



## Assessment of Insulin Resistance And Sensitivity By Homa-Ir And Quicki in Patient With Metabolic Syndrome & Type-2 Diabetes Mellitus

## KEYWORDS

Metabolic Syndrome, Type-2 Diabetes Mellitus, HOMA-IR, QUICKI.

**Dabir Shakib**

Department of biochemistry M.G.M  
Medical college Kamothe, Navi  
Mumbai – 410 209

**Badade ZG**

Director Professor, MGM Medical  
College, MGM Institute of Health  
Sciences, Kamothe, Navi Mumbai –  
410 209

**Yadav Shashi RM**

MGM Medical College, Navi  
Mumbai-410209

**ABSTRACT**

*Insulin Resistance (IR) is a condition in which the body produces insulin but does not use it effectively. When people have insulin resistance, glucose rises in the blood instead of being absorbed by the cells, leading to type-2 diabetes or pre-diabetes.*

*Insulin resistance is an assurance of metabolic syndrome. It is important to identify Insulin resistance as it is early stage before development of diabetes mellitus (Type-2). Insulin sensitivity is inverse of insulin resistance. The standard method to measure Insulin resistance is Homeostasis Model Assessment of Insulin Resistance (HOMA-IR) and for Insulin sensitivity it is Quantitative Insulin Sensitivity Check Index (QUICKI). Therefore present study is to assess insulin resistance and sensitivity by HOMA-IR and QUICKI.*

**OBJECTIVES:** *The aim of the present study is to assess insulin resistance and sensitivity by HOMA-IR and QUICKI in patients with Metabolic Syndrome and Type-2 Diabetes Mellitus.*

**MATERIALS AND METHODS:** *Total 120 volunteers were enrolled in present study, carried out in the Department of Biochemistry and Department of General Medicine, MGM Medical College, Navi Mumbai. Total 120 volunteers were divided into three groups. Group-I included 40 metabolic syndrome patients as per NCEP ATP III criteria. Group-II included 40 Type-2 Diabetes mellitus patients as per WHO criteria and Group-III included 40 healthy individuals. BMI, Waist/Hip ratio of subjects were calculated. Blood samples were collected from vein under condition of 12 hours of fasting. Triglyceride and High density lipoprotein levels were also estimated in all subjects.*

**RESULTS:** *The significant difference was observed in BMI, Waist/Hip ratio, TG and HDL in control and study groups ( $p \leq 0.001$ ). The scatter diagram of HOMA-IR and QUICKI shows negative correlation in patients with metabolic syndrome. ( $r = -0.918$ ,  $p \leq 0.0001$ ) and also with Type-2 Diabetes mellitus ( $r = -0.949$ ,  $p \leq 0.0001$ ).*

**CONCLUSION:** *Present study shows reciprocal relationship between HOMA-IR and QUICKI in metabolic syndrome and type-2 diabetes mellitus.*

**INTRODUCTION:**

Insulin resistance is a physiologic state in which the ability of target tissues (e.g., muscle, liver, fat) to respond to the normal actions of insulin is diminished. Consequently, the ability of insulin to promote glucose uptake, inhibit hepatic glucose production, and suppress lipolysis in target tissues is decreased.<sup>(1)</sup>

When people have insulin resistance, glucose rises in the blood instead of being absorbed by the cells, leading to type-2 diabetes or pre-diabetes. Most people with insulin resistance are not aware of it for many years-until they develop type-2 diabetes. A serious, lifelong disease Insulin resistance can lead to a variety of serious health disorders. If insulin resistance is detected at early stage, the consequences of IR can be prevented.<sup>(2)</sup>

The insulin resistance syndrome consists of the co-occurrence of metabolic risk factors for type-2 diabetes and cardiovascular disease, including overall obesity, central obesity, dyslipidemia (characterized by elevated levels of triglycerides and low levels of high-density lipoprotein cholesterol), hyperglycemia and hypertension. Using criteria proposed by the National Cholesterol Education Program Adult Treatment Panel III, national survey data suggest the insulin resistance syndrome is very common, affecting about 24% of US adults aged greater than 20 years.<sup>(3)</sup>

In insulin resistance, muscle, fat, and liver cells do not re-

spond properly to insulin and thus cannot easily absorb glucose from the blood stream. As a result, the body needs higher levels of insulin to help glucose to enter cells. The beta cells of Langerhans produce more insulin to keep with this increased demand. As long as the beta cells are able to produce enough insulin to overcome the insulin resistance, blood glucose levels stay in the healthy range. Over time, insulin resistance can lead to type-2 diabetes and pre-diabetes because the beta cells fail to keep with the body's increased need for insulin. Without enough insulin, excess glucose builds up in the blood stream, leading to diabetes, pre-diabetes, and other serious health disorders.<sup>(2)</sup>

The homeostasis model assessment is a method which is used to quantify insulin resistance, developed by Matthews et al in 1985.<sup>(4)</sup> It is calculated by multiplying fasting plasma insulin (FPI) to fasting plasma glucose (FPG), and dividing by 405,

$$\text{HOMA-IR} = (\text{FPI} \times \text{FPG}) / 405$$

QUICKI: It is a simple, accurate method for assessing insulin sensitivity in humans and is derived using the inverse of the sum of the logarithms of the fasting insulin and fasting glucose:

$$1 / [\log (\text{fasting insulin } \mu\text{U/mL}) + \log (\text{fasting glucose mg/dL})].^{(5)}$$

QUICKI provides consistent and precise index of insulin sensitivity (6, 7-8)

The purpose of this study is to assess Insulin resistance and sensitivity by HOMA and QUICKI in patients with metabolic syndrome and type-2 diabetes mellitus

**AIM:**

To assess insulin resistance and sensitivity by HOMA-IR and QUICKI in patients with metabolic syndrome and type-2 diabetes mellitus

**OBJECTIVES:**

- To estimate fasting plasma glucose, fasting plasma insulin and lipid profile.
- To calculate insulin resistance by the formula:
- $HOMA-IR = [Fasting\ glucose\ (mg/dL) \times Fasting\ insulin\ (\mu U/mL)] / 405$
- To calculate insulin sensitivity by the formula:
- $QUICKI = 1 / [\log (fasting\ insulin\ \mu U/mL) + \log (fasting\ glucose\ mg/dL)]$

**MATERIALS AND METHODS:**

- Necessary approval from the Institutional Ethics Committee was obtained before initiating the study.
- **Study site** - The study was conducted in the Departments of Biochemistry and Department of General Medicine, MGM Medical College & Hospital, Navi Mumbai.
- **Study period** - The study was an observational study completed over a period of 12 months from February 2014 to February 2015.
- **Study design** : Prospective, observational
- **Sample size**: 120 volunteers were divided into three groups:
- **Group-I**: 40 patients with metabolic syndrome as per NCEP ATP III criteria
- **Group-II**: 40 patients with type-2 diabetes mellitus as per WHO criteria
- **Group-III**: 40 healthy individuals as control.
- **Exclusion criterion**: Patients seriously ill or with any other endocrinological disorder other than diabetes were excluded. Measurements of height and weight were done with subjects standing. Body mass index was calculated as weight in kg divided by height in meter square.
- **Study Procedure**: Blood samples were collected for Fasting Plasma Glucose, Triglyceride, HDL cholesterol and Fasting Plasma Insulin.

**STATISTICAL ANALYSIS:**

Data is presented as mean ± SD; t-test was used to compare BMI, W/H ratio, TG, HDL between patients and controls. The correlation of HOMA-IR and QUICKI was determined by Pearson correlation coefficient and scatter diagram was obtained.  $P \leq 0.05$  was considered statistically significant.

**RESULTS:**

Table-1 show anthropometric and clinical characteristics of study and control group. There are significant differences in the values of BMI, W/H Ratio, TG and HDL levels in study and control group  $p \leq 0.01$ . Comparison of HOMA-IR and QUICKI was done in metabolic syndrome and type-2 diabetes mellitus. The scatter diagram of HOMA-IR and QUICKI in metabolic syndrome ( $r = -0.918, p \leq 0.0001$ ) and type-2 diabetes mellitus ( $r = -0.949, p \leq 0.0001$ ) shows negative correlations.

**Table No.1: Descriptive statistics for different groups**

Parameters	Metabolic Syndrome Group I (mean ± SD)	T-2 Diabetes Mellitus Group II (mean ± SD)	Healthy Individual Group III (mean ± SD)
BMI	30.23 ± 3.65**	27.02 ± 4.43**	22.78 ± 1.88
W/H Ratio	0.99 ± 0.06**	0.98 ± 0.06**	0.84 ± 0.06
TG	179.21 ± 27.41**	161.15 ± 78.82**	114.1 ± 28.07
HDL	37.22 ± 4.99**	39.75 ± 9.82**	47.93 ± 9.47

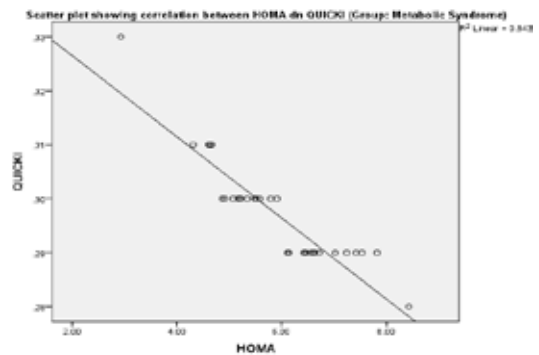
\*\*  $P \leq 0.01$  Statistically significant

**Table No.2: Correlation between HOMA-IR and QUICKI in patients with metabolic syndrome**

	QUICKI (m=0.297)	
HOMA-IR (m=5.898)	Pearson Correlation	-.918**
	Sig. (2-tailed)	0
	N	40

\*\* . Correlation is significant at the 0.01 level (2-tailed).

The above table shows correlation between HOMA and QUICKI in patients with metabolic syndrome. The result of Karl Pearson's correlation coefficient indicates a very high degree negative correlation between HOMA-IR and QUICKI. ( $r = -0.918, p \leq 0.0001$ ). The result is also shown in the scatter diagram below.

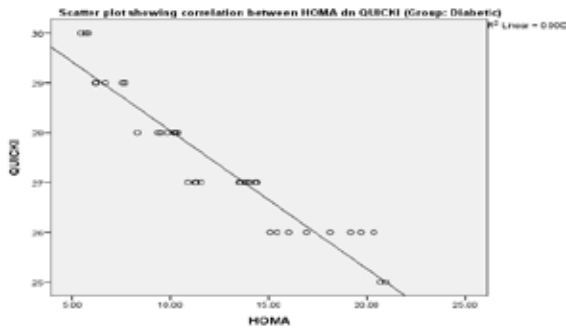


**Table No. 3: Correlation between HOMA-IR and QUICKI in type-2 diabetes mellitus**

	QUICKI (m=0.274)	
HOMA (m=12.271)	Pearson Correlation	-.949**
	Sig. (2-tailed)	0
	N	40

\*\* . Correlation is significant at the 0.01 level (2-tailed).

The above table shows correlation between HOMA-IR and QUICKI in diabetic patients. The result of Karl Pearson's correlation coefficient indicates a very high degree negative correlation between HOMA-IR and QUICKI. ( $r = -0.949, p \leq 0.0001$ ). The result is also shown in the scatter diagram below.



### DISCUSSION:

In present study, we calculated insulin resistance and sensitivity by HOMA-IR and QUICKI. Comparison of HOMA-IR and QUICKI in patients with metabolic syndrome shows negative correlation. ( $r=-0.918$ ,  $p\leq 0.0001$ ). Our results are in correspond with **Conwell L S** et al. (2004) showed HOMA-IR was in significantly negative correlation with QUICKI ( $p\leq 0.01$ ).<sup>(9)</sup>

Comparison of HOMA-IR and QUICKI in patients with type-2 diabetes mellitus shows negative correlation. Similar results was carried out by **Foss-Freitas** et al. (2004) in their study of 167 subjects observed increased HOMA ( $1.88 \pm 0.14$ ) and decreased QUICKI ( $0.36 \pm 0.004$ ). When compared between type-2 diabetes and healthy individuals which is concurrent to the our study.<sup>(10)</sup>

### CONCLUSION:

Present study demonstrates that HOMA-IR is negatively correlated with QUICKI for type-2 diabetes mellitus and metabolic syndrome subjects, since insulin resistance is inversely related to insulin sensitivity. The measurement of insulin resistance and insulin sensitivity by the two different methods, HOMA-IR and QUICKI respectively, shows reciprocal correlation between themselves. Thus the observation from our study in different groups such as metabolic syndrome and type-2 diabetes mellitus, HOMA and QUICKI shows reciprocal relationship.

### REFERENCES:

1. National heart lung and blood institute health information for public metabolic syndrome nov 3, 2011
2. [http://diabetes.niddk.nih.gov/dm/pubs/insulinresistance/Insulin\\_Resistance\\_Prediabetes-508](http://diabetes.niddk.nih.gov/dm/pubs/insulinresistance/Insulin_Resistance_Prediabetes-508).
3. Mechanisms of obesity-associated insulin resistance: many choices on the menu Mohammed Qatanani and Mitchell A. Lazar Genes & Dev. 2007. 21: 1443-1455
4. Matthews DR, Hosker JP, Rudenski AS, Naylor BA, Treacher DF, et al. Homeostasis model assessment: insulin resistance and  $\beta$ -cell function from fasting plasma glucose and insulin concentrations in man. Diabetologia. 1985; 28:412-419.
5. Katz A, Nambi SS, Mather K, Baron AD, Follmann DA, Sullivan G, Quon MJ. Quantitative insulin sensitivity check index: a simple, accurate method for assessing insulin sensitivity in humans. J Clin Endocrinol Metab. 2000; 85(7):2402-10.
6. DeFronzo RA, Tobin JD, Andreas R. Glucose clamp technique: a method for quantifying insulin secretion and resistance, Am J Physiol 1979; 237:E214-E223.
7. Chen H, Sullivan G, Lue LQ, Katz A, Quan MJ. Quicki is a useful index of insulin sensitivity in subjects with hypertension. Am J Physiol Endocrinol Metab 2003; 284:E804-E812.
8. H, Sullivan G, Quan MJ. Assessing the predictive accuracy of QUICKI as a surrogate index for insulin sensitivity using a calibration model. Diabetes 2005; 54:1914-1925.

9. Conwell LS, Stewart G, Wendy J, Jennifer A. Index of insulin resistance and secretion in obese children and adolescent. Diabetes Care 2004; 27:314-319.
10. MC Fross-Freitas and MC Foss. Comparison of the homeostasis model assessment and quantitative insulin sensitivity check index with data from forearm metabolic studies for the in vivo assessment of insulin sensitivity. Braz J Med Biol Res 2004; 37(5)