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# Interrelation Of HOMA-IR and QUICKI with Anthropometry and Cardio-Metabolic Indicators in Young Adults

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## **ABSTRACT**

Background: The HOMA-IR (Homeostatic Model Assessment for Insulin Resistance) and QUICKI (Quantitative Insulin Sensitivity Check Index) are used to assess insulin resistance.

Insulin resistance is accepted to be a major risk factor in the aetiology of type 2 diabetes mellitus, hypertension, dyslipidaemia, atherosclerotic vascular disease. It may be a risk factor for coronary heart disease and stroke as well. Some recent studies suggest that QUICKI can be a good alternative for HOMA-IR. With this information, we decided to investigate the cut-off values of HOMA-IR and QUICKI to assess cardiometabolic indicators in young adults.

Methods: 105 samples of young adults aged 15 - 25 years were collected after informed written consent. The study was conducted at Dhanalakshmi Srinivasan Medical College and Hospital, following approval from the institutional research and ethics committee. It is a descriptive cross-sectional study. The study parameters include HOMA-IR, QUICKI, and lipid profile. Type 1 Diabetics, Smokers, connective tissue disorder, or those with any other metabolic disorders are excluded from the study. The obtained data were analyzed using SPSS software.

Result: We found a significant positive correlation between TG and HOMA-IR, a significant negative correlation between HDL and HOMA-IR, a significant negative correlation between TG and QUICKI, and a significant positive correlation between HDL and QUICKI.

Conclusion: In the study, we found no much difference between QUICKI and HOMA-IR when compared with lipid profile in young adults.

Key-words: Anthropometry, Cardio-metabolic indicators, HOMA-IR, Insulin Index, QUICKI

# **INTRODUCTION**

Insulin resistance is not simply a problem of incomplete glucose uptake in response to insulin, but a complex condition that significantly increases the risk of cardiovascular disease.

#### How to cite this article

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The associations between insulin resistance and dyslipidemia, hypertension, and atherosclerosis are numerous and complex. Hyperinsulinemia and Insulin resistance are associated with most of the underlying abnormalities in the body, leading to cardiovascular disease, a major cause of morbidity and mortality worldwide. [1,2]. The incompetence of insulin-resistant fat cells to store triglycerides is to be expected as the first step in the progression of dyslipidaemia, a hallmark of insulin resistance [3].

Several methods have been proposed for assessing insulin resistance (IR). Among these indices, the most recognized is the Homeostatic Model Assessment for Insulin Resistance (HOMA-IR) index, as well as a newly

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proposed index, the Quantitative Insulin Sensitivity Check Index (QUICKI), which are increasingly used [4]. Modern studies recommend that QUICKI can be a good substitute for HOMA-IR, and it is also stated that HOMA-IR displays reduced insulin resistance compared to the actuality [5-7].

Hyperinsulinemia and central obesity that typically accompany insulin resistance are thought to lead to overproduction of very low-density lipoprotein [8]. Much of the cardiovascular (CV) disease associated with metabolic syndrome may be explained by the presence of insulin resistance (IR). There is compelling, longstanding evidence that both high triglycerides and low plasma HDL-C are frequent consequences of IR [3]. Elevated LDL is not a characteristic of the dyslipidemia of insulin resistance; however, in the insulin-resistant state, the composition of LDL is altered. The dyslipidemia associated with insulin resistance plays a major role in the development of atherosclerosis [8].

The incidence of metabolic syndrome increased rapidly from 15 to 25 years of age [9,10]. Analysis of Insulin resistance in the pre-disease state will lead to the commencement of prophylactic actions, such as modifications in lifestyle, diet & physical activity, thereby avoiding high-risk subjects from progressing to sickness. This information helps us develop the idea of correlating HOMA-IR and QUICKI indices with anthropometry and cardiometabolic indicators in young adults.

# **MATERIALS AND METHODS**

Place of the study- The descriptive cross—sectional study was conducted in the Central Biochemistry Laboratory in Dhanalakshmi Srinivasan Medical College and Hospital, Perambalur, Tamil Nadu, India, for a period of one year from April 2022. One hundred five samples were collected from young adults aged 19 to 22, with informed written consent from subjects who had come for a Master's health check-up at Dhanalakshmi Srinivasan Medical College and Hospital.

Methodology- Patients' details were collected, and anthropometric parameters (height, weight, waist circumference, BMI, and height-to-waist circumference ratio) were measured. Height was measured using a stadiometer (wall-mounted stadiometer with range 200cm) without shoes and weight was measured without shoes and wearing light clothes, using a digital weighing

scale (Flexscale Smart Bluetooth Weighing Scale). Height and weight measurements were used to calculate BMI (Body Mass Index = weight (kg) divided by height (m) squared). Waist circumference (WC) was measured using a measuring tape at the midpoint between the superior border of the iliac crest and the lowermost margin of the rib. Systolic and Diastolic Blood pressure were measured according to the 2020 International Society of hypertension [9]. After 10 - 12 hour of overnight fasting, venous blood samples were drawn from the participants to analyse fasting glucose (glucose oxidase-peroxidase method), fasting insulin (ELISA method), triglyceride, high-density lipoprotein cholesterol (HDL-C), low-density lipoprotein cholesterol (LDL-C)and total cholesterol levels were estimated using an automated analyser (Erba XL640) and commercially available kit.

# HOMA-IR = (fasting insulin $\mu$ m/ml) + (fasting glucose mg/dl)/405

# QUICKI= $1/[\log(\text{fasting insulin }\mu\text{U/mI}) + \log(\text{fasting insulin }\mu\text{U/mI})$ glucose mg/dl)]

Inclusion Criteria- Diabetic, Obese, or normal young adults within the age group 15-25.

**Exclusion Criteria-** Known case of cardiovascular diseases, Smokers, connective tissue disorder, or people with any other metabolic disorders are excluded from the study.

Statistical Analysis- All the collected data were entered into Microsoft Excel. We used SPSS software, version 20, for the statistical analysis of the data. Pearson correlation coefficient was used to analyse the data. The significance level for all analyses was p<0.05.

Ethical Approval- Approval for the study was obtained from the institutional ethics committee and institutional committee (IECHS/IRCHS/No: research 166) Dhanalakshmi Srinivasan Medical College and Hospital, Perambalur, Tamil Nadu, India, on 22 March 2022.

#### **RESULTS**

In Table 1, Pearson's correlation analysis of QUICKI and HOMA-IR with the participants' mean and standard deviation of anthropometric and lipid profiles is summarized (n=105). There was a significant positive correlation between HOMA-IR and weight, and a nonsignificant positive correlation with waist circumference, BMI, Systolic blood pressure, and diastolic blood pressure. QUICKI showed a significant negative

correlation with weight and a non-significant negative correlation with waist circumference, BMI, Systolic blood pressure and diastolic blood pressure.

Table 1: Pearson's correlation analysis of QUICKI and HOMA-IR with anthropometry and lipid profile (n=105)

Parameters	Mean ± SD	QUICKI		HOMA-IR	
		R-value	p-value	R-value	p-value
Weight	$62.54 \pm 8.52$	-0.29	0.002	0.48	< .00001
Waist circumference	$86.48 \pm 8.98$	-0.09	0.32	0.14	0.12
BMI	24.02 ± 3.25	-0.17	0.07	0.51	< .00001
SBP	$116.95 \pm 15.49$	-0.14	0.12	0.13	0.16
DBP	75.14 ± 10.36	-0.12	0.20	0.51	< .00001
Triglycerides	$120.60 \pm 29.30$	-0.51	< .00001	0.88	< .00001
Total cholesterol	$169.35 \pm 23.81$	-0.42	< .00001	0.47	< .00001
HDL	50.35 ± 11.00	0.43	< .00001	-0.47	< .00001
LDL	99.69 ± 28.52	-0.44	< .00001	0.43	< .00001

We have performed an unpaired t-test for insulin resistance indices, and the mean and standard deviation were calculated. QUICKI has mean of 0.46 and standard deviation of  $\pm 0.086$ , while HOMA-IR has mean of 1.35 and standard deviation of  $\pm 0.25$ .

Fig. 1a is a Scatter Plot Interpretation. The dots show individual values of LDL (x-axis) vs. HOMA-IR (y-axis). The trendline slopes upward, indicating that as LDL levels increase, HOMA-IR tends to rise. This suggests there is a positive correlation between LDL and HOMA-IR, indicating that higher LDL levels are associated with increased insulin resistance. Fig. 1b is a scatter plot showing a strong positive correlation between triglycerides and HOMA-IR, indicating that higher triglyceride levels are closely associated with increased insulin resistance. Fig. 1c is a scatter plot demonstrating a mild positive correlation between cholesterol and HOMA-IR, suggesting that higher cholesterol levels are modestly associated with increased insulin resistance.

Fig. 1d is a scatter plot that indicates a negative correlation between HDL and HOMA-IR, showing that higher HDL levels are associated with lower insulin resistance, which highlights HDL's protective role in metabolic health. Fig. 1e,f,g, QUICKI showed negative correlations with total cholesterol, triglycerides, and LDL, indicating that higher levels of these atherogenic lipids are associated with reduced insulin sensitivity. This supports their role in worsening metabolic health. Fig. 1h is a scatter plot showing a positive correlation between HDL cholesterol and QUICKI, indicating that higher HDL levels are associated with improved insulin sensitivity, reflecting a beneficial metabolic effect.

Our results show that higher LDL, triglycerides, and total cholesterol are linked with increased HOMA-IR and reduced QUICKI, indicating worsening insulin resistance. In contrast, higher HDL improved QUICKI and reduced HOMA-IR, reflecting protective effects.

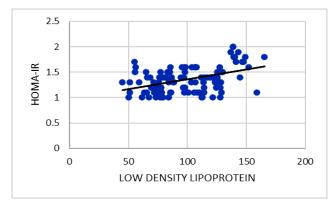


Fig. a: Correlation between HOMA-IR and LDL

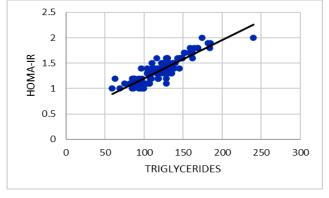
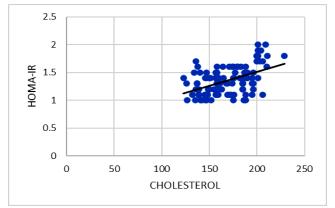


Fig. b: Correlation between HOMA-IR and Triglycerides





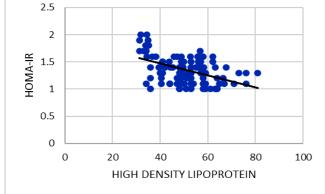
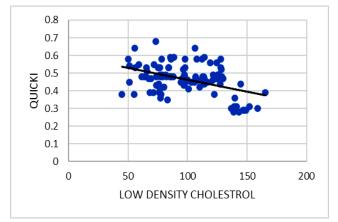


Fig. c: Correlation between HOMA-IR and cholesterol

Fig. d: Correlation between HOMA-IR and High **Density Lipoprotein** 



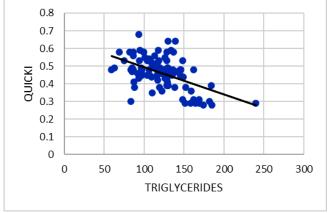
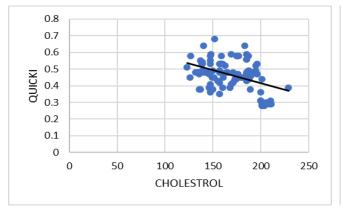


Fig. e: Correlation between QUICKI and LDL

Fig. f: Correlation between QUICKI and Triglycerides



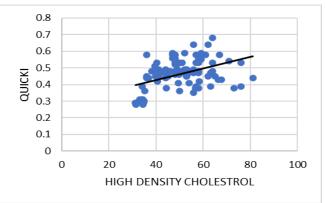


Fig. g: Correlation between QUICKI and Cholesterol

Fig. h: Correlation between QUICKI and HDL

Fig. 1 (a-h): Correlation of HOMA-IR with Lipid and Metabolic Parameters

# **DISCUSSION**

QUICKI and HOMA are based on fasting glucose and insulin values; hence, these indices primarily reflect hepatic insulin sensitivity [4]. Our study results for anthropometric indicators were like previous reports, where QUICKI showed a negative correlation with weight, waist circumference and blood pressure [5]. Analysis of lipid parameters in relation to insulin resistance indices revealed consistent patterns. HOMA-IR showed positive correlations with LDL, triglycerides, and total cholesterol, with the strongest association observed for triglycerides, while HDL demonstrated a negative correlation, suggesting a protective role [3,5]. Conversely, QUICKI exhibited opposite trends, with negative correlations to LDL, triglycerides, and total cholesterol, and a positive correlation with HDL [3,5]. Collectively, these findings highlight that atherogenic (LDL, triglycerides, total cholesterol) detrimental to insulin sensitivity, whereas HDL exerts a beneficial, protective metabolic effect.

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A study conducted by Yaser Mirzaalian et al. showed a negative correlation between TG and QUICKI and a positive correlation between TG, SBP and HOMA-IR [5]. In Michael Vogeser et al. study, results show that fasting insulin and the HOMA-IR decreased significantly in obese individuals [2] but in our study, we observed no such similar finding. Another study conducted by Shengfu Chen et al. there was positive correlation between QUICKI and HDL [10], which is like our study.

An adolescent study conducted by Morais et al indicates a positive correlation between HOMA-IR and BMI, which is like our study [11]. The study also has a significant moderate correlation between body mass index and HOMA-IR in the group with altered blood pressure. A study conducted by Sergio Martinez-Hervas et al showed a lower prevalence of hypertension and abdominal obesity in non-IR individuals. Whereas the IR state (IR post-OGTT and basal IR) showed increased prevalence of a cluster of cardiovascular disease risk factors [12]. Our findings are consistent with previous evidence linking adiposity to insulin resistance. Tabata et al. reported that a one standard deviation increase in BMI and waist circumference was associated with a 42.0% and 43.7% rise in HOMA-IR, respectively, highlighting the strong influence of both general and central adiposity [13].

Similarly, in our study, HOMA-IR showed a significant positive correlation with weight and non-significant positive correlations with waist circumference, BMI, systolic blood pressure, and diastolic blood pressure. QUICKI demonstrated inverse relationships, with a significant negative correlation with weight and nonassociations with significant negative other anthropometric and blood pressure measures [13]. This complementary pattern reflects the mathematical relationship between the two indices: both are derived from the same glucose-insulin data but differ in formulation, leading to HOMA-IR representing resistance and QUICKI representing sensitivity, say the study conducted by Milos Zarkovic et al. Taken together, these findings support the robustness of our results and reinforce the role of adiposity in impairing insulin sensitivity [14].

The findings in our study align with the previous report of Singh et al., showing strong associations of HOMA-IR with BMI and waist circumference, and a weaker link with WHR. Like earlier studies, we observed triglycerides as the lipid parameter most strongly correlated with HOMA-IR. Unlike those studies, however, HDL in our analysis showed a negative trend with HOMA-IR, suggesting a potential protective role [15]. Another study conducted by Ramachandran et al. says children with a family history of diabetes or overweight showed higher HOMA-IR and clustered risk factors. Similarly, our study found that excess weight and adiposity were strongly linked to insulin resistance [16].

In general, our study was conducted on young adults of both genders and the relationships of both insulin resistance indices with anthropometric and cardiometabolic indicators were investigated and compared. This study has some limitations, as it's a cross-sectional study, the cause-and-effect relationship between the variables is impossible to establish. We also did not collect data on diet and physical activity, which can have a role as confounders. The other disadvantage is that our sample size was small.

#### **CONCLUSIONS**

In conclusion, the present study conducted in 105 subjects from the age of 15 to 25, all anthropometric including weight, BMI indicators, circumference, had significant negative correlations with QUICKI and significant positive correlations with HOMA-IR. Moreover, among cardiometabolic indicators, there was a significant negative correlation between TG and QUICKI, whereas systolic blood pressure and TG had a significant positive correlation with HOMA-IR. Therefore, in our study correlating both insulin indices parameters, appearance is indistinguishable.

#### **ACKNOWLEDGMENTS**

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## **CONTRIBUTION OF AUTHORS**

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Supervision- Ganesan Subramanyam

Materials- Emi Goldy E

Data collection- Emi Goldy E

Data analysis and interpretation- Aishwarya Guru, Rijoe Rajulin

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