparison of lipid profile in type-2 obese diabetics and obese non-diabetic individuals. A hospital based study from Western Nepal. Kathmandu University Medical Journal. 2012;10(3):44-7.
7. McGinnis JM, Foege WH. Actual causes of death in the United States. Jama. 1993 Nov 10;270(18):2207-12.
8. Bakshi AA, Bavikar JS, Asegaonkar SB, Bardapurkar JS, Domple V, Rai PS, Pawar S. Evaluation of usefulness of serum insulin as

Journal of Chemical Health Risks
www.jchr.org
JCHR (2025) 15(6), 1610-1614 | ISSN:2251-6727

sensitive predictor of cardiovascular dysfunction in obese individuals with normal lipid profile. Journal of Clinical and Diagnostic Research: JCDR. 2014 Oct 20;8(10):CC10.
9. Aziz J, Siddiqui NA, Siddiqui IA, Omair A. Relation of body mass index with lipid profile and blood pressure in young healthy students at Ziauddin Medical University. Journal of Ayub Medical College Abbottabad. 2003;15(4).
10. World Health Organization. Physical and overweight. Geneva: WHO; [date unknown]. Archived from the original on 2008 Dec 18. Retrieved 2009 Jan 10. Available from: https://www.who.int/health-topics/obesity
11. Zheng C, Liu Y, Xu C, Zeng S, Wang Q, Guo Y, Li J, Li S, Dong M, Luo X, Wu Q. Association between obesity and the prevalence of dyslipidemia in middle-aged and older people: an observational study. Scientific Reports. 2024 May 25;14(1):11974.
12. Bays HE, Toth PP, Kris-Etherton PM, Abate N, Aronne LJ, Brown WV, Gonzalez-Campoy JM, Jones SR, Kumar R, La Forge R, Samuel VT. Obesity, adiposity, and dyslipidemia: a consensus statement from the National Lipid Association. Journal of clinical lipidology. 2013 Jul 1;7(4):304-83.
13. Giudetti AM. Lipid metabolism in obesity. Frontiers in Physiology. 2023 Aug 31;14:1268288.
14. Vekic J, Stefanovic A, Zeljkovic A. Obesity and dyslipidemia: a review of current evidence. Current Obesity Reports. 2023 Sep;12(3):207-22.
15. Hierons SJ, Abbas K, Sobczak AI, Cerone M, Smith TK, Ajjan RA, Stewart AJ. Changes in plasma free fatty acids in obese patients before and after bariatric surgery highlight alterations in lipid metabolism. Scientific reports. 2022 Sep 12;12(1):15337.
16. Mongraw-Chaffin M, Foster MC, Anderson CA, Burke GL, Haq N, Kalyani RR, Ouyang P, Sibley CT, Tracy R, Woodward M, Vaidya D. Metabolically healthy obesity, transition to metabolic syndrome, and cardiovascular risk. Journal of the American College of Cardiology. 2018 May 1;71(17):1857-65.
17. Rey-López JP, De Rezende LF, Pastor-Valero M, Tess BH. The prevalence of metabolically healthy obesity: a systematic review and critical evaluation of the definitions used. Obesity reviews. 2014 Oct;15(10):781-90.
18. Shabana S, Ramya K, Sasisekhar TV, Santosh Kumar A. Hba1c as an index of glycemic status in obese type 2 diabetics. International Journal of Clinical and Biomedical Research. 2017 Jul 28:34-9.
19. Khan M, Khaleel M. Comparative study of serum lipid profile of obese and non-obese students (male) of Aljouf University. IJBAR. 2016 Jan 31;7(1):35-7.
20. Al-Jawadi AA, Priliani L, Oktavianthi S, Febinia CA, Daya M, Artika IM, Malik SG. Association of FTO rs1421085 single nucleotide polymorphism with fat and fatty acid intake in Indonesian adults. BMC Research Notes. 2021 Nov 7;14(1):411.
21. Gupta RD, Haider SS, Eusufzai SZ, Hoque Apu E, Siddika N. Differences in prevalence and associated factors of underweight and overweight/obesity among Bangladeshi adults by gender: Analysis of a nationally representative survey. International journal of environmental research and public health. 2022 Aug 27;19(17):10698.
22. Saraswati MR, Nugraha IBA, Suastika K. Similar Blood Glucose Pattern with Highest Peak at Minute 45 on Oral Glucose Tolerance Test Despite Higher Fasting Insulin and Insulin Resistance in Healthy Obese than Non-Obese Subject. Acta Med Indones. 2022 Apr;54(2):210-217.
23. Akter R, Nessa A, Husain MF, Wahed F, Khatun N, Yesmin M, Nasreen S, Tajkia T. Effect of Obesity on Fasting Blood Sugar. Mymensingh Med J. 2017 Jan;26(1):7-11.
24. Mahaur A. Comparison of lipid profile among obese and non-obese subjects: A biochemical study.
25. Taqiuddin R, Gupta H, Sufian Z, Kamaluddin K, El Rashid Mohamed YA, Khan A, Orfali HA, Lohana N, Ibrahim Mohamed MM, Mateen MA. Cardiovascular risk evaluation using lipid profile and blood pressure among obese and non-obese individuals in India. Bioinformation. 2024 Jul 31;20(7):723-727.
26. Alshuweishi Y, Almufarrih AA, Abudawood A, Alfayez D, Alkhowaiter AY, AlSudais H, Almuqrin AM. Patterns of Lipid Abnormalities in Obesity: A Comparative Analysis in Normoglycemic and Prediabetic Obese Individuals. Journal of Personalized Medicine. 2024 Sep 15;14(9):980.