Journal of Rare Cardiovascular Diseases
ISSN: 2299-3711 (Print) | e-ISSN: 2300-5505 (Online)
www.jrcd.eu
RESEARCH ARTICLE

Distribution of Insulin Resistance Among Normal Body Weight Individuals Defined by Bmi in Patients Presenting to Tertiary Care Hospital of Chennai

Dr. Gokul K[1], Dr. Ramachandran V[2] and Dr. Kishore Karthik S[3]
[1]Junior Resident, Department of General Medicine, Sree Balaji MedicalCollege and Hospital, Chennai, India
[2]DNB Professor of Medicine, Department of General Medicine, Sree Balaji Medical College and Hospital, Chennai, India
[3]Assitant Professor, Department of General Medicine, Sree Balaji Medical College and Hospital, Chennai, India

*Corresponding Author
Gokul K
(gokulkrish018@gmail.com)

Article History
Received: 21.07.2025
Revised: 30.08.2025
Accepted: 15.09.2025
Published: 30.09.2025

Abstract: Insulin resistance (IR) is a major contributor to type 2 diabetes, cardiovascular disorders, and metabolic syndrome. Although often associated with obesity, increasing evidence shows that individuals with normal body mass index (BMI) may also develop IR, especially within Asian populations where a "thin-fat" phenotype is common. This cross-sectional study was conducted at a tertiary care hospital in Chennai to evaluate the prevalence and distribution of IR among normal-BMI adults aged 18–50 years. Using the Homeostatic Model Assessment of Insulin Resistance (HOMA-IR), 67.2% of participants were found to have varying degrees of IR, despite their BMI being within the normal range. No significant associations were observed with age or gender, but fasting blood glucose showed a strong correlation with IR. The findings emphasize that BMI alone is insufficient for screening and that early detection of IR using markers such as HOMA-IR and fasting glucose may help in preventing long-term metabolic complications.

Keywords: Insulin resistance, Normal BMI, Fasting blood glucose, Metabolic syndrome, HOMA-IR.

INTRODUCTION

IR, a key metabolic abnormality, contributes to "type 2 diabetes mellitus" (T2DM), metabolic syndrome and cardiovascular disease development. IR is characterised by impairment of insulin-mediated glucose metabolism of peripheral cells (eg, endothelial tissue, hepatic tissue, skeletal muscle tissue and adipose tissue). Previously, IR was strongly related to obesity, hence, emerging evidence emphasises that individuals who have a normal "body mass index" (BMI) can develop IR, often going undiagnosed because of reliance on BMI as a tool for primary screening. This sheltered risk is relevant in the Asian populations, where "thin-fat phenotype" predisposes normal-weight persons to metabolic disturbances. Early IR detection in these groups is crucial, as timely intervention may allow for the prevention of long-term complications. The "Homeostatic Model Assessment of Insulin Resistance" (HOMA-IR) is mainly recognised as an effective tool for analysing IR in research and clinical settings. This study focuses on exploring the distribution and prevalence of insulin resistance in normal BMI individuals presenting to Chennai tertiary care hospital.

Aim:

The research aims to analyse the prevalence of IR by using "Homeostatic Model Assessment of Insulin Resistance" (HOMA-IR) among normal-weight individuals defined by BMI in a hospital setting of Chennai tertiary care.

Objective:

- To assess prevalence of insulin resistance in normal BMI individuals by using HOMA-IR.
- To explore insulin resistance distribution as per demographic factors like gender and age.
- To analyse the association of lipid profile with insulin resistance, fasting blood sugar and fasting insulin in normal BMI individuals.
- To evaluate clinical features observed among the normal BMI individuals, along with insulin resistance.

LITERATURE REVIEW

In "first-degree relatives" (FDRs), both pancreatic β-cell dysfunction and insulin resistance are intimate phenomena that are proposed to increase cardiometabolic risk and synergistically exacerbate type 2 diabetes. The relative roles of insulin resistance and decreased β-cell function in the development of Type 2 diabetes have debated during the last three decades. Different conclusions are drawn from several prospective studies examining FDRs of Type 2 DM in various ethnic groups1. In Pima Indians, African-Americans, and Indians, insulin resistance identified as a significant risk factor for the development of diabetes. In Japan, Caucasians, China and Korea, impaired β-cell function is proposed to contribute significantly to Type 2 DM than insulin resistance. Others, however, showed that before glucose tolerance was aberrant, type 2 DM patients' FDRs exhibited both insulin resistance and compromised β-cell function. Since obesity is a common feature of type 2 diabetes, we hypothesised that it mediated the conflicting link between leptin and diabetic nephropathy2. Identifying leptin as a risk factor for

diabetic nephropathy in both non-obese and obese patients with type 2 diabetes is worthwhile.

Patients with type 2 diabetes who are fat or overweight had higher levels of IL-6 and lower levels of adiponectin, which may indicate more adipose tissue (AT) dysfunction. Extremely hypertrophied adipocytes linked to obesity cause oxidative stress and elevated TNF-α production in AT, which further suppresses adiponectin mRNA expression while increasing IL-6 mRNA expression3. These cell-level factors also increase macrophage infiltration and inflammatory cytokine production, which results in insulin resistance in the adipose tissue. A quantitative insulin sensitivity index and a homeostatic IR evaluation model are used to determine the severity of IR. However, these methods include insulin monitoring or invasive testing, which makes them unsuitable for extensive epidemiological investigations4. Similar to earlier epidemiological studies, the study adopted non-insulin-based fasting IR indicators, or surrogates, for measure IR levels. These indicators included "metabolic score for insulin resistance" (METS-IR), the ratio of "triglycerides to high-density lipoprotein cholesterol" (TG/HDL-C), the "triglyceride glucose" (TyG), and "TyG with body mass index" (TyG-BMI).

A report by5, indicates that regardless of the ischemia cause, IR is common in "Heart Failure" (HF) patients. IR was linked to incident HF in a community cohort, whereas BMI did not affect this association. In patients of HF without known diabetes, IR demonstrated to be a predictor of death. Patients with "HF and reduced left ventricular ejection fraction" (HFrEF) are known to exhibit IR. It is not quite apparent whether patients with "HF with preserved ejection fraction" (HFpEF) have a different prevalence of IR. Older patients (70–90 years) with either diastolic or systolic heart failure are more insulin resistant than the same age healthy volunteers, according to a recent investigation on a small series. According to another study, patients with HFrEF had more severe IR than those with HFpEF. Hence, early identification of IR is essential for preventing "metabolic syndrome" (MetS), type 2 diabetes, and "cardiovascular diseases" (CVD), as these disorders are known to be significantly influenced by it6. Although the glucose clamp technique is the gold standard for measuring IR8, its frequent use in laboratories is limited by its complexity, expense, and invasiveness.

Therefore, since its introduction in 1985, more straightforward techniques such as the HOMA-IR have gained widespread use. However, issues with the availability and uniformity of insulin measurements have led to research into alternate methods for IR prediction, such as "visceral fat index" (VAI) and lipid ratios6. Human IR is associated with decreased mammalian/insulin target of the rapamycin complex-2 signalling pathway in visceral fat and increased synthesis of monocyte chemoattractant protein-1. Insulin-resistant macrophages are linked by the researchers to cardiovascular problems in type 2 diabetes and metabolic syndrome in a mouse model. Insulin-resistant macrophages are typically more likely to undergo apoptosis within atherosclerotic plaques. As a result, when HOMA-IR rises, atherosclerosis may proceed more quickly7. Lastly, vasoconstriction and vascular smooth muscle cell proliferation can be brought on by a malfunctioning insulin signalling system. Insulin can stimulate both endothelin-1-dependent vasoconstriction and NO-dependent vasodilation. On the other hand, preferential activation of the "Raf/mitogen-activated protein kinase pathway" causes excessive vasoconstriction and vascular smooth muscle cell proliferation in the presence of vascular IR.

Diabetes, a metabolic disease, concealed IR, while subclinical hypothyroidism did not influence insulin resistance. However, in the population with normal blood glucose, the impact of subclinical hypothyroidism on insulin resistance was thoroughly demonstrated8. IR indicates that the target organs are less sensitive and responsive to the effects of insulin. It is characterised by increased hepatic gluconeogenesis and decreased muscle and fat use of glucose, leading to metabolic and hemodynamic abnormalities9. The metabolic syndrome is the main risk factor for a number of metabolic and cardiovascular diseases. Even in otherwise healthy people, proactive IR identification is essential due to the high frequency of IR and metabolic syndrome. Even though the "hyperinsulinemic-euglycemic clamp" (HEC) is considered the gold standard for IR measurement, clinical applications are hampered by its invasiveness, complexity, and cost. Thus, it is essential to detect metabolic abnormalities in normal-weight people early on, especially in individuals who are more likely to develop visceral fat accumulation and IR, even though their absolute BMI is lower than that of Western cultures.

METHODOLOGY

- Study Design: Cross-sectional study
- Study Setting: This research was place in the academic and tertiary care facility Sree Balaji Medical College and Hospital in Chennai, India, specifically in the Department of General Medicine.
- Study Population: Adults more than 18 years of age to 50 years.
- Study Duration: 18 months
- Sampling Methodology: Purposive sampling
- Sample Size Calculation:
- Sample Size Formula For Pooled Variance

$$S_p = \sqrt{\frac{(n_1-1)s_1^2+(n_2-1)s_2^2}{(n_1+n_2-2)}}$$

SP = 18.5
N=2{ $z\sigma^2$ E^2 }
N = 56
Z 2 1 -α/2 = 95% level of confidence
E^2= error margin, i.e., 5%
σ = is calculated by pooling the sd, i.e., calculated using S P formulae, i.e., 18.5
Sample Size=58

Inclusion and Exclusion Criteria
Inclusion Criteria:
1. Ages: 18-50 Years
2. Normal-weight individuals defined by BMI

Exclusion Criteria:
1. Age: more than 50 years
2. During the trial, a history of hyperglycemia or fasting blood sugar levels of 126 or above, if known.
3. History of hypertension and dyslipidemia
4. History of chronic kidney, liver, heart, cancer, thyroid, or tuberculosis while on medicine
5. Stimulants, antipsychotics, statins, and diuretics users.
6. Not willing to participate

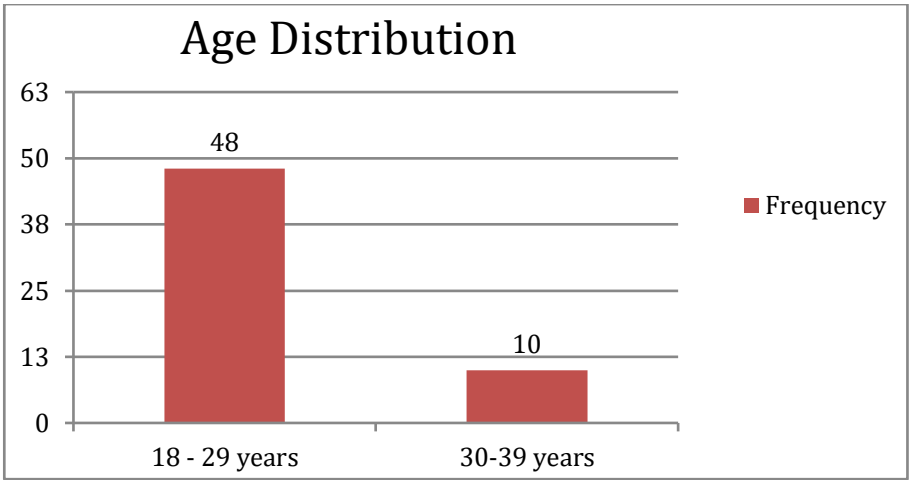
Study Procedure
Participants are adults (between the ages of 18 and 50) who visited "Sree Balaji Medical College and Hospital" between August 2023 and February 2024 and have a normal BMI. During admission, a detailed history was collected, and general and systemic examinations were done. Anthropometric assessments are also done to collect BMI, weight, and height, followed by investigations. After acquiring informed consent, detailed anthropometric measurements and clinical histories are recorded. Biochemical investigations involve fasting LDL cholesterol, blood glucose, HDL cholesterol, fasting insulin and triglycerides. IR is assessed by utilising the HOMA-IR, calculated from values of insulin and fasting glucose. Participants are also then categorised as borderline, normal or having significant IR as per cut-offs of HOMA-IR. Statistical analysis was performed by conducting inferential and descriptive methods. Relevance among biochemical variables or IR and demographic variables is explored by using the ANOVA or t-test for means and chi-square test for proportions. Pearson's correlation is also applied to emphasise the relationships between HOMA-IR and some other parameters.

RESULT

Age Distribution
Participants in the study were split up among several age groups. Ten individuals (17.3%) were between the ages of 10 and 39, while 48 participants (82.7%) were between the ages of 18 and 29.

TABLE 1: Age Distribution Of Participants

Age Distribution		
Age	Frequency	Percent
18 - 29 years	48	82.7
30-39 years	10	17.3



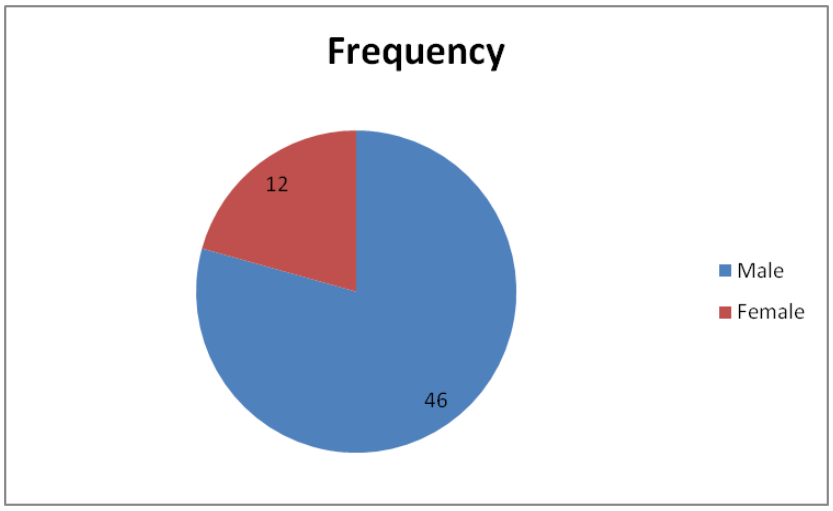
Graph 1: Age Distribution of Participants

Gender Distribution

Male participants outnumbered female participants in the study. There were 12 participants (20.6%) who were female, and 46 participants (79.4%) who were male.

TABLE 2: Gender Distribution of Participants

Gender Distribution		
Gender	Frequency	Percent
Male	46	79.4
Female	12	20.6
Total	58	100



Graph 2: Gender Distribution of Participants

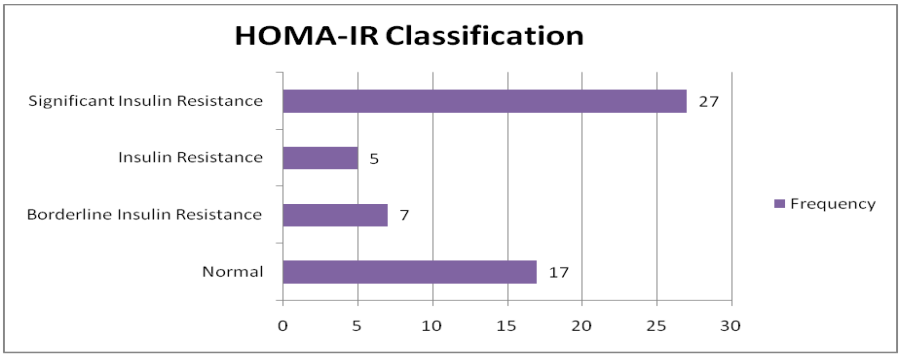
HOMA-IR Classification

Nineteen (32.75%) of the study subjects had no insulin resistance, seven (12.06%) had borderline insulin resistance, five (8.6%) had insulin resistance, and twenty-seven (46.55%) had considerable insulin resistance.

TABLE 3: HOMA-IR Classification among Participants

HOMA-IR Classification		
HOMA-IR	Frequency	Percent

Normal	17	32.75
Borderline Insulin Resistance	7	12.06
Insulin Resistance	5	8.6
Significant Insulin Resistance	27	46.55
Total	58	100



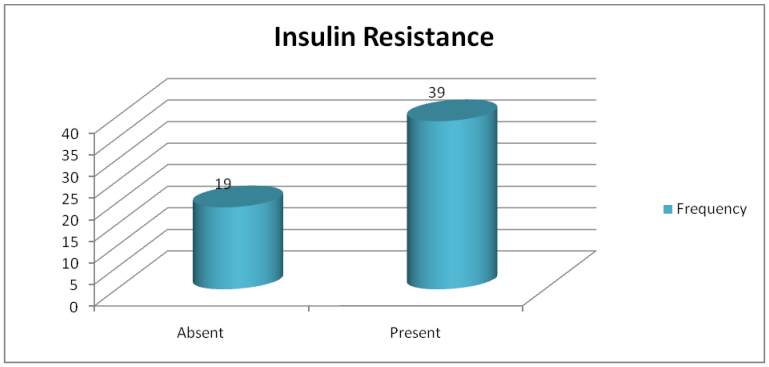
Graph 3: Gender Distribution among Participants

Insulin Resistance

In all, 39 people (67.24%) had insulin resistance, while 19 persons (32.76%) did not.

TABLE 4: Insulin Resistance among Participants

Insulin Resistance		
Insulin Resistance	Frequency	Percent
Absent	19	32.76
Present	39	67.24
Total	58	100



Graph 4: Insulin Resistance among Participants

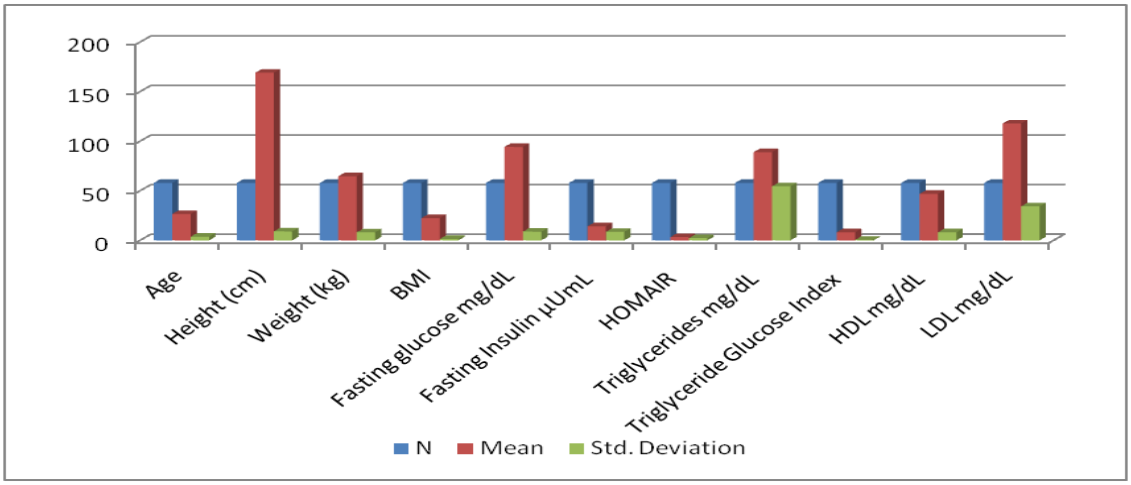
Descriptive Statistics for Anthropometric and Laboratory Parameters

Participants were 26.64 ± 3.70 years old on average. With an average BMI of 22.48 ± 1.42 kg/m2, the weight range was considered normal. Insulin and glucose levels at fasting were 14.17 ± 8.61 µU/mL and 94.29 ± 8.76 mg/dL, respectively. A trend toward insulin resistance was indicated by the mean HOMA-IR of 3.40 ± 2.34. With a mean TyG index of 8.20 ± 0.52, metabolic risk may also be indicated. The average HDL and LDL levels in the lipid profile were 46.98 ± 8.20 mg/dL and 117.90 ± 34.31 mg/dL, respectively.

TABLE 5: Descriptive Statistics for Anthropometric and Laboratory Parameters

Descriptive Statistics for Anthropometric and Laboratory

Parameters			
Anthropometric and Laboratory Parameters	N	Mean	Std. Deviation
Age	58	26.636	3.703
Height (cm)	58	169.103	9.014719
Weight (kg)	58	64.689	8.122355
BMI	58	22.4789	1.422145
Fasting glucose mg/dL	58	94.2931	8.763184
Fasting Insulin µUmL	58	14.17155	8.613562
HOMAIR	58	3.3994	2.338358
Triglycerides mg/dL	58	89.034	54.57199
Triglyceride Glucose Index	58	8.1988	0.52036
HDL mg/dL	58	46.9827	8.20117
LDL mg/dL	58	117.896	34.3108



Graph 5: Descriptive Statistics for Anthropometric and Laboratory Parameters

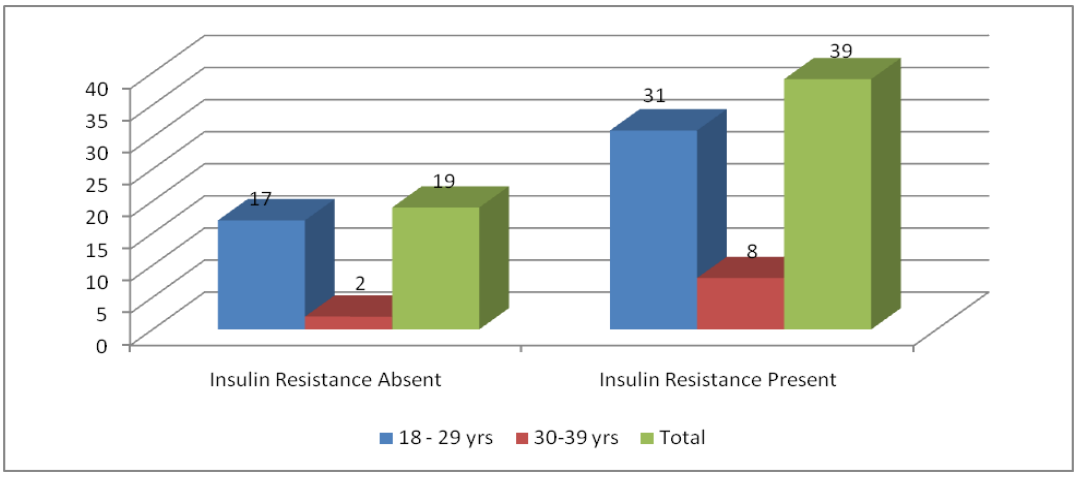
Association between Age and Insulin Resistance

There was no statistically significant relationship between age group and insulin resistance ($\chi^2 = 0.893$, $p = 0.3447$). Insulin resistance was more common in people aged 30 to 39 (80%) than in people aged 18 to 29 (64.59%), although the difference was not statistically significant.

TABLE 6: Association between Age and Insulin Resistance

ASSOCIATION BETWEEN AGE AND INSULIN RESISTANCE					
Age	Insulin Resistance		Total	chi-square value	p-value
	Insulin Resistance Absent	Insulin Resistance Present			
18 - 29 yrs	17	31	48	0.893	0.3447
	35.41%	64.59%	100.00%		

30-39 yrs	2	8	10		
	20%	80%	100%		
Total	19	39	58		
	32.75%	67.24%	100.00%		



Graph 6: Association between Age and Insulin Resistance

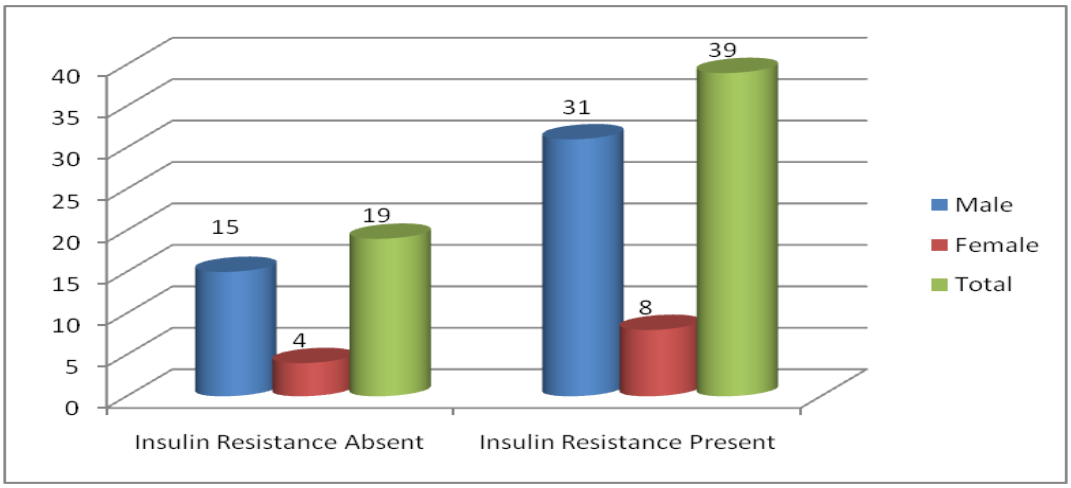
Association between Gender and Insulin Resistance

There was no statistically significant relationship between gender and insulin resistance ($\chi^2 = 0.002$, $p = 0.9620$). Insulin resistance was roughly equally prevalent in men (67.40%) and women (66.67%) in this study sample, indicating that gender did not significantly affect this finding.

TABLE 7: Relationship between Stimulin Resistances And Gender

RELATIONSHIP BETWEEN STIMULIN RESISTANCE AND GENDER RESISTANCE					
Age	Insulin Resistance		Total	chi-square value	p-value
	Insulin Resistance Absent	Insulin Resistance Present			
Male	15	31	46	0.002	0.962
	32.60%	67.40%	100.00%		
Female	4	8	12		
	33%	67%	100%		
Total	19	39	58		
	32.75%	67.25%	100.00%		

193



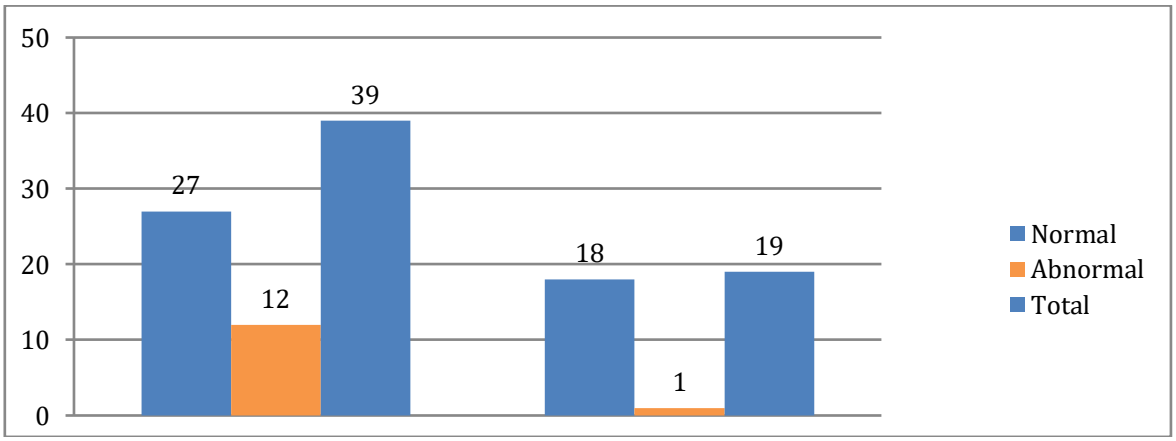
Graph 7: Association between Gender and Insulin Resistance

Association between FBS and Insulin Resistance

Insulin resistance and FBS levels were statistically significantly correlated (χ288; p = 0.0289). Insulin resistance was present in 92.31% of people with abnormal FBS and 60% of people with normal FBS. This implies that people who have high fasting blood sugar levels are far more likely to have insulin resistance.

TABLE 8: Association between FBS and Insulin Resistance

Association Between FBS and Insulin Resistance					
FBS	Insulin Resistance		Total	chi-square value	p-value
	Insulin Resistance Absent	Insulin Resistance Present			
Normal	27	18	45	4.779	0.0288
	60.00%	40.00%	100.00%		
Abnormal	12	1	13		
	92%	8%	100%		
Total	39	19	58		
	67.24%	32.75%	100.00%		



Graph 8: Association between FBS and Insulin Resistance

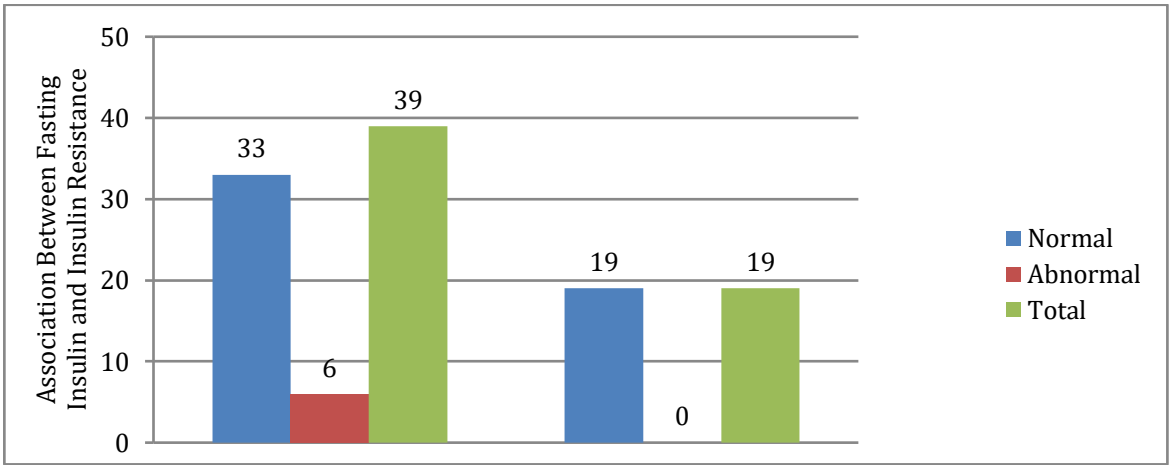
Association between Fasting Insulin and Insulin Resistance

Six (100%) of the 58 subjects were insulin resistant, and six of them had abnormal fasting insulin levels. 33 (63.46%) of the 52 individuals with normal insulin levels were insulin resistant, while 19 (36.54%) were not. According to the Chi-

square test result ($\chi^2 = 3.26$ with a p-value of 0.0710), there is not a statistically significant correlation between insulin resistance and aberrant fasting insulin levels at the 0.05 level, but it is close to significance.

TABLE 9: Association between Fasting Insulin and Insulin Resistance

Association Between Fasting Insulin and Insulin Resistance					
FBS	Insulin Resistance		Total	chi-square value	p-value
	Insulin Resistance Absent	Insulin Resistance Present			
Normal	33	19	52	3.26	0.071
	63.46%	36.54%	100.00%		
Abnormal	6	0	6		
	100%	0%	100%		
Total	39	19	58		
	65.51%	34.49%	100.00%		



Graph 9: Association between Fasting Insulin and Insulin Resistance

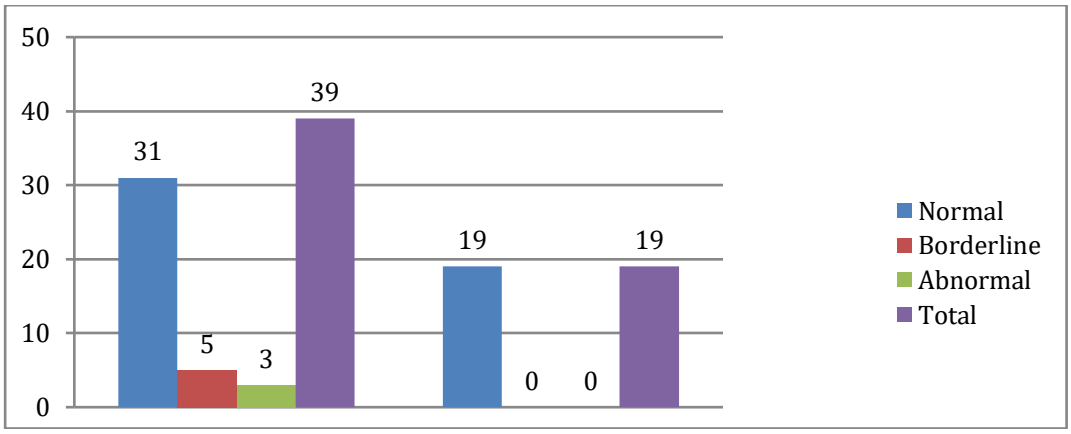
Association between Triglycerides and Insulin Resistance

Only 62% of people with normal triglyceride levels exhibited insulin resistance, compared to 100% of those with excessive levels. Given that the p-value was 0.104, there was no statistically significant association.

TABLE 10: Association between Triglycerides and Insulin Resistance

Association Between Triglycerides and Insulin Resistance					
FBS	Insulin Resistance		Total	chi-square value	p-value
	Insulin Resistance Absent	Insulin Resistance Present			
Normal	31	19	50	4.521	0.104
	62.00%	38.00%	100.00%		
Borderline	5	0	5		
	100%	0%	100%		
Abnormal	3	0	3		

	100.00%	0.00%	100.00%		
Total	39	19	58		
	67.24%	32.76%	100.00%		



Graph 10: Association between Triglycerides and Insulin Resistance

DISCUSSION

The study findings indicate that nearly two-thirds (67.24%) of normal-weight individuals, as defined by BMI, disclose IR when assessed using HOMA-IR. It challenges the notion that IR is confined to obese and overweight populations. It highlights that disturbances in metabolism may be highly prevalent among individuals with normal body weight. In this study, the study group's mean age was 26.64 +/- 3.70 years. It was shown that the age group of 30-39 (80%) had a higher prevalence of IR than the age group of 18-29 (64.59%). However, the lack of a statistically significant correlation ($p = 0.3447$) raises the possibility that age is not a determining factor in IR in individuals of normal weight. This variability is attributed to genetic predisposition, unique patterns of phenotypic and lifestyle factors. Overall, a high prevalence of IR in normal-weight individuals is observed in Indian and other Asian populations, where lean individuals develop cardiovascular disease and type 2 diabetes at younger ages as compared to the Western populations.

IR was nearly equally common in males (67.40%) and females (66.67%), with no discernible difference between the sexes ($p = 0.9620$). IR and fasting blood glucose levels were statistically correlated ($p = 0.0288$), more precisely, 92.31% of people with higher levels of fasting blood glucose also had IR. Although the association between the two heterogeneously failed to reach statistical significance ($p = 0.0710$), a high level of fasting insulin also had strong clinical significance due to IR found to happen in six individuals with a high level of concentration. The “thin-fat” phenotype described in South Asians, characterised by increased visceral fat and central adiposity despite normal BMI, provides a plausible explanation for the observed high prevalence in study set. Similar to epidemiological studies, the study adopted non-insulin-based fasting IR indicators, or surrogates, for measure IR levels. The study findings reveal that people who have high fasting blood sugar levels are likely to have IR.

Metabolic abnormalities in normal-weight people are develop visceral fat accumulation and IR, even though their absolute BMI is lower than the Western cultures. IR was also confirmed by other laboratory markers in patients with this condition. The result indicates that those with IR had significantly higher:

Fasting glucose: 97.20 ± 7.99 vs. 88.31 ± 7.07 mg/dL ($p = 0.000147$).
TyG index: 8.35 ± 0.53 vs. 7.87 ± 0.29 ($p < 0.001$)
Fasting insulin: 17.66 ± 8.47 vs. 6.995 ± 1.55 µU/mL ($p < 0.001$)
Triglycerides: 101.84 ± 60.90 vs. 62.73 ± 21.19 mg/dL ($p < 0.001$)
Although there are no statistically significant changes in HDL and LDL levels ($p = 0.0788$ and $p = 0.1908$, respectively), the cohort of IR individuals tends to have lower HDL levels, which is consistent with the profile of metabolic syndrome as a whole.

CONCLUSION

Studies exhibit that IR is highly prevalent in individuals who have normal BMI, with 67.24% participants classified as IR by HOMA-IR. Absence of signs of contraction with gender and age are offer that normal rates among demographic groups are vulnerable to dysfunction of early metabolism. The fasting blood sugar indicates a strong association with IR, emphasising its role as an accessible marker. Findings of the study underline BMI inadequacy as a sole criterion for support and screening for the use of HOMA-IR in early detection. The literature of the study emphasises that IR macrophages linked by researchers for cardiovascular problems in metabolic syndrome and type 2 diabetes of

mouse model. IR macrophages are undergoing dynamic related to apoptosis within the atherosclerotic plaques. Even among the healthy people, proactive IR identification is important because of high frequency of metabolic syndrome and IR.

REFERENCE

1. Ahmed F, Al-Habori M, Al-Zabedi E, Saif-Ali R. Impact of triglycerides and waist circumference on insulin resistance and β-cell function in non-diabetic first-degree relatives of type 2 diabetes. BMC Endocrine Disorders. 2021 Jun 16;21(1):124.
2. Huang J, Peng X, Dong K, Tao J, Yang Y. The association between insulin resistance, leptin, and resistin and diabetic nephropathy in type 2 diabetes mellitus patients with different body mass indexes. Diabetes, Metabolic Syndrome and Obesity. 2021 May 24:2357-65.
3. Yadav U, Kumar N, Sarvottam K. Obese have comparable ankle brachial pressure index but higher β-cell function and insulin resistance as compared to normal-weight type 2 diabetes mellitus patients. Acta Diabetologica. 2025 Apr;62(4):543-50.
4. Wang H, Zhang J, Pu Y, Qin S, Liu H, Tian Y, Tang Z. Comparison of different insulin resistance surrogates to predict hyperuricemia among US non-diabetic adults. Frontiers in Endocrinology. 2022 Dec 15;13:1028167.
5. Son TK, Toan NH, Thang N, Le Trong Tuong H, Tien HA, Thuy NH, Van Minh H, Valensi P. Prediabetes and insulin resistance in a population of patients with heart failure and reduced or preserved ejection fraction but without diabetes, overweight or hypertension. Cardiovascular Diabetology. 2022 May 14;21(1):75.
6. Bazyar H, Zare Javid A, Masoudi MR, Haidari F, Heidari Z, Hajializadeh S, Aghamohammadi V, Vajdi M. Assessing the predictive value of insulin resistance indices for metabolic syndrome risk in type 2 diabetes mellitus patients. Scientific Reports. 2024 Apr 18;14(1):8917.
7. Lee JH, Jeon S, Joung B, Lee HS, Kwon YJ. Associations of homeostatic model assessment for insulin resistance trajectories with cardiovascular disease incidence and mortality. Arteriosclerosis, thrombosis, and vascular biology. 2023 Sep;43(9):1719-28.
8. Yang W, Jin C, Wang H, Lai Y, Li J, Shan Z. Subclinical hypothyroidism increases insulin resistance in normoglycemic people. Frontiers in Endocrinology. 2023 Jul 6;14:1106968.
9. Xiao D, Sun H, Chen L, Li X, Huo H, Zhou G, Zhang M, He B. Assessment of six surrogate insulin resistance indexes for predicting cardiometabolic multimorbidity incidence in Chinese middle-aged and older populations: Insights from the China health and retirement longitudinal study. Diabetes/Metabolism Research and Reviews. 2024 Jan;40(1):e3764.