



The Correlation between HOMA-IR, QUICKI and HbA1c as predictors of type 2 diabetes in a Moroccan population

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ABSTRACT

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Introduction : HOMA-IR and QUICKI are indexes that estimate insulin resistance and sensitivity.

Objective : This study assesses their correlation with HbA1c to determine their potential as predictors of type 2 diabetes.

Material and methods : The study group comprise 91 subjects divided into three subgroups: G0 (n=52) healthy individuals, G1 (n=21) subjects with controlled T2DM and G2 (n=18) subjects with unbalanced T2DM. The HOMA-IR and QUICKI indices are calculated using a mathematical formula based on fasting blood glucose and insulin levels.

Results : The correlation between HbA1c and HOMA-IR showed a significant correlation in all 3 groups of patients : G0 ($r = 0.47, p < 10^{-3}$), G1 ($r = 0.42, p = 0,05$) et G2 ($r = 0.81, p < 10^{-3}$). Moderate correlations were found in groups G0 and G1 : G0 ($r = 0.47, p < 10^{-3}$), G1 ($r = 0.42, p = 0,05$). A weak correlation was found in group G2.

Conclusion : HOMA-IR and QUICKI show potential as early indicators of the onset of type 2 diabetes. Their use could improve the screening.

KEYWORDS:

HOMA-IR, QUICKI, insulin resistance, type 2 diabetes

INTRODUCTION

Type 2 diabetes (T2DM), a chronic metabolic disease manifested by persistent elevation of blood glucose levels, is a major international public health issue.

Despite significant progress in prevention and treatment, the number of people with diabetes continues to rise at an alarming rate. The WHO estimates that there are already more than 420 million cases worldwide, and predicts an increase to 570 million by 2030 and 700 million by 2045 [1].

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The complications of T2DM and its complexity, linked to the multiplicity of risk factors involved, both genetic and environmental, underline the urgent need to step up preventive and therapeutic measures.

Insulin resistance, which manifests itself as the need for extra insulin to achieve a normal physiological response, is a major risk factor for T2DM. This metabolic alteration, central to the pathophysiology of the disease [2], is at the heart of prevention and screening strategies. Traditionally, glycated haemoglobin (HbA1c) has been used as a marker of glycaemic control over an average period of 2 to 3 months [3], and as a diagnostic or screening tool for diabetes [4], but it does not directly reflect insulin resistance.

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Given the close relationship between insulin secretion and its action on target tissues, it is desirable to assess these two phenomena concomitantly.

The indicators used include the HOMA-IR index (Homeostasis Model Assessment for Insulin Resistance) and QUICKI (Quantitative Insulin Sensitivity Check Index). These are estimates of insulin resistance and sensitivity, based on a mathematical model using fasting blood glucose and insulin levels [5].

In the present study, we propose to evaluate the correlation of these two indices with HbA1c to determine their potential as predictive factors for T2DM.

MATERIAL AND METHODS

91 individuals were recruited and subdivided into three groups according to glycemic control:

- Group 0 (n=52): healthy non-diabetic subjects with an age range comparable to that of the patients (39 to 78 years).
- Group 1 (n=21): diabetic patients with HbA1c levels between 6.1% and 8%, indicating good glycemic control.
- Group 2 (n=18): 18 diabetic patients with an HbA1c level above 8%, indicating poor glycemic control.

All patients and controls were sampled in the morning after prolonged fasting. The biological parameters studied are :

- **Glycated hemoglobin (A1c)**

The assay was performed out on whole venous blood taken from an EDTA tube, using high-performance liquid chromatography (HPLC) on ADAMS ARKRAY® analyser.

➤ **Blood glucose**

Blood glucose was determined on serum obtained after centrifugation of venous blood from a dry tube at 3600 rpm for ten minutes. The assay was performed using the hexokinase enzymatic method on Alinity Abbott® analyser.

➤ **Insulin levels**

Insulin levels were determined by chemiluminescence immunometry on Alinity Abbott® analyser.

➤ **The HOMA-IR index**

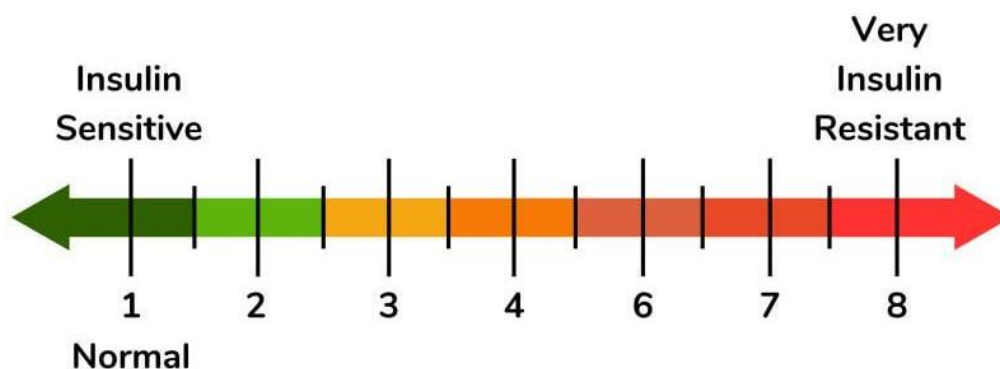
It was calculated using the formula : [Fasting blood glucose (mmol/L) × fasting insulin (mIU/ml)] / 22.5

Insulin resistance is defined by an HOMA-IR index > 2.4.

➤ **The QUICKI index**

It was calculated using the formula : $1 / [\log \text{fasting plasma glucose (mg/dl)} + \log \text{fasting plasma insulin } (\mu\text{U/ml})]$

Insulin sensitivity is considered to be significantly reduced when the QUICKI index is < 0.3.



The data were entered and analysed using Excel 2016 and MedCalc version 19.1. Graphs were obtained using MedCalc version 19.1 software. Results were expressed as means ± standard deviations. Pearson's coefficient (Pearson's r) was used to assess the correlation between parameters. A regression analysis was performed to assess the predictive potential of the two indices in relation to HbA1c. The threshold of statistical significance was set at $p < 0.05$.

RESULTS

The socio-demographic and biological characteristics of the patients are shown in **Table 1**. The mean age was 60.05 ± 8.57 years (with extremes ranging from 39 to 78 years) and the M/F sex ratio was 1.45.

Table 1 : Distribution of patients according to socio-demographic and biological characteristics.

Characteristics of the population (n=91)	
Average age (years)	$60,05 \pm 8,57$
Sex ratio (M/F)	1,45
HbA1c moyenne (%)	$7,24 \pm 2,15$

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Average blood glucose (g/l)	1,37 ± 0,69
Average blood Insulin (mUI/l)	13,10 ± 9,16
Medium HOMA-IR	4,10 ± 2,87
Medium QUICKI	0,31 ± 0,03

In the study population as a whole, the HOMA-IR index was higher in men (5.02 ± 1.63 compared with 4.65 ± 2.15 in women) and in subjects aged over 60 (7.012 ± 1.63 compared with 4.14 ± 2.15 for the rest of the population).

According to **Table 2**, the study of the correlation between HbA1c and the HOMA-IR index showed a significant result

in all three groups of patients. As regards the QUICKI index, a better prediction was obtained in the group of non-diabetic subjects followed by patients with controlled diabetes, while no significant correlation was found in the group of patients with poor glycemic control.

Table 2 : Correlations between HbA1c and insulin resistance parameters in the 3 patient groups.

Group	Glucose	Insulin	HOMA-IR	QUICKI
0	$r = 0,54$ $p < 10^{-3}$	$r = -0,8$ $p = NS$	$r = 0,47$ $p < 10^{-3}$	$r = -0,51$ $p < 10^{-3}$
1	$r = 0,49$ $p < 10^{-3}$	$r = -0,45$ $p = NS$	$r = 0,42$ $p < 10^{-3}$	$r = -0,43$ $p = 0,04$
2	$r = 0,60$ $p < 10^{-3}$	$r = 0,31$ $p = NS$	$r = 0,15$ $p = NS$	$r = 0,23$ $p = NS$

The regression lines between HbA1c and HOMA-IR in the three groups studied are presented in **Figure 1**, and show an inverse relationship in poorly controlled diabetics.

Our study revealed a negative and statistically significant correlation ($r = -0.85$, $p < 0.001$) between the HOMA-IR index and the QUICKI index in the three groups of patients (**Figure 2**).

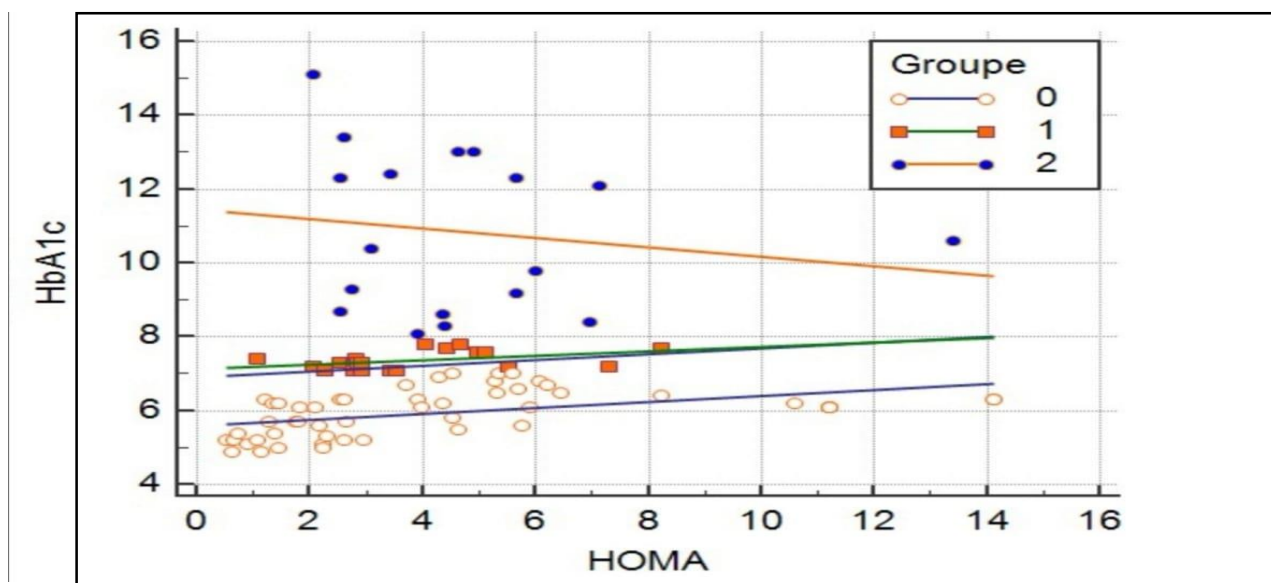


Figure 1 : HbA1c=f(HOMA) regression lines for healthy subjects and patients with good and poor glycemic control.

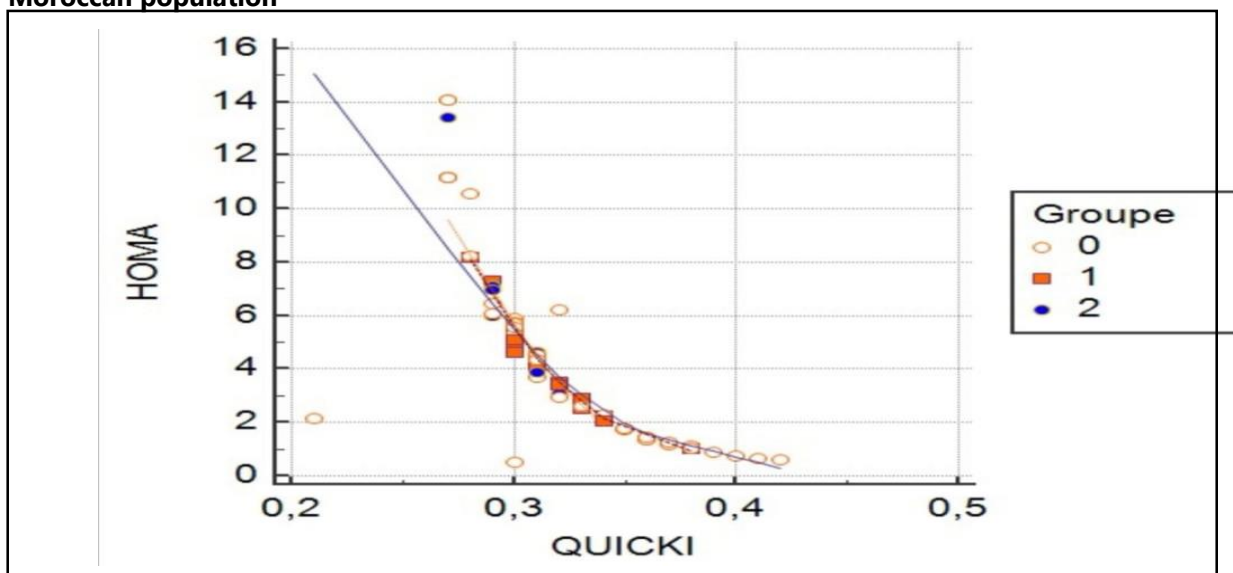


Figure 2 : HOMA=f(QUICKI) regression lines for healthy subjects and patients with good and poor glycaemic control.

DISCUSSION

This study assesses the performance of the HOMA-IR and QUICKI indices in predicting type 2 diabetes. The correlation of these indices with HbA1c was analysed and compared between three groups of patients according to their glycaemic control.

The results show significant correlations.

Indeed, we found moderate correlations in the non-diabetic and balanced diabetic groups, suggesting that these indices are useful tools for assessing insulin function at an early stage of glycaemic dysregulation.

In the study by Djoghlafl and Rahal, a higher HOMA-IR was found in the group of 1st degree relatives of T2DM patients compared with normoglycaemic control subjects [6], which confirms our results.

In the group of unbalanced diabetics, no significant correlation was found. This result raises several questions. On the one hand, it suggests that other vulnerability factors, such as genetic factors, glycaemic variability and heterogeneity of treatments [7,8], may play a more important role in glycaemic regulation in these patients.

The results showed that HbA1c was also correlated with QUICKI in the three groups studied. This indicates that the function of beta cells, which are responsible for insulin secretion, is the major determinant of HbA1c, i.e. improved beta cell function is associated with better glycaemic control.

These data are perfectly consistent with the findings of Al-Hakeim et al [9]. HbA1c also appears to be influenced by age. In the study by Kilpatrick et al, HbA1c levels rose from 3.98% to 4.44% between the ages of 20 and 70 ($r = 0.49$), while fructosamine levels did not vary with age [10]. This clearly suggests that glycation of haemoglobin increases with age, but not that of fructosamine.

Insulin levels were not significantly different between the three groups studied. Insulin levels are considered to be an indicator of insulin resistance, but have two major limitations:

the specificity of the assays, and the difficulty of interpretation in the event of insulin secretion deficiency [11]. Based on fasting serum insulin and glucose concentrations, the HOMA-IR model provides a quantitative estimate of insulin resistance [12], so fasting blood glucose is the key factor in insulin resistance in the mathematical models used [3].

In addition, our study showed that age, and in particular male gender, are associated with a progressive alteration in insulin sensitivity, confirming existing data [13,14]. Other studies, however, suggest that age per se is not the main factor, and that changes in body mass and composition are also involved [15,16,17]. The study by Pitteloud et al suggests that a drop in testosterone levels may also be involved in the insulin resistance observed in the elderly [18].

There are a number of limitations to this study that need to be taken into account when interpreting the results. Firstly, the size of the samples per group, the factors other than insulin resistance that are likely to influence the HOMA-IR and QUICKI indices, in particular circadian variations in insulin secretion, age, sex, obesity, lifestyle, and so on. Finally, a better understanding of beta cell function, particularly through mathematical models such as the HOMA- β index or promising genomic approaches, seems essential for refining the prediction of type 2 diabetes.

CONCLUSION

The HOMA-IR and QUICKI indices are correlated with HbA1c and show potential as early indicators of the onset of type 2 diabetes. Their use could improve screening. However, their usefulness remains limited. In fact, these mathematical models provide a static assessment of insulin sensitivity at a given time, without taking into account the dynamics or factors involved in the development of T2DM.

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