International Journal of Creative and Open Research in Engineering and Management
ISSN: **3108-1754 (Online)**
Volume 02 Issue 07 Jul-2026 | Impact Factor: 3.5

"Postprandial Serum Triglyceride and Glucose Levels in Obese versus Non-Obese Young Adults: A Comparative Cross-Sectional Study"

Marli Ango[1], Avolu Kotso[1], Jackricky N Sangma[1] , Linanku Deori[2], Tanuja Deori[2], Philip Pegu[2], Yoezer Sony Tshomo[2], Babul Deori[2], Gunlu Gangmei[3] , Samir Moirangthem[1]*

1. Faculty of Allied & Healthcare Sciences, Assam down town University, Sankar Madhab Path, Gandhi Nagar, Panikhaiti, Guwahati, Assam, India, PIN 781026
2. BMLT Students of Allied & Healthcare Sciences,Assam down town University,Sankar Madhav Path, Gandhi Nagar, Panikhaiti, Gwahati, Assam, India, PIN-781026
3. Medical Lab.Technologist, Department of Pathology,AIIMS Bathinda, Punjab ,India PIN 151001

***Corresponding author: Samir Moirangthem,** Assistant Professor, Programme of Medical Laboratory Technology, Assam down town University
Email Id: samirthem1999@gmail.com

How to Cite this Article:

Ango, M., Kotso, A., Sangma, J. N., Deori, L., Deori, T., Pegu, P., Tshomo, Y. S., Deori, B., Gangmei, G. & Moirangthem, S. (2026). Postprandial Serum Triglyceride and Glucose Levels in Obese versus Non-Obese Young Adults: A Comparative Cross-Sectional Study. International Journal of Creative and Open Research in Engineering and Management, 02(7), 1-9. https://doi.org/10.55041/ijcope.v2i7.181

https://doi.org/10.55041/ijcope.v2i7.181

Background: Obesity is a major global health concern associated with dyslipidaemia, insulin resistance, and increased cardiovascular risk. Because individuals spend much of the day in a postprandial state, postprandial metabolic parameters may provide a more sensitive assessment of early metabolic dysfunction than fasting measurements.

Objective: To compare postprandial serum triglyceride and blood glucose concentrations between obese and non-obese young adults using Asian body mass index (BMI) criteria.

Methods: A comparative cross-sectional study was conducted among 52 participants (18–30 years) at Assam down town University, Guwahati, India. Participants were classified as obese (n = 24; BMI ≥25 kg/m²) or non-obese (n = 28; BMI 18.5–22.9 kg/m²). Postprandial blood glucose was measured by the glucose oxidase–peroxidase (GOD-POD) method, and serum triglycerides were determined using the glycerol phosphate oxidase (GPO) enzymatic method. Group comparisons were performed using Welch's independent-samples *t*-test, Mann–Whitney *U* test, and chi-square test.

Results: Mean postprandial glucose levels were comparable between obese and non-obese participants (86.52 ± 37.44 vs. 89.84 ± 35.13 mg/dL; $p = 0.74$). Serum triglyceride concentrations were numerically higher in the obese group (211.50 ± 166.28 vs. 195.75 ± 132.29 mg/dL), but the difference was not statistically significant ($p = 0.71$). Sensitivity analysis after excluding triglyceride outliers produced similar findings, and sex distribution did not differ significantly between groups.

Conclusion: Obese young adults demonstrated a non-significant trend toward higher postprandial triglyceride concentrations, whereas postprandial glucose levels were similar to those of non-obese individuals. Larger, adequately powered multicentre studies are needed to clarify the relationship between obesity and postprandial metabolic alterations.

Keywords: *Obesity; Body Mass Index; Postprandial Period; Blood Glucose; Triglycerides; Dyslipidaemia; Insulin Resistance; Young Adults.*

1. Introduction

Obesity is a chronic, multifactorial disease characterised by excessive accumulation of adipose tissue that adversely affects metabolic health [1]. It has emerged as a global epidemic driven by rapid urbanisation, sedentary living, and increasing consumption of energy-dense foods [2]. At the tissue level, obesity is marked by adipocyte hypertrophy and hyperplasia, altered secretion of adipokines such as leptin and adiponectin, and chronic low-grade inflammation characterised by elevated free fatty acids and pro-inflammatory cytokines including interleukin-1β, interleukin-6, tumour necrosis factor-α, and monocyte chemoattractant protein-1 [3,4,7]. This inflammatory milieu contributes to insulin resistance and endothelial dysfunction [4], while altered hepatic triglyceride synthesis and reduced lipid clearance raise circulating triglyceride concentrations [6]. Visceral adiposity, owing to its greater metabolic activity, is more strongly associated with cardiometabolic complications than subcutaneous fat [5].

The global prevalence of excess weight has more than doubled since 1980, with rising rates across both sexes and all age groups, although the magnitude varies by region and ethnicity [8]. The World Health Organization estimates that approximately 40% of adults are overweight and nearly 15% obese globally, with the highest burden in the American and European regions [9]. Sedentary lifestyles, reduced physical activity, and diets high in sugar and refined carbohydrates, layered upon genetic, endocrine, and environmental susceptibility, are considered principal drivers of this epidemic [10]. In India, obesity prevalence has risen rapidly and varies by geography, urbanicity, and lifestyle [11]. National survey data indicate that approximately 23% of women and 22% of men in India are overweight by BMI criteria [12], with abdominal obesity affecting more than half of individuals in some cohorts [13].

BMI, calculated as weight in kilograms divided by the square of height in metres, remains a simple, low-cost tool for population-level assessment of nutritional status and obesity-related disease risk, despite its inability to distinguish lean from fat mass or describe fat distribution [14,15]. Individuals with higher BMI carry an elevated risk of insulin resistance, dyslipidaemia, and cardiovascular disease [19]. Triglycerides, the principal storage form of dietary fat, are transported as chylomicrons and very-low-density lipoproteins; after a meal, triglyceride-rich lipoprotein concentrations rise, producing postprandial lipaemia [16,17]. Elevated postprandial triglycerides are an independent predictor of atherosclerosis and cardiovascular disease, and in some studies carry stronger predictive value than fasting measurements [17,18,19]. In obesity, impaired lipoprotein lipase activity and increased hepatic lipid output prolong postprandial triglyceride elevation [6]. Postprandial glucose, typically assessed roughly two hours after a meal, reflects insulin-mediated glucose disposal and is associated with oxidative stress, inflammation, and endothelial dysfunction when elevated, even in individuals with normal fasting glucose [23,25]. Because most people spend the majority of their waking hours in a postprandial state, these dynamic measures may be more clinically informative than fasting values alone [26,34].

Much of the existing evidence on postprandial dysmetabolism derives from older adults or individuals with established diabetes or dyslipidaemia. Comparable data describing postprandial glucose and triglyceride status specifically among young (18–30 years), apparently healthy adults stratified by regionally validated Asian BMI cut-offs remain limited, particularly within Indian university populations where sedentary behaviour and dietary transition are increasingly prevalent. This study aimed to compare postprandial glycaemic status and serum

triglyceride levels between obese and non-obese young adults, applying formal inferential statistical testing to the biochemical comparisons. It was hypothesised that obese individuals would demonstrate higher postprandial glucose and triglyceride concentrations than their non-obese counterparts.

2. Materials and Methods

2.1 Study design and setting

This was a comparative, cross-sectional, observational study conducted in the Department of Medical Laboratory Technology, Faculty of Allied and Healthcare Sciences, Assam down town University, Guwahati, Assam, India.

2.2 Study population and sampling

Participants comprised students and individuals affiliated with Assam down town University, aged 18–30 years, of both sexes, recruited by convenience sampling from the university population. Inclusion criteria were age 18–30 years and classification into a non-obese (BMI 18.5–22.9 kg/m²) or obese (BMI ≥25 kg/m²) group using Asian BMI criteria. Individuals with known diabetes mellitus or dyslipidaemia currently under treatment, and those below 18 years of age, were excluded. A formal a priori sample-size calculation was not performed; the achieved sample of 52 participants is noted as a limitation (Section 4.6).

2.3 Ethical considerations

Ethical clearance was obtained from the Ethical Clearance Committee of Assam down town University (**Memo No. ADTU/Ethics/Stdnt-Lett/2026/175**), with additional permission from the Dean, Faculty of Allied and Healthcare Sciences. Written informed consent was obtained from all participants prior to enrolment. Participant identity and contact information collected during the original data-collection exercise were excluded from this manuscript and from all analyses reported here; participants are referenced only by de-identified serial number, consistent with standard confidentiality practice for human-subjects research.

2.4 Data collection and anthropometry

Socio-demographic and lifestyle information (physical activity, personal and family history of diabetes, history of high cholesterol, dietary pattern, and prior glucose/cholesterol testing) was collected using a structured, interviewer-administered questionnaire. Height and weight were measured and BMI was calculated as weight (kg) divided by height (converted from feet to metres) squared, and participants were classified using Asian BMI cut-offs (non-obese 18.5–22.9 kg/m²; obese ≥25 kg/m²).

2.5 Sample collection and laboratory methods

Venous blood was collected by phlebotomy from the cubital vein into plain and sodium fluoride vacutainers, refrigerated prior to processing, and centrifuged to separate serum (Figure 8). Serum triglycerides were estimated by an enzymatic method (lipoprotein lipase–glycerol kinase–glycerol phosphate oxidase–peroxidase reaction) using a colorimetric endpoint assay read at 546 nm at 37 °C, with triglyceride concentration calculated as (absorbance of test / absorbance of standard) × 200 mg/dL. Postprandial blood glucose was estimated by the glucose oxidase–peroxidase (GOD-POD) method, with absorbance read against a reagent blank following incubation at 37 °C for 10 minutes, and glucose concentration calculated as (absorbance of test / absorbance of standard) × 100 mg/dL. Each assay run included a reagent blank and calibrated standard alongside test samples, consistent with routine internal quality-control practice for colorimetric assays.

2.6 Statistical analysis

Data were analysed in Python (pandas 2.x, SciPy 1.x). Continuous variables are reported as mean ± standard deviation. Between-group comparisons (obese vs non-obese) for continuous variables were made using Welch's independent-samples t-test (which does not assume equal variances) and cross-checked with the Mann–Whitney U test given the

wide standard deviations and modest group sizes. The chi-square test was used to compare the categorical distribution of sex across BMI groups. A sensitivity analysis for serum triglycerides was performed after excluding statistical outliers identified by the interquartile-range (IQR) method, to assess the influence of extreme values on the group comparison. Statistical significance was set at $\alpha = 0.05$ (two-tailed).

Fifty-two participants (n = 52) aged 18–28 years (recruitment criterion 18–30 years) were evaluated. Based on Asian BMI classification, 24 participants (46.2%) were categorised as obese (BMI ≥25 kg/m²) and 28 (53.8%) as non-obese (BMI 18.5–22.9 kg/m²) (Table 1; Figure 1). As expected given the classification criterion, mean BMI differed strongly between groups (31.00 ± 4.79 vs 22.71 ± 2.97 kg/m²; Welch t = 7.35, p < 0.001).

3. Results

Table 1. Baseline characteristics of study participants by BMI group (n = 52)

Characteristic	Non-obese (n = 28)	Obese (n = 24)	Test statistic	p-value
Age, years (mean ± SD)	22.57 ± 2.12	21.75 ± 2.01	t = 1.45	0.157
BMI, kg/m² (mean ± SD)	22.71 ± 2.97	31.00 ± 4.79	t = 7.35	< 0.001
Sex, male / female, n	17 / 11	13 / 11	$\chi^2 = 0.04$	0.845

SD = standard deviation. Age and BMI compared with Welch's independent-samples t-test; sex distribution compared with the chi-square test.

3.1 Demographic characteristics

Of the 52 participants, 30 (57.7%) were male and 22 (42.3%) female, and sex distribution was closely similar across BMI groups (Table 1; Figure 3). Participant age ranged from 18 to 28 years and did not differ significantly between groups (22.57 ± 2.12 vs 21.75 ± 2.01 years; p = 0.157) (Figure 2). By design, BMI differed markedly between groups (Figure 1).

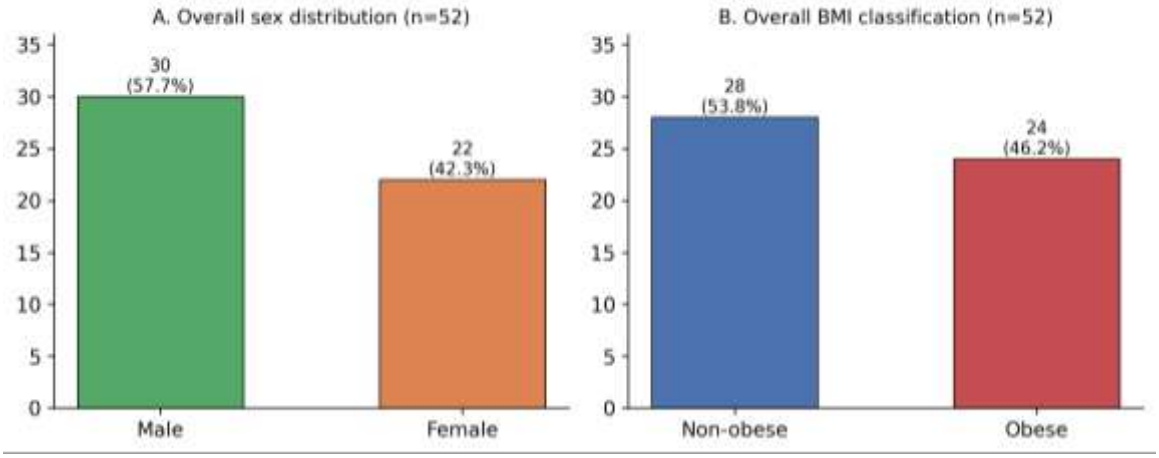


Figure 1. Overall sex distribution (A) and Asian BMI classification (B) of the study cohort (n = 52).

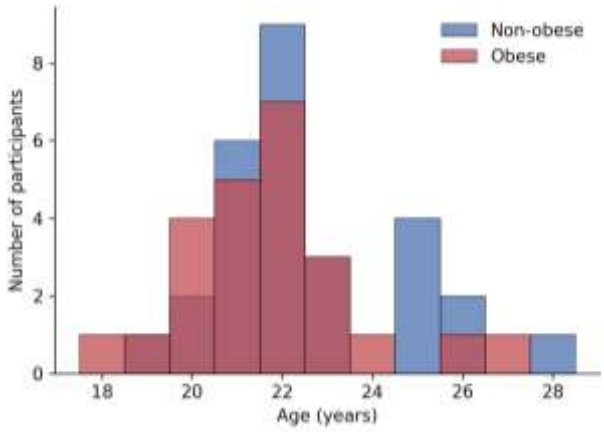


Figure 2. Age distribution of participants by BMI group.

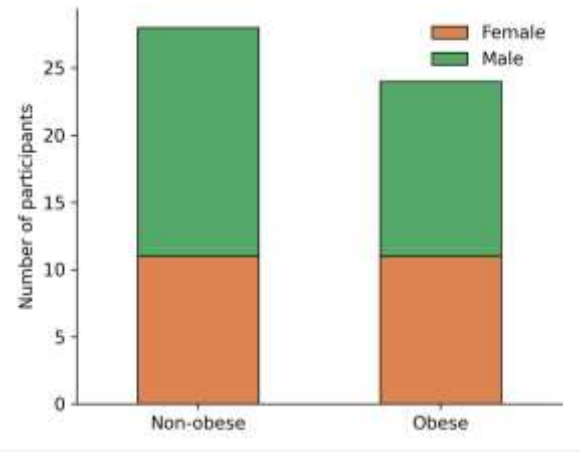


Figure 3. Sex distribution within non-obese and obese groups.

3.2 Postprandial biochemical comparison

Mean postprandial glucose was 86.52 ± 37.44 mg/dL among obese participants (n = 24) and 89.84 ± 35.13 mg/dL among non-obese participants (n = 28); the mean difference of −3.32 mg/dL was not statistically significant (Welch t = −0.33, p = 0.74; Mann–Whitney U p = 0.73) (Table 2; Figures 4–5). Mean serum triglycerides were 211.50 ± 166.28 mg/dL among obese participants and 195.75 ± 132.29 mg/dL among non-obese participants; the mean difference of +15.75 mg/dL was likewise not statistically significant (Welch t = 0.37, p = 0.71; Mann–Whitney U p = 0.66) (Table 2; Figures 4–5). After exclusion of statistical outliers in the triglyceride distribution using the IQR method (four values excluded), the between-group difference remained non-significant (171.6 vs 168.4 mg/dL; p = 0.90), indicating the primary result was not driven by extreme values. Figure 5 displays individual participant values as box plots with overlaid data points, which makes visible the wide spread and outliers (e.g. one triglyceride value of 800 mg/dL) that the summary means in Figure 4 do not show.

Table 2. Comparison of postprandial glucose and serum triglyceride levels between BMI groups

Parameter	Non-obese (n = 28) Mean ± SD	Obese (n = 24) Mean ± SD	Mean difference	Welch t	p-value	Mann–Whitney p
Postprandial glucose (mg/dL)	89.84 ± 35.13	86.52 ± 37.44	−3.32	−0.33	0.744	0.734
Serum triglycerides (mg/dL)	195.75 ± 132.29	211.50 ± 166.28	+15.75	0.37	0.711	0.660

SD = standard deviation. Neither comparison reached statistical significance at α = 0.05.

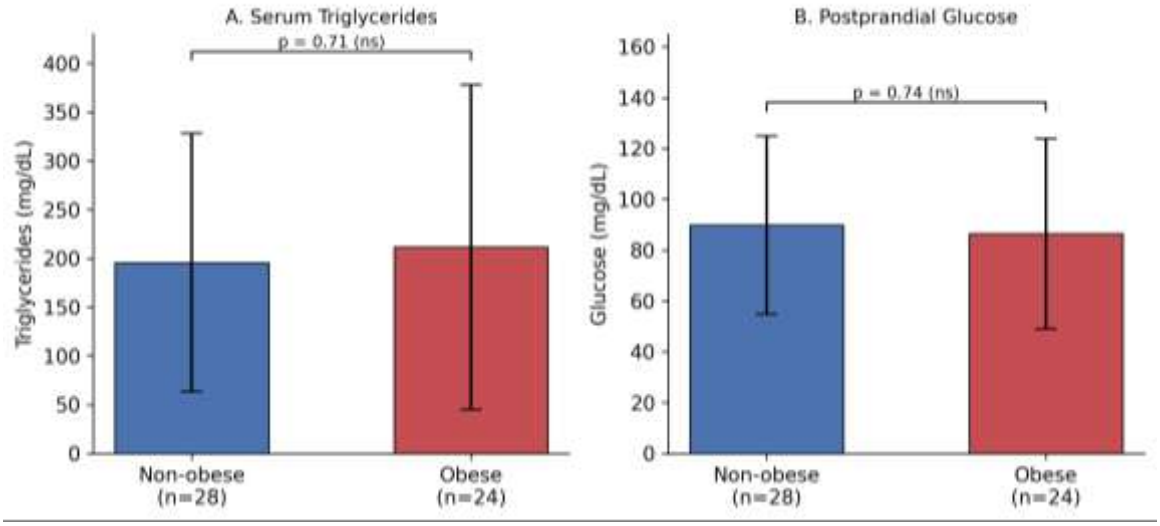


Figure 4. Postprandial serum triglycerides (A) and postprandial glucose (B) by BMI group, mean ± SD. Neither between-group difference was statistically significant (ns).

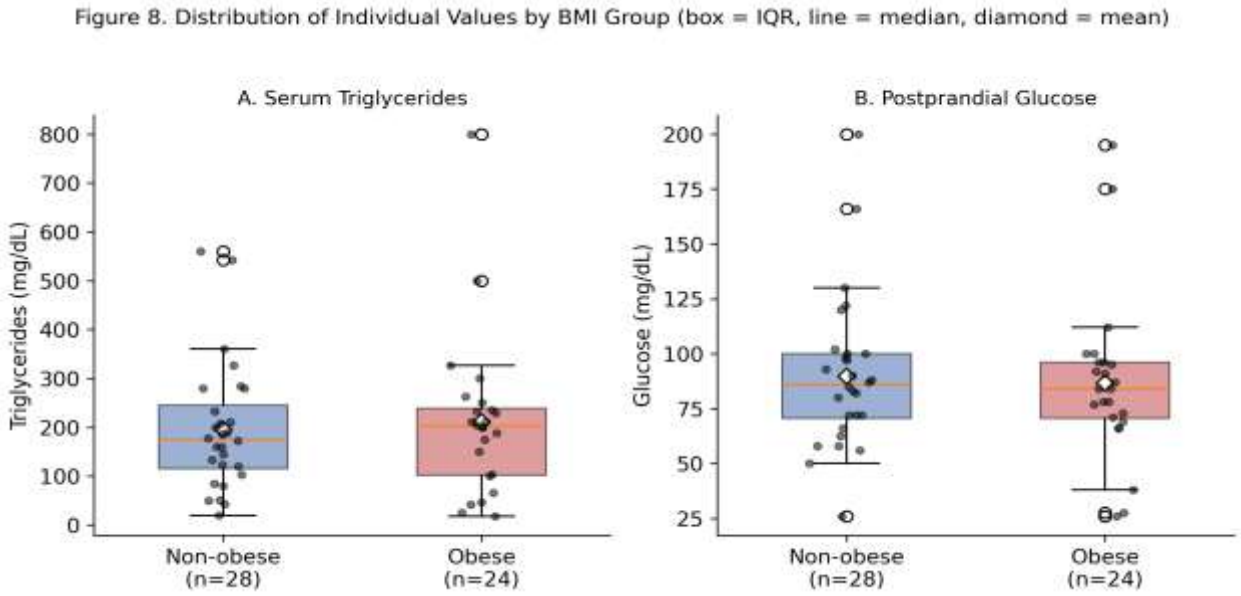


Figure 5. Distribution of individual postprandial triglyceride (A) and glucose (B) values by BMI group. Box = interquartile range, horizontal line = median, diamond = mean, dots = individual participants.

3.3 Relationship between BMI and biochemical parameters

Across the full cohort (n = 52), BMI was not significantly correlated with postprandial glucose (Pearson r = −0.11, p = 0.44) or with serum triglycerides (r = −0.11, p = 0.45) (Figure 6), consistent with the group-level comparisons in Table 2 and reinforcing that a linear dose–response relationship between adiposity and either biochemical parameter was not detectable in this sample.

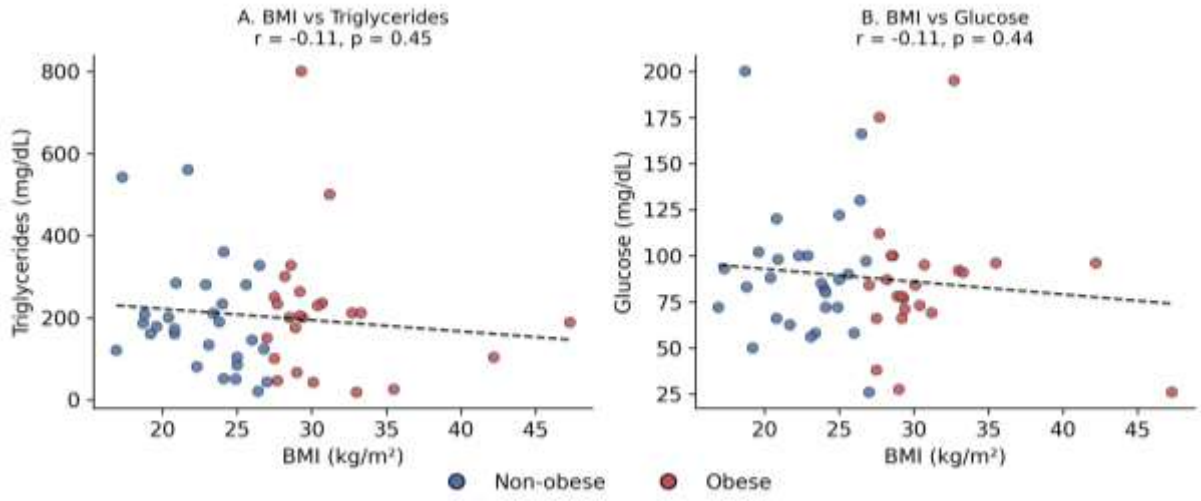


Figure 6. Scatter plots of BMI versus serum triglycerides (A) and postprandial glucose (B), with linear fit (dashed line). Each point represents one participant.

4. Discussion

4.1 Principal findings

In this comparative cross-sectional study of 52 young adults, obese participants showed a numerically higher mean postprandial serum triglyceride concentration than non-obese participants (211.50 vs 195.75 mg/dL), and postprandial glucose concentrations were closely comparable between the two groups (86.52 vs 89.84 mg/dL). With formal inferential testing applied, neither difference reached statistical significance, and the triglyceride result was stable to a sensitivity analysis excluding outliers. Sex distribution did not differ meaningfully between groups, reducing the likelihood that sex confounded the biochemical comparisons.

4.2 Comparison with previous studies

The direction of the triglyceride difference observed here (higher in obesity) is broadly consistent with prior work describing postprandial hypertriglyceridaemia as a sensitive marker of early lipid and cardiometabolic disturbance, in some studies exceeding fasting measurements in predictive value [17,18]. Studies in Indian populations have similarly linked postprandial hypertriglyceridaemia with hyperleptinaemia and incipient insulin resistance [32], and have shown that postprandial lipid responses differ across glycaemic status even before overt diabetes develops [33]. However, unlike some of this prior literature, the present comparison did not reach statistical significance, most plausibly reflecting the modest sample size and wide inter-individual variability (SDs comparable to or exceeding the group means) rather than a true absence of biological effect. The near-identical postprandial glucose values between groups are consistent with reports that glucose homeostasis can remain preserved in early or uncomplicated obesity despite adiposity-related lipid changes, particularly in younger cohorts [28,29].

4.3 Biological plausibility

Mechanistically, obesity is thought to elevate postprandial triglycerides through increased hepatic triglyceride synthesis, greater circulating free fatty acid flux from expanded adipose stores, and reduced peripheral lipoprotein lipase-mediated clearance of triglyceride-rich lipoproteins [6,17]. The same free-fatty-acid and inflammatory pathways are implicated in impaired insulin signalling [37], but their glycaemic consequences may become measurable only after a longer

duration of metabolic stress than lipid changes require. This offers one plausible explanation, consistent with though not proven by the present data, for why a numerical triglyceride difference was observed without an accompanying glucose difference in this young cohort.

4.4 Clinical and public health relevance

If confirmed in larger samples, these patterns would support the view that postprandial lipid screening may help identify at-risk young adults earlier than glucose-based screening alone. Given the descriptive, non-significant nature of the present comparisons, this should currently be regarded as a hypothesis for future testing rather than an evidence-based screening recommendation.

4.5 Strengths

The study benefits from a well-defined, homogeneous age range, use of regionally validated Asian BMI cut-offs for classification, direct biochemical measurement of both postprandial glucose and triglycerides using standard enzymatic/colorimetric methods, formal inferential statistical testing with a non-parametric cross-check, and a sensitivity analysis for outlier influence.

4.6 Limitations

- The sample size (n = 52) was modest and no a priori power calculation was performed; the study may be underpowered to detect the group differences it was designed to test, and the non-significant findings should not be interpreted as evidence of no effect.
- The study was restricted to a narrow age band (18–30 years) and a single institutional setting, limiting generalisability.
- Additional biochemical markers relevant to insulin resistance (fasting insulin, HbA1c, HOMA-IR, full fasting lipid profile) were not measured.
- Convenience sampling was used, raising the possibility of selection bias.
- Socio-demographic and lifestyle data (exercise, family history of diabetes, dietary pattern) were available only as cohort-level summary figures from the original data-collection exercise and could not be linked to individual biochemical results or independently re-analysed for this manuscript; authors should supply the underlying line-level responses if these associations are to be reported with formal statistical support.
- Meal timing and composition prior to postprandial sampling were not standardised or reported in the available records; this should be clarified by the study team.
- Several triglyceride values were markedly elevated (maximum 800 mg/dL) and a small number of glucose values were unusually low (minimum 26 mg/dL); while retained in the primary analysis and confirmed not to drive the main conclusions (Section 3, sensitivity analysis), these warrant verification against original assay records for transcription or analytical error.

4.7 Future research directions

Larger, adequately powered studies incorporating multivariable adjustment for confounders, additional markers of insulin resistance (fasting insulin, HOMA-IR, HbA1c, triglyceride-glucose index), standardised postprandial test-meal protocols, and longitudinal follow-up are warranted to clarify whether postprandial lipid disturbance precedes glycaemic dysregulation in young obese adults.

5. Conclusion

In this modest cross-sectional sample of young Indian adults, obese participants showed numerically, but not statistically significantly, higher postprandial serum triglyceride concentrations than non-obese peers, while

postprandial glucose concentrations were closely comparable between groups. These descriptive and preliminary inferential findings are best regarded as hypothesis-generating; larger, adequately powered studies with standardised protocols and additional metabolic biomarkers are needed before they can inform clinical or public health screening recommendations.

Acknowledgements

The authors thank the participants who volunteered for sample collection and questionnaire completion, and acknowledge the support of the Faculty of Allied and Healthcare Sciences, Assam down town University.

Funding

No external funding was reported for this study (to be confirmed by authors).

Conflict of interest

The authors declare no conflict of interest.

Ethical approval

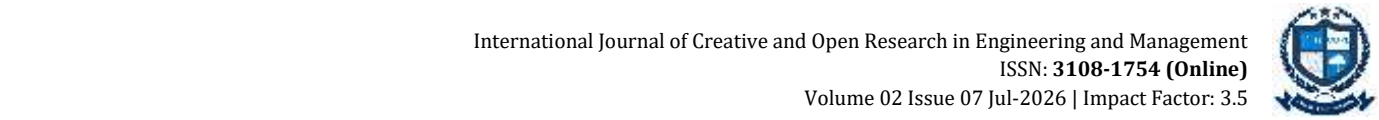
This study was approved by the Ethical Clearance Committee of Assam down town University(**Memo No. ADTU/Ethics/Stdnt-Lett/2026/175**).

Data availability

The de-identified dataset analysed during the current study is available from the corresponding author on reasonable request.

Author contributions

Conceptualization: Marli Ango. Methodology: Marli Ango. Investigation: Linanku Deori, Tanuja Deori, Philip Pegu, Yoezer Sony Tshomo, Babul Deori. Data curation: Marli Ango Formal analysis: Marli Ango , Samir Moirangthem . Writing – original draft:Marli Ango . Writing – review & editing: Marli Ango, Samir Moirangthem. Supervision: Marli Ango.



Supplementary Figures: Study Workflow

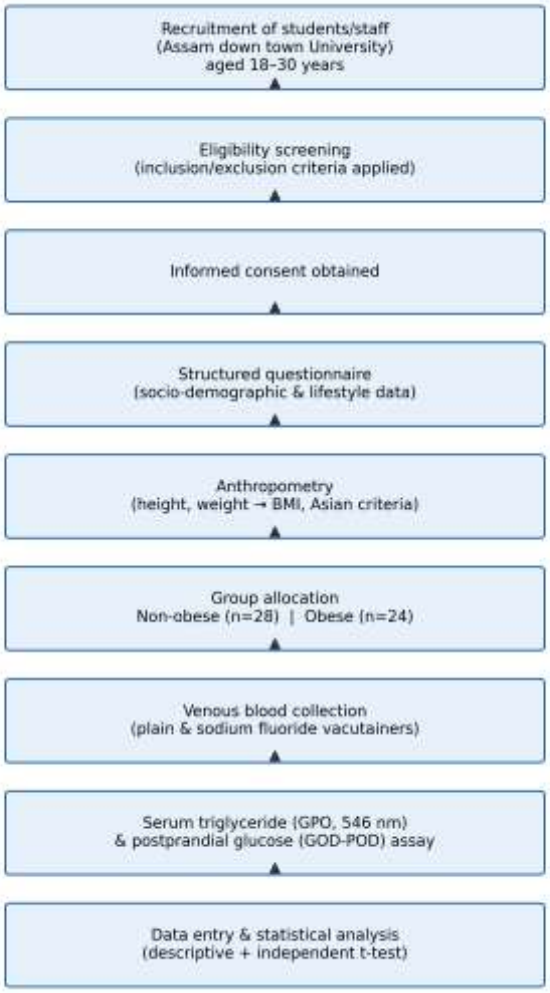


Figure 7. Overall study workflow, from recruitment through data analysis.

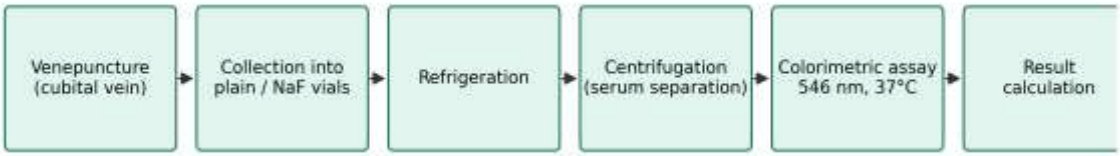


Figure 8. Sample processing flowchart, from venepuncture to biochemical result.

References

1. Bays HE, Kirkpatrick CF, Maki KC, Toth PP, Morgan RT, Tondt J, Christensen SM, Dixon DL, Jacobson TA. Obesity, dyslipidemia, and cardiovascular disease: a joint expert review from the Obesity Medicine Association and the National Lipid Association 2024. *J Clin Lipidol.* 2024;18(3):e320-e350. doi:10.1016/j.jacl.2024.04.001.
2. World Health Organization. *Obesity and overweight* [Internet]. Geneva: World Health Organization; 2024 [cited 2026 Jul 15]. Available from: https://www.who.int/news-room/fact-sheets/detail/obesity-and-overweight.
3. Shahamati D, Akhavan NS, Rosenkranz SK. Postprandial inflammation in obesity: dietary determinants, adipose tissue dysfunction, and the gut microbiome. *Biomolecules.* 2025;15(11):1516.
4. Saltiel AR, Olefsky JM. Inflammatory mechanisms linking obesity and metabolic disease. *J Clin Invest.* 2017;127(1):1-4.
5. Neeland IJ, Poirier P, Després JP. Cardiovascular and metabolic heterogeneity of obesity: clinical challenges and implications for management. *J Am Coll Cardiol.* 2018;72(12):1361-1375.
6. Wilson SM, Maes AP, Yeoman CJ, Walk ST, Miles MP. Determinants of the postprandial triglyceride response to a high-fat meal in healthy overweight and obese adults. *Lipids Health Dis.* 2021;20(1):107.
7. Gkrinia EMM, Belančić A. The mechanisms of chronic inflammation in obesity and potential therapeutic strategies: a narrative review. *Curr Issues Mol Biol.* 2025;47(5):357.
8. Lin X, Li H. Obesity: epidemiology, pathophysiology, and therapeutics. *Front Endocrinol (Lausanne).* 2021;12:706978.
9. Omer T. The causes of obesity: an in-depth review. *Adv Obes Weight Manag Control.* 2020;10(4):90-94.
10. Boutari C, Mantzoros CS. A 2022 update on the epidemiology of obesity and a call to action: as its twin COVID-19 pandemic appears to be receding, the obesity and dysmetabolism pandemic continues to rage on. *Metabolism.* 2022;133:155217.
11. Ahirwar R, Mondal PR. Prevalence of obesity in India: a systematic review. *Diabetes Metab Syndr.* 2019;13(1):318-321.
12. Chaudhary M, Sharma P. Abdominal obesity in India: analysis of the National Family Health Survey-5 (2019-2021) data. *Lancet Reg Health Southeast Asia.* 2023;14:100208. doi:10.1016/j.lansea.2023.100208.
13. Gupta RD, Swaminathan S, Minocha S, et al. Obesity and abdominal obesity in the Indian population: findings from a nationally representative study of 698,286 participants. *Epidemiologia.* 2023;4(2):163-172.
14. Oussaada SM, van Galen KA, Cooiman MI, Kleinendorst L, Hazebroek EJ, van Haelst MM, et al. The pathogenesis of obesity. *Metabolism.* 2019;92:26-36.
15. Purnell JQ. What is obesity? Definition as a disease, with implications for care. *Gastroenterol Clin North Am.* 2023;52(2):261-275.
16. Wu J, He L. The relationship between body mass index and physical activity participation rate. *J Sens.* 2022;2022:8744880.
17. Kolovou GD, Watts GF, Mikhailidis DP, Pérez-Martínez P, Bilianou H, Panotopoulos G, et al. Postprandial hypertriglyceridaemia revisited in the era of non-fasting lipid profile testing: a 2019 expert panel statement. *Curr Vasc Pharmacol.* 2019;17(5):498-514.
18. Nordestgaard BG, Benn M, Schnohr P, Tybjærg-Hansen A. Nonfasting triglycerides and risk of myocardial infarction, ischemic heart disease, and death in men and women. *JAMA.* 2007;298(3):299-308.
19. Mikkelsen M, Hansen CP, Bergholdt HKM, et al. Postprandial triglycerides and cardiovascular disease risk. *Front Nutr.* 2023;10:1240083.
20. Borén J, Packard CJ, Taskinen MR. The role of triglyceride-rich lipoproteins in atherosclerotic cardiovascular disease. *Eur Heart J.* 2020;41(1):99-109.
21. Goyal R, Singhal M, Jialal I. Type 2 diabetes. In: *StatPearls* [Internet]. Treasure Island (FL): StatPearls Publishing; 2025.
22. American Diabetes Association. Diagnosis and classification of diabetes mellitus. *Diabetes Care.* 2010;33(Suppl 1):S62-S69.

23. Thota RN, Moughan PJ, Garg ML. Significance of postprandial insulin and triglycerides to evaluate the metabolic response of composite meals differing in nutrient composition: a randomized cross-over trial. *Front Nutr.* 2022;9:816755.
24. American Diabetes Association Professional Practice Committee. Standards of Care in Diabetes—2024. *Diabetes Care.* 2024;47(Suppl 1):S1-S350.
25. Ceriello A, Colagiuri S, Gerich J, Tuomilehto J. Guideline for management of postmeal glucose. *Diabetes Care.* 2008;31(7):1516-1524.
26. Orringer CE, Jacobson TA, Saseen JJ, Brown AS, Gotto AM Jr, Ross JL, et al. Update on the use of nonfasting lipid testing: scientific statement from the National Lipid Association. *J Clin Lipidol.* 2019;13(5):623-634.
27. Indira V, et al. Comparative study on postprandial lipid profile in diabetes mellitus. *Healthcare Bulletin.* 2024.
28. Hiyoshi T, Fujiwara M, Yao Z, et al. Postprandial hyperglycemia and postprandial hypertriglyceridemia in type 2 diabetes. *J Biomed Res.* 2019;33(1):1-16.
29. Shibib K, Bhat OA, et al. Manipulation of postprandial hyperglycaemia in type 2 diabetes: an update. *Diabetes Metab Syndr Obes.* 2024;17:299-312.
30. Tang Y, Wang L, Zhang Y, et al. Triglyceride-glucose index and its association with insulin resistance: a systematic review and meta-analysis. *Front Endocrinol (Lausanne).* 2024;15.
31. Boushehri YG, Meymanatabadi Z, Tanha AE, Azami P, Alaei M, Alamdari AA, et al. Association of triglyceride glucose-body mass index (TyG-BMI) with metabolic dysfunction-associated steatotic liver disease: a systematic review and meta-analysis. *PLoS One.* 2025;20(8):e0324483. doi:10.1371/journal.pone.0324483.
32. Aslam M, Madhu SV, Sharma KK, Sharma AK, Galav V. Hyperleptinaemia and its association with postprandial hypertriglyceridemia and glucose intolerance. *Indian J Endocrinol Metab.* 2021;25(5):443-449. doi:10.4103/ijem.ijem_393_21.
33. Madhu SV, Bhardwaj S, Mishra BK, Aslam M. Total cholesterol and postprandial triglyceride levels as early markers of GDM in Asian Indian women. International Journal of Diabetes in Developing Countries. 2022 Oct;42(4):630-5.
34. Bansal S, Buring JE, Rifai N, Mora S, Sacks FM, Ridker PM. Fasting compared with nonfasting triglycerides and risk of cardiovascular events in women. *JAMA.* 2007;298(3):309-316.
35. Monnier L, Colette C. Glycemic variability and its contribution to oxidative stress in diabetes: a review. *Diabetes Metab.* 2008;34(Suppl 2):S120-S127.
36. DeFronzo RA, Ferrannini E, Groop L, Henry RR, Herman WH, Holst JJ, et al. Type 2 diabetes mellitus. *Nat Rev Dis Primers.* 2015;1:15019.
37. Gastaldelli A, Ferrannini E. Pathophysiology of Prediabetes: Role of Lipotoxicity?. InGlobal health perspectives in prediabetes and diabetes prevention 2014 (pp. 31-47).
38. Matteo Ciccone M, Scicchitano P, Cameli M, Cecere A, Mattioli AV. Endothelial function in pre-diabetes, diabetes and diabetic cardiomyopathy: a review. Journal of Diabetes & Metabolism. 2014;5:1-0.
39. Ferrannini E. Physiology of glucose homeostasis and insulin therapy in type 1 and type 2 diabetes. Endocrinology and Metabolism Clinics. 2012 Mar 1;41(1):25-39.