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HOMA-IR and HOMA-Beta as Predictors of Metabolic Syndrome: Associations with Cardio-Metabolic risk Factors Across Distinct Obesity Phenotypes in Young Adults from Central India.

Shipra Das[1,3], Anil Baran Choudhury[2], Sanjay Kumar[3]
[1]Department of Physiology, Bharat Ratna Late Shri Atal Bihari Vajpayee Memorial Medical College, Rajnandgaon, Chhattisgarh, India.
[2]Department of Biochemistry, Bharat Ratna Late Shri Atal Bihari Vajpayee Memorial Medical College, Rajnandgaon, Chhattisgarh, India.
[3]Department of Physiology, Sikkim Manipal Institute of Medical Sciences, Sikkim Manipal University, Gangtok, Sikkim, India.

Email: shipra.ds@rediffmail.com

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ABSTRACT

Introduction: Metabolic syndrome (MetS) is a growing health hazard, characterized by central adiposity, hypertension, dyslipidaemia, and hyperglycaemia. This study assesses the predictive utility of HOMA-IR and HOMA-Beta (β) for MetS among young adults. Methods: A cross-sectional study was conducted with 403 college going young adults, categorized into four obesity phenotypes: Metabolically Healthy Normal Weight (MHNW), Metabolically Healthy Obese (MHO), Metabolically Unhealthy Normal Weight (MUNW), and Metabolically Unhealthy Obese (MUO). Correlations of HOMA-IR and HOMA- β with cardiometabolic parameters were assessed by using Pearson's correlation. Logistic regression, and ROC analysis were done to determine predictive accuracy of HOMA-IR and HOMA- β for MetS. Results: HOMA-β exhibited better predictive accuracy for MetS than HOMA-IR. MUO had significantly lower HOMA-Beta (75.28 ± 13.35) than MHNW (123.03 ± 72.21). MHO showed well-maintained β-cell function (HOMA-β: 103.73 ± 38.60) despite higher HOMA-IR (2.03 ± 0.61). HOMA-β correlated negatively with fasting blood glucose (r = -0.89, MHO; r = -0.59, MUO), while HOMA-IR correlations were weaker (r = 0.17 to 0.35). ROC analysis showed HOMA-β (AUC = 0.77) outperformed HOMA-IR (AUC = 0.38), insulin levels (AUC = 0.49), and HbA1c (AUC = 0.25). Conclusion: HOMA- β is a superior predictor of MetS, highlighting β-cell function over insulin resistance in young adults. Early detection and phenotype-based interventions are crucial for prevention.

INTRODUCTION

Clustering of cardio-metabolic risk factors, including central adiposity, hypertension, hyperglycemia, and high triglycerides (TG) with low high-density lipoprotein (HDL) cholesterol levels, is termed as Metabolic syndrome (MetS). Predominant driving force behind occurrence of MetS can be obesity. Obesity is commonly predictable as a risk factor for type 2 diabetes mellitus (T2DM). Additionally, it is crucial to understand how non-obese individuals with similar

Journal of Molecular Science
Volume 35 Issue 2, Year of Publication 2025, Page 238-245
DoI-17.4687/1000-9035.2025.033

insulin sensitivity or beta cell functionality like their obese counterparts in terms of T2DM risk are exist. Contrastingly, existence of obese individuals with normal insulin sensitivity and less risk of T2DM, particularly among Asian adults, is not well understood.[1,2] Its essential to understand the interplay among β-cell functionality, insulin resistance (IR) and cardiometabolic risk factors to address the complex patho-physiology underlying metabolic dysfunction across diverse obesity phenotypes. Previous research reveals that β-cell dysfunction is a critical factor in the progression of T2DM and its associated complications, while IR significantly contributes to MetS and cardiovascular risks among adult population.[3,4] Numerous studies have confirmed significant associations among these markers and metabolic syndrome components, emphasizing their prognostic utility.[5,6] This study was aimed to provide novel insights into the roles of HOMA-β and HOMA-IR as predictors of MetS and explore the correlation between those markers with cardio-metabolic risk factors across distinct phenotypic young adults from Central India, demographically unique and understudied population. By identifying significant associations between these markers and cardiometabolic risk factors, it highlights the importance of early detection and targeted interventions to mitigate risks of metabolic syndrome.

MATERIALS AND METHODS:

Study Design: This is a cross-sectional study which was performed according to STROBE guidelines. College-going students of both genders aged 18–25 years were included for this study from Rajnandgaon, a district of Chhattisgarh central India.

Sampling Technique: A multi-stage sampling technique was implemented for this study. Representation of both urban and rural young adult populations were ensured. The process involved:

1. **Geographical Stratification**: Urban and rural areas in the district were identified to account for socio-economic and lifestyle differences.
2. **Cluster Selection**: Five urban and five rural clusters were randomly chosen for participant recruitment.
3. **Institutional Sampling**: Within selected clusters, educational institutions were randomly selected, and college-going students were systematically enrolled for this study. A total of 403 participants of both male and female were recruited, who were meeting inclusion criteria (18–25 years, non-smoker, non-alcoholic, no chronic metabolic diseases, willing for participation). Exclusion criteria included pregnancy, lactation, medication affecting metabolism, chronic systemic illnesses or undergoes any surgical methods within last six months.

Metabolic Syndrome Definition: Metabolic syndrome (MetS) was defined based on the modified criteria of the International Diabetes Federation (IDF) for South Asian populations.[7,8] Participants were identified as having MetS if they exhibited at least three criteria among following five components: 1. **Abdominal Obesity**: Waist circumference ≥90 cm for men and ≥80 cm for women. 2. **Elevated Triglycerides**: TG ≥150 mg/dL 3. **Reduced HDL Cholesterol**: HDL <40 mg/dL for men and <50 mg/dL for women. 4. **Elevated Blood Pressure**: Systolic BP ≥130 mmHg or diastolic BP ≥85 mmHg. 5. **Elevated Fasting Glucose**: FBG ≥100 mg/dL. **Obesity Phenotype distribution:** Participants were categorized into four phenotypes based on BMI and accordance of metabolic syndrome: 1. Metabolically Healthy Normal Weight (MHNW), 2. Metabolically Healthy Obese (MHO), 3. Metabolically Unhealthy Normal Weight (MUNW), and 4. Metabolically Unhealthy Obese (MUO).[9]

Data Collection and biochemical analysis methods: Demographics and lifestyle data were collected by using structured questionnaires. Anthropometric measurements like Height, weight, BMI (classified based on the revised consensus guidelines for India), waist circumference (WC), and hip circumference (HC) were measured by following standard protocol.[10] Blood Pressure was Measured using an automated sphygmomanometer after the participant had rested for at least 5 minutes. Biochemical Analysis like Fasting blood glucose (FBG), lipid profiles (TG, HDL), HbA1C, fasting serum Insulin (FSI) level were assessed from venous blood samples collected after an overnight fast of 8-12 hours by using fully automated biochemistry analyzer (Beckman Coulter-Au680). HOMA-IR and HOMA-β were calculated by using those formula HOMA-IR= FBG (mmol/L) *FSI(Mu/ml)/22.5, and HOMA-β =20*FSI(Mu/ml)/ [FBG (mmol/L)-3.5] respectively. [11,12]

Statistical Analysis: Descriptive statistics summarized population characteristics. Pearson's correlations and logistic regression models examined relationships and predictors of metabolic outcomes. Statistical analysis was done by using statisty software and graphical representation was generated by using google colab.

RESULTS:

The study included 403 participants, with 63.56%

Journal of Molecular Science
Volume 35 Issue 2, Year of Publication 2025, Page 238-245
DoI-17.4687/1000-9035.2025.033

residing in urban areas and 36.44% in rural regions. Among urban participants, 58.2% were female and 41.8% were male, while in rural areas, 48.1% were female and 51.9% were male. Regarding lifestyle factors, 52.6% engaged in regular physical activity, while 47.4% did not. Dietary habits showed 38.2% were vegetarian and 61.8% were non-vegetarian. Family history of metabolic disorders was reported as follows: hypertension (26.8%), diabetes (18.3%), and dyslipidemia (14.6%). The results are summarized in the following tables (table no. 1-3) and figures (Fig no.1-3):

The results represented in Table 1 highlight distinct metabolic profiles across the four obesity phenotypes, highlighting the critical role of β-cell function in metabolic health. While HOMA-IR is highest in the MHO group, suggesting increased insulin resistance, their relatively preserved β-cell function (HOMA-β: 103.73 ± 38.60) indicates a compensatory mechanism that may protect against metabolic dysfunction. In contrast, MUO individuals exhibit the lowest HOMA-β values (75.28 ± 13.35) and reduced insulin levels (7.74 ± 1.16), suggesting significant β-cell dysfunction, which aligns with their poorer glycemic control (HbA1c: 5.52 ± 0.43).

Table 2 indicates that lower β-cell function (HOMA-β) quartiles correlate strongly with adverse cardiometabolic profiles, including higher fasting glucose, blood pressure, and waist circumference. In contrast, insulin resistance (HOMA-IR) quartiles show less consistent associations. This highlights β-cell dysfunction as a critical early indicator of metabolic risk in young adults.

The logistic regression analysis in Table 3 indicates significant metabolic predictors associated with metabolic syndrome (MetS) in young adults. Among the evaluated markers, HOMA-Beta demonstrated a significant positive association (coefficient = 0.09, $p < 0.001$), suggesting that each unit increase in β-cell function increases the odds of MetS. Conversely, fasting serum insulin (FSI) showed a significant negative association (coefficient = -1.13, $p < 0.001$), implying reduced odds of MetS with elevated insulin levels. Interestingly, neither HOMA-IR nor HbA1c reached statistical significance, highlighting that insulin resistance and glycaemic control, traditionally regarded as prominent predictors, might have limited predictive utility in young adult populations compared to β-cell function.

DISCUSSION:

Observation of cardio-metabolic profiles across obesity phenotypes among young adults displays a complex interplay between obesity and metabolic wellbeing. Remarkably MHO individuals show higher HOMA-IR levels compared to their MHNW counterparts, suggesting an inherent insulin resistance often linked with obesity. Though, their well-maintained HOMA-BETA and insulin levels indicate a compensatory β-cell function that capable of conserving glucose homeostasis, a phenomenon that corroborates findings by Primeau *et al.*, who noted a similar metabolic resilience in MHO individuals.[13] Contrastingly, MUNW individuals display metabolic profiles resembling those of Metabolically Unhealthy Obese (MUO) individuals, characterized by compromised β-cell function and elevated HOMA-IR. This indicates significant metabolic risks independent of excess body fat, supporting study by Stefan *et al.*, which identified metabolic dysfunction in non-obese individuals.[14] These findings challenge the conventional perspective that links obesity directly with metabolic diseases and emphasize the need for a nuanced approach to assess metabolic health that goes beyond body weight. These diverse metabolic disturbances seen in MHO and MUNW phenotypes is highlighting the variability in metabolic responses among distinct individuals which underscore the complexity of metabolic regulation in obesity. Kahn and Flier's report shows the broad spectrum of insulin resistance in obesity which aligning with the higher insulin resistance observed in our MHO group despite their relatively stable β-cell function.[15] Furthermore, pronounced β-cell dysfunction observed in MUO phenotypes supports the "lipotoxicity" theory proposed by Unger and Zhou, where chronic exposure to high free fatty acids leads to β-cell impairment.[16] To understand this pathophysiology is important to develop effective therapeutic interventions targeted at specific metabolic profiles for improving the management strategies of metabolic dysfunctions and potentially delaying or preventing the onset of type 2 diabetes in young adults.

Our report demonstrations a detailed breakdown of cardiometabolic risk factors distributed across three quartiles of HOMA-β and HOMA-IR among a young adult population. This analysis helps explicate the relationships between insulin resistance and β-cell functionality with various cardiometabolic components, including waist circumference (WC), systolic blood pressure (SBP), diastolic blood pressure (DBP), fasting blood glucose (FBG), triglycerides (TG), and high-density lipoprotein cholesterol (HDL). Our findings from young adults indicate that higher levels of insulin resistance (HOMA-IR) correlate with adverse cardiometabolic markers such as increased waist circumference and elevated triglyceride levels, while reduced β-cell function (HOMA-β) is

Journal of Molecular Science
Volume 35 Issue 2, Year of Publication 2025, Page 238-245
DoI-17.4687/1000-9035.2025.033

associated with higher fasting blood glucose and lower HDL cholesterol. Similar relationships, demonstrated by Matthews D R *et al.* (1985), which also highlight the impact of insulin resistance on metabolic syndrome components.[17] However, contrasting studies like that of Ferrannini E *et al.* (2005) suggest that insulin resistance may have a less pronounced effect on HDL cholesterol in different populations, representing that metabolic responses can vary significantly by demographic characteristics.[18] Additionally, Kahn B B and Flier J S (2000) emphasized that even minor alterations in insulin sensitivity could substantially affect cardiovascular risk factors among young adults.[15]

Insulin resistance provides an overview of endothelial dysfunction and heightened cardiovascular risks, while inadequate β-cell function leads to persistent hyperglycemia, underscoring the development of metabolic syndrome. The scatter plots distinctly illustrate the correlations between HOMA-β and HOMA-IR with various cardiometabolic markers across different obesity phenotypes, revealing critical insights into the pathophysiological mechanisms behind metabolic health in diverse groups. In MHO individuals, the strong negative correlation between HOMA-β and FBG supports the notion that despite higher adiposity, effective β-cell compensatory mechanisms help maintain glucose homeostasis. This finding aligns with research by Bluher *et al.* (2019), who demonstrated that some obese individuals maintain metabolic health through robust β-cell function.[1] However, this contrasts with findings by Wang et al. (2020), who suggest that many MHO individuals eventually experience β-cell failure, leading to diabetes onset.[19] For MUO individuals, higher correlations between HOMA-IR and adverse markers such as increased TG and WC emphasize the role of insulin resistance in driving metabolic syndrome components, consistent with study by Samuel and Shulman (2016), who explained the pathways by which insulin resistance worsens lipid dysregulation and adiposity.[20] Conversely, insights from the research by Smith *et al.* (2017) highlight that not all obese individuals exhibit this pattern, which specify that changeability may be due to dissimilar distribution of regional adipose tissue, genetic and lifestyle factors.[21] Patho-physiologically, insulin resistance in MUO is often linked to chronic inflammation and ectopic fat deposition, which impairs insulin signaling pathways, as detailed was described by Johnson et al. (2018).[22]

In this study logistic regression analysis reveals that enhanced β-cell functionality (HOMA-β) significantly reduces the risk of metabolic syndrome in young adults, as indicated by its strong statistical significance. Conversely, hyperinsulinemia, although initially compensatory, may lead to increased metabolic syndrome risk over time due to β-cell fatigue. Our report also shows traditional markers like HOMA-IR and HbA1c were not significant predictors, highlighting the need for early interventions targeting insulin sensitivity and β-cell health to effectively manage and prevent metabolic complications in this population. Gastaldelli A *et al*. emphasize that β-cell dysfunction significantly predicts metabolic syndrome.[23] Holman RR *et al.* linked early hyperinsulinemia to later insulin resistance and metabolic complications, supporting the theory of compensatory insulin secretion becoming pathological over time.[24] Additionally, Sakurai M *et al*. identified genetic markers predisposing individuals to β-cell dysfunction and insulin resistance, suggesting a targeted approach for early detection.[25] Lastly, Lee I *et al.* , Naja *et al.* and Garg M K *et al.* suggest that dietary modifications can significantly improve insulin dynamics and reduce metabolic syndrome risk by enhancing insulin sensitivity.[26-28] Previous research collectively underscores the multifactorial nature of metabolic syndrome, advocating for integrated strategies that address lifestyle, genetic, and physiological factors to prevent its onset in young adults.

The ROC curve analysis targeting young adults in central India provides insightful data on the predictive utility of metabolic markers for metabolic syndrome. Notably, HOMA-β emerges with the highest AUC (0.77), indicating that β-cell functionality is a crucial determinant in preventing early metabolic disturbances in younger populations. This result reflects the β-cells' capacity to adequately respond to insulin demands before the onset of more severe insulin resistance—a key pathophysiological factor in maintaining early metabolic health. Inversely, insulin levels and HOMA-IR show lower AUCs (0.49 and 0.38, respectively), suggesting that HOMA-IR is less effective to predict the initial stages of MetS among young adults. Makers like HOMA-IR and HbA1c are unable to determine the early patho-physiological changes such as insulin signaling efficiency and peripheral tissue response, which are important in young adults who may not yet exhibit pronounced MetS.[20] The less predictability of HbA1c (AUC: 0.25) to diagnose MetS further supports that, HOMA-IR and HbA1c may a good predictor for longer-term glycemic and metabolic imbalance rather than acute metabolic shifts, which are more relevant in younger diagnosed with early stage Mets.[29] Understanding these patho-physiological interphase is vital to develop early

Journal of Molecular Science
Volume 35 Issue 2, Year of Publication 2025, Page 238-245
DoI-17.4687/1000-9035.2025.033

intervention approaches tailored to young adults. Current report also emphasizing the need for a multi-directional intervention that integrates both glycemic and insulin response assessments to effectively identify and manage emerging metabolic risks. HOMA-IR, which assesses fasting insulin and glucose levels to determine insulin resistance, may not capture the early, understated stages of metabolic disturbances in younger populations who are generally healthier or at the initial phases of insulin resistance.

Our result is reliable with previous findings of Matthews et al., who noted that HOMA-IR is more effective in diagnosing clear, clinically evident insulin resistance rather than the subtler, initial stages often seen in younger adults.[17] Moreover, the population-specific factors such as genetic predispositions, dietary habits, and physical activity levels significantly influence the manifestation and detection of insulin resistance. The impact of these factors is supported by the study of Kahn and Flier (2000), who addressed how insulin dynamics could vary based on an individual's genetic and environmental context, particularly in less diverse or younger populations.[15] Furthermore, in young individuals, compensatory mechanisms such as hyperinsulinemia can mask early insulin resistance, making it difficult to detect with HOMA-IR. In this phenomenon elevated insulin levels temporarily compensate for insulin resistance, which was described by Ferrannini *et al*. (2005), highlighting how such compensation can delay the clinical presentation of metabolic syndrome.[18]

Our study provides novel insights into the predictive utility of HOMA-β for metabolic syndrome (MetS) in young adults, challenging the conventional reliance on insulin resistance markers such as HOMA-IR. Unlike previous studies that primarily emphasize insulin resistance in MetS pathophysiology, current study highlights β-cell function as a superior predictor, suggesting that early impairments in β-cell activity may play a more pivotal role in MetS progression than insulin resistance alone. Furthermore, current study advances the understanding of obesity phenotypes, demonstrating that metabolically healthy obese (MHO) individuals maintain preserved β-cell function despite elevated insulin resistance, whereas metabolically unhealthy obese (MUO) individuals exhibit significant β-cell dysfunction. These findings reinforce the necessity for phenotype-specific screening and intervention strategies, potentially refining current MetS risk stratification models. Among its strengths, our study benefits from a well-characterized group of young adults and employs rigorous statistical approaches, including Pearson's correlation, logistic regression, and ROC analysis, to establish the predictive accuracy of metabolic markers. However, the cross-sectional design impedes causal inferences, limiting the ability to determine whether β-cell dysfunction precedes MetS onset or arises as a consequence. Additionally, the study's focus on a specific age group and potentially homogeneous population may restrict generalizability to broader demographics. Future longitudinal investigations incorporating diverse populations and additional metabolic biomarkers are essential to validate these findings and further elucidate the role of β-cell function in MetS pathogenesis.

CONCLUSION:

Finding from this current study on the utility of HOMA-IR and HOMA-Beta in detecting metabolic syndrome in a young adult population in central India highlight higher predictive capability of HOMA-Beta. This marker, reflecting β-cell functionality, is more effective than HOMA-IR in identifying early signs of metabolic syndrome, suggesting that β-cell health is vital in assessing metabolic risks in young adults. The inadequate utility of HOMA-IR underscores the need to improve current screening protocols to incorporate more sensitive indicators of early metabolic changes. By prioritizing β-cell functionality through markers like HOMA-Beta, healthcare providers can better target preventative measures and interventions, enhancing the management of metabolic syndrome. This approach ensures a more proactive and tailored strategy in tackling metabolic health issues among young adults, aiming to curb the progression of metabolic syndrome at its nascent stage.

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Table 1: Phenotype-Specific Mean ± Standard Deviation for HOMA-IR, HOMA-BETA, Insulin Levels, and HbA1c

Markers	MHNW	MHO	MUNW	MUO
HOMA-IR	1.80 ± 0.48	2.03 ± 0.61	1.98 ± 0.58	1.90 ± 0.50
HOMA-BETA	123.03 ± 72.21	103.73 ± 38.60	96.29 ± 29.29	75.28 ± 13.35
Insulin Level	8.19 ± 0.15	8.29 ± 0.98	8.08 ± 1.25	7.74 ± 1.16
HbA1c	5.01 ± 0.61	5.27 ± 0.30	5.40 ± 0.64	5.52 ± 0.43

This table summarizes the central tendencies (mean) and variability (standard deviation) of these markers for each phenotype.

Table 2: Cardiometabolic Risk Factor Distribution Across HOMA- β and HOMA-IR Quartiles Among Young Adults population

Metabolic risk factors	HOMA-β_Q1 (Mean±SD)	HOMA-IR_Q1 (Mean±SD)	HOMA- β_Q2 (Mean±SD)	HOMA-IR_Q2 (Mean±SD)	HOMA- β_Q3 (Mean±SD)	HOMA-IR_Q3 (Mean±SD)
WC (cm)	84.19 ± 9.57	79.80 ± 7.27	82.28 ± 8.76	81.50 ± 8.32	78.93 ± 5.17	84.20 ± 8.85
	95% CI: 1.63	95% CI: 1.23	95% CI: 1.49	95% CI: 1.43	95% CI: 0.89	95% CI: 1.52
	IQR: 12.50	IQR: 8.00	IQR: 13.00	IQR: 12.00	IQR: 7.00	IQR: 12.00
SBP (mmHg)	125.99 ± 5.91	122.12 ± 8.62	122.92 ± 8.55	123.34 ± 8.43	121.80 ± 9.33	125.32 ± 7.29
	95% CI: 1.01	95% CI: 1.46	95% CI: 1.46	95% CI: 1.45	95% CI: 1.60	95% CI: 1.25

Journal of Molecular Science

Journal of Molecular Science
Volume 35 Issue 2, Year of Publication 2025, Page 238-245
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	IQR: 10.00	IQR: 6.00	IQR: 8.00	IQR: 8.00	IQR: 6.00	IQR: 10.00
DBP (mmHg)	83.35 ± 5.08	79.12 ± 6.62	80.52 ± 6.47	80.82 ± 6.25	78.83 ± 6.69	82.83 ± 5.72
	95% CI: 0.87	95% CI: 1.12	95% CI: 1.10	95% CI: 1.07	95% CI: 1.15 IQR: 12.00	95% CI: 0.98
	IQR: 9.00	IQR: 8.00	IQR: 4.00	IQR: 6.00		IQR: 8.00
FBG (mg/dl)						
	97.53 ± 4.00	87.96 ± 7.34	92.20 ± 4.44	90.79 ± 6.08	83.65 ± 4.30	94.83 ± 6.13
	95% CI: 0.68	95% CI: 1.24	95% CI: 0.76	95% CI: 1.04	95% CI: 0.74	95% CI: 1.05
	IQR: 3.00	IQR: 10.00	IQR: 5.90	IQR: 10.00	IQR: 7.80	IQR: 6.00
TG (mg/dl)	150.50 ± 7.20	148.43 ± 6.69	148.56 ± 5.95	148.51 ± 6.41	148.47 ± 6.47	150.62 ± 6.55
	95% CI: 1.23	95% CI: 1.13	95% CI: 1.01	95% CI: 1.10	95% CI: 1.11	95% CI: 1.12
	IQR: 6.00	IQR: 3.00	IQR: 3.00	IQR: 3.00	IQR: 3.00	IQR: 5.00
HDL (mg/dl)	47.05 ± 8.34	49.96 ± 8.08	47.62 ± 8.28	48.88 ± 8.70	50.89 ± 8.23	46.64 ± 8.23
	95% CI: 1.42	95% CI: 1.37	95% CI: 1.41	95% CI: 1.49	95% CI: 1.41	95% CI: 1.41
	IQR: 16.00	IQR: 14.00	IQR: 15.50	IQR: 14.98	IQR: 17.00	IQR: 14.00

This table represents the mean ± standard deviation, 95% confidence intervals (CI), and interquartile ranges (IQR) for cardiometabolic components—across three quartiles of HOMA-BETA and HOMA-IR.

Table 3: Logistic Regression analysis for Metabolic Predictors to predict metabolic syndrome among young adult

Predictors	Coefficient	Standard Error	z-value	p-value	95% CI Lower	95% CI Upper
Const.	5.56	2.96	1.88	0.06	-0.22	11.37
HOMA-IR	0.33	0.33	0.99	0.32	-0.32	0.97
HOMA-β	0.09	0.02	4.93	<0.001	0.052	0.12
FSI (mircoU/mL)	-1.13	0.23	-4.89	<0.001	-1.59	-0.68
HbA1c (%)	-0.60	0.41	-1.48	0.14	-1.41	0.20

This table presents the logistic regression coefficients, standard errors, z-values, p-values, and 95% confidence intervals (CI) for key metabolic markers—HOMA-IR (insulin resistance), HOMA-BETA (β-cell function), insulin levels, and HbA1c—used to predict the likelihood of metabolic syndrome in young adults.

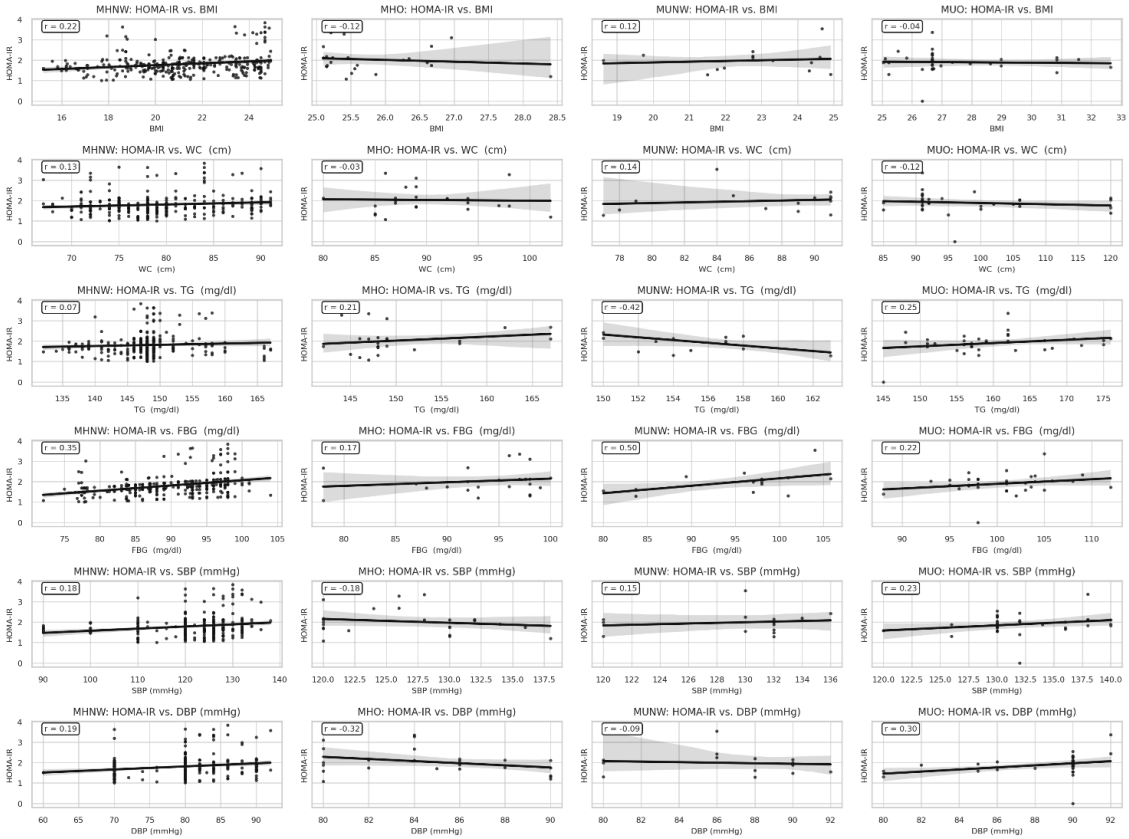

Figure 1: Scatter Plots shows Correlations between HOMA-IR and *cardiometabolic components* across phenotype

Figure Legend: The scatter plots illustrate the correlations between insulin resistance (HOMA-IR) and various *cardiometabolic components* across different obesity phenotypes. The correlation coefficients (r) values are provided for each plot.

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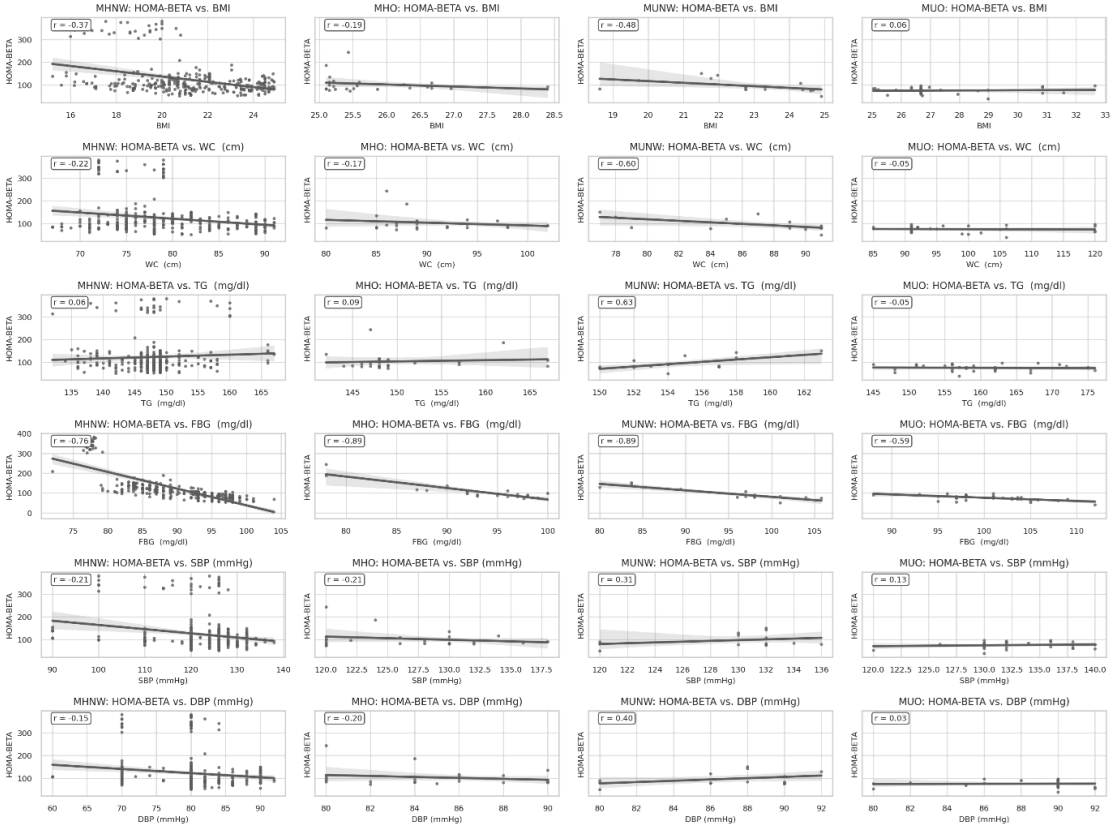

Figure 2: Scatter Plots shows Correlations between HOMA-*Beta* and *cardiometabolic components* across phenotype

Figure Legend: This scatter plots illustrate the correlations between pancreatic Beta cell function (HOMA-β) and *cardiometabolic components* for different obesity phenotypes. The correlation coefficients (r) values are provided for each plot.

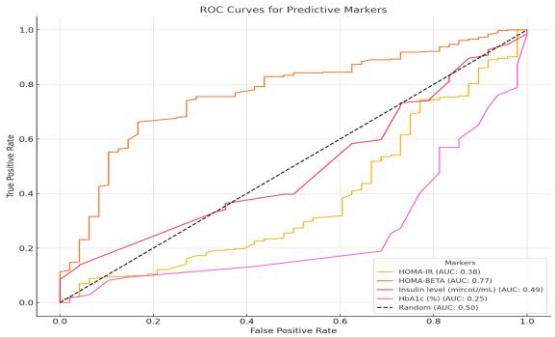

Figure 3: Receiver Operating Characteristic (ROC) curves for four different metabolic markers.

Footnote: Each curve plots the true positive rate (sensitivity) against the false positive rate (specificity), providing a measure of each marker's ability to correctly identify individuals with metabolic syndrome.