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*Short Communication*

# Insulin resistance and blood pressure in Nigerian type 2 diabetic patients

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**Considerable interest has been generated on the possible role of insulin resistance (IR) in the etiology and sustenance of hypertension among type-2 diabetic subjects. There is paucity of data on this relationship on African diabetic patients. The aim of this study is to show the relationship between homeostasis model assessment (HOMA) derived insulin resistance (IR) scores and blood pressure among type-2 diabetic patients in Nigeria. Anthropometric data, age, sex and duration of diagnosis of diabetes mellitus were taken. Blood pressures were measured using standard methods. Fasting plasma insulin (FPI) levels were measured using an ELISA technique. Fasting plasma glucose (FPG) levels were measured using standard methods. Using the HOMA method, insulin resistance scores were derived for each subject. Forty type-2 diabetic patients 13 (32.5%) of whom were hypertensive and 27 (67.5%) of whom were normotensive were studied. Mean HOMA-IR scores were 1.96 ± 1.04 (range 0.49 – 2.92) among diabetic-hypertensive individuals and 2.28 ± 1.89 (range 0.39 – 7.6) among normotensive-diabetic patients (p = 0.5350). There was no significant correlation between mean arterial blood pressure (MAP) and HOMA-IR score among the normotensive group was (r = +0.087, p > 0.5). There is no statistically significant relationship between mean arterial blood pressure and HOMA derived Insulin resistance in type-2 diabetic patients in this study.**

**Key words:** Insulin resistance, blood pressure, type-2 diabetes, Nigerians, homeostasis model assessment (HOMA).

## INTRODUCTION

Cardiovascular disease especially hypertension is commoner among type-2 diabetic patients than the general population (UK prospective diabetes study group, 1998). Several reasons have been advanced to explain the higher prevalence of hypertension among type-2 diabetic subjects including the commonality of such risk factors as obesity and sedentary life styles in the etiology of both conditions (Sjostrom et al., 1999). Furthermore, it has been suggested that either insulin resistance or the attendant hyperinsulinaemia could provide the unifying mechanism for these observations (Haffner, 1999; Defronzo and Ferrarini, 1991). However, racial factors seem to be important in the role of insulin resistance in the etiology of cardiovascular disease (Sa'ad et al., 1991). Although extensive literature has been generated on the possible role of insulin resistance in the etiology sustenance of hypertension amongtype-2 diabetic subjects else where, there is paucity of such data among Africans generally, and Nigerians in particular.

Recently, the HOMA method has been revalidated as a reliable method to assess insulin resistance in clinical practice as the HOMA IR score has been shown to closely mirror insulin resistance values obtained by the euglycaemic glucose clamp technique in the assessment of insulin sensitivity (Bonora et al., 2000). More importantly, and more recently, it has been shown that HOMA estimated insulin resistance is an independent predictor of cardiovascular disease in type-2 diabetic subjects (Bonora et al., 2002). This study reports the relationship between insulin resistance derived by the homeostasis model assessment (HOMA) method, and blood pressure among Nigerian type-2 diabetics.

## METHODS AND SUBJECTS

Type 2 diabetic patients attending the diabetic clinic and had 'good' glycaemic control, defined as fasting blood sugar (FBS) of 4.4 to

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**Table 1.** Characteristics of type-2 diabetic patients studied.

| | |
|---|---|
| **Age (years)** | 49.4± 9.7 |
| **BMI (Kg/M$^2$)** | 24.93± 4.43 |
| **WHR** | 1.03 ± 0.08 |

6.7 mmol/l, and or a 2 h postprandial blood sugar of 4.4 to 8.9 mmol/l and 'acceptable' glycaemic control (FBS of 6.7 to 7.8 mmol/l and or 2 HPP of 8.9 to 10.0 mmol/l) (Williams, 1994); on at least three clinic visits while on dietary therapy alone, or dietary therapy in addition to oral hypoglycemic agent(s) formed the subjects of this study.

Thirty-six healthy, age, sex and socio economic status matched volunteers who had no personal or family history of diabetes mellitus or hypertension were recruited to serve as controls. The exclusion criteria were clinical evidence of any illness, personal or family history of diabetes mellitus or hypertension, current use of any form of medication and engagement in competitive sport

Information on age, sex and anthropometric measures were obtained from all patients and control subjects. Weights (in Kg) were taken with only undergarments to the nearest 0.5 kg. Heights (in meters) were taken to the nearest 0.5 cm with subjects standing erect without shoes or headgear. Body Mass Index (BMI) was derived by dividing the weight by the square of the height (World Health Organization Expert Committee, 1995). Blood pressures (BP) were measured in a recumbent position after 10 min of rest and repeated after 5 min. Phases 1 and 5 were respectively taken as systolic BP (SBP) and diastolic BP (DBP). Mean of two values taken as subjects BP. Mean arterial blood pressure (MAP) were computed from the following formula MAP = 1/3 pulse pressure +DBP. Hypertension was defined as current use of antihypertensive (s) or a BP of 140/90 mmHg (World Health Organization, 1999).

Following an overnight 10 - 12 h fast commencing between 21.00 to 22.00 h the preceding night, 5 ml of venous blood were drawn from each subject into EDTA treated tubes and promptly centrifuged. Aprotinin 200 KIU per ml of plasma (Verstraete, 1985) was added to an aliquot meant for insulin assay; this was kept at – 20$^{O}$C until analysis. Plasma glucose analysis was done within an hour of collection using a glucose oxidase method (Trinder, 1969). Plasma insulin assays were performed using a commercially available ELISA human insulin kit (DRG instruments Gmbh, Marburg, Germany, Cat no. EIA 2935). The kit has an inter-assay and intra-assay coefficients of variation of 5.2 and 4.8%, respectively, sensitivity of 99% for human insulin and no cross-reaction with pro insulin. Insulin resistance values were derived from the formula reproduced below using the homeostasis model assessment (HOMA) developed by Matthews et al. (1985).

HOMA-IR = [FPI (µU/L) X FPG (mmol/L)] / 22.5

Results are presented as mean ± standard deviation. Unpaired student's t -test was used to determine the differences between continuous variables while chi-square test was used for categorical variables. Pearson's correlation coefficient was used to define correlation between variables. The level of statistical significance in each case was taken as $P < 0.05$

## RESULTS

Forty type-2 diabetic patients and 36 healthy control volunteers were studied. Of the diabetic patients, 13(32.5%) were hypertensive, and 27(67.5%) were normotensive. The characteristics of diabetic patients and control volunteers are shown on Table 1.

The mean HOMA scores were respectively 1.96 ± 1.04 (range 0.49 – 2.92) among diabetic hypertensive patients and 2.28 ± 1.89 (range 0.39 – 7.6) among normotensive diabetic patients. The difference between the two groups were however not significant statistically ($p = 0.5350$). Furthermore, although the correlation between blood pressure and HOMA- IR score among the normotensive group was positive, it did not reach statistical significance ($r = + 0.087$, $p > 0.5$).

## DISCUSSION

The homeostasis model assessment method is cheap, simple, and relatively non-invasive. Only single fasting plasma insulin and the corresponding glucose concentrations are required to calculate HOMA-IR values. Recently, the method has been revalidated as a reliable method to assess insulin resistance in clinical practice as the HOMA IR score has been shown to closely mirror insulin resistance values obtained by the euglycaemic glucose clamp technique in the assessment of insulin sensitivity (Bonora et al., 2000). It is therefore appropriate in developing countries, where more complex dynamic tests such as the hyperinsulinaemic euglycaemic clamp studies' though the gold standard (Defronzo et al., 1979) but requiring more sophisticated equipment are lacking.

In this study there is no significant relationship between mean arterial blood pressure and the HOMA-IR score. This is contrary to observations in western societies where it has been observed that insulin resistance correlates with blood pressure and other cardiovascular risk factors (Haffner, 1999; Defronzo and Ferrarini, 1991; Bonora et al., 2002). This observation is however consistent with earlier findings of Sa'ad and co-workers, in Blacks and Pima Indians (Sa'ad et al., 1991). Several reasons including genetic factors could explain these differences.

The heterogeneous nature of type- 2 diabetes may be the most plausible explanation for this observation. There is evidence to suggest that type-2 diabetes in the black populations is to some extent etiologically and phenoltypically different from the classic type-2 diabetes described in Caucasian populations. Whereas Martin et al. (1992), in a follow up study of normoglycaemic offspring of Caucasian diabetic patients at the Joslin Diabetes Center, established that decrease in insulin sensitivity precedes and strongly predicts the development of type 2 diabetes. Banerji and Lebovitz (1989) have shown that up to 59% of African-American type 2 diabetic patients in the USA do not exhibit insulin resistance even when type 2 diabetes is manifested, similar to findings of Chaiken and co-workers (Chaiken et al., 1993). Similarly, using HOMA method only 40% of type-2 diabetic Nigerians were adjudged to exhibit insulin resistance (Bakari and Onyemelukwe, 2005). Furthermore, there are other strong ar-

guments from Ghana (Amoah et al., 2002), Cameroon (Mbanya et al., 2000) and Nigeria (Bakari and Onyemelukwe, 2006) suggesting that Pancreatic beta- cell abnormalities may be the predominant and most significant underlying factor in the etiology of type-2 diabetes in West African patients.

The absence of significant correlation between HOMA-IR scores and mean arterial blood pressures suggests that other mechanisms may be more important than insulin resistance in the etiology of hypertension in type-2 diabetic patients in this study.

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