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Glucose Tolerance in Non-Diabetic Adult Subjects of an Urban West-African Population

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Summary: The present study was carried out to determine glucose tolerance and insulin sensitivity in adult subjects of a west African population. 103 subjects recruited in the town of Cotonou were included in the study. After anthropometric measurement, they were subjected to an oral glucose tolerance test (OGTT). Serum glucose and insulin levels were determined throughout the OGTT. Homeostatic model of assessment of insulin resistance (HOMA-IR), MATSUDA insulin sensitivity index (MATSUDA-ISI) and insulinogenic index (IGI) have been determined to evaluate insulin sensitivity and beta cells function. Normal glucose tolerance (NGT), isolated impaired fasting glucose (IFG), isolated impaired glucose tolerance (IGT) and combined glucose intolerance (CGI) were observed in respectively 53.40%, 1.94%, 35.92% and 8.74% of subjects. The prevalence of IFG and or IGT (IFG/IGT) was higher in obese subjects (66.67%) than in subjects with normal BMI (41.17%). Fasting hyperinsulinemia was observed in 82% of subjects. Mean values of HOMA-IR were not significantly different in NGT (6.86 ± 0.7) and in IFG/IGT subjects (7.47 ± 0.57). In contrast to HOMA-IR, mean value of Matsuda-ISI was significantly lower in IFG/IGT than in NGT subjects (1.47 ± 0.1 versus 1.96 ± 0.13, $p<0.01$). Matsuda-ISI values were also significantly lower in obese subjects (1.33 ± 0.12) than in subjects with normal BMI (1.93 ± 0.13). The mean insulinogenic index value in IFG/IGT subjects (42.5 ± 4.36) was not significantly different of that in NGT subjects (50.3 ± 5.21). These data show that the glucose tolerance disorders observed in subjects of the present study are more related to a decrease in insulin sensitivity than to an alteration of the beta cells function.

Keywords*:* Glucose tolerance, Insulin sensitivity, Beta cell function, Overweight, Diabetes

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INTRODUCTION

Diabetes mellitus constitutes an important health problem worldwide. According to the international diabetes federation (IDF), about 485 million of people suffered of diabetes in the world in 2015 (IDF, 2015).

With changes in lifestyle characterized by sedentary and changes in nutrition, a rapid progression of diabetes prevalence is observed especially in low and intermediate income countries. In Africa, about 14.2 million of people were diabetics in 2015 (IDF, 2015). In Benin, diabetes prevalence rate increased from 1.1% in 2001 to 2.3% in 2008 (Djrolo et al. 2003; Houehanou et al. 2015). In 2011, a prevalence rate of 4.6% has been reported in the town of Cotonou (Djrolo et al. 2012).

Amongst the two main types of diabetes, type II diabetes (T2D) is the most common (90%). This type II diabetes is preceded by a prediabetes state during which a progressive development of insulin resistance and a deterioration of insulin secretion by pancreatic beta cells occur and result in an abnormal glucose tolerance state (impaired fasting glucose and impaired glucose tolerance). Prediabetes, is a reversible state during which preventive actions must be implemented to avoid or delay onset to diabetes. Indeed, beneficial effects of exercise on glucose tolerance and insulin sensitivity have been reported by many authors (Nelson and Horowitz, 2014; Savoye et al. 2014; AbouAssi et al. 2015). Pharmacological interventions, especially preventive treatment with metformin, have also been reported to improve prediabetes conditions (Hostalek et al. 2015). Screening of prediabetes and implementation of appropriated preventive measure could thus prevent the rapid increase in the prevalence of diabetes.

In sub-Saharan Africa, recently, prevalence rates of impaired glucose tolerance of 8.1%, 11.2% and 12% have been reported respectively in Angola, South

Africa and Kenya (Christensen, 2009; Evaristo-Neto, 2010; Peer, 2012). In Benin, no study on impaired glucose tolerance and its pathophysiological mechanisms has never been published. In the present study, glucose tolerance in relationship with insulin sensitivity and pancreatic beta cell function in a Benin urban adult population have been investigated.

MATERIALS AND METHODS

Ethical approval

The study was approved by the Comité d'Ethique de la Recherche de la Faculté des Sciences de la Santé (reference No. 31-CER-FSS).

Subjects

Adult non-diabetic subjects of both sexes, living in the town of Cotonou and recruited from May to November 2012, were included in the study. After information session on diabetes, a screening for diabetes was made by measurement of glucose levels on capillary blood. 150 subjects with normal fasting capillary blood glucose level (<1.1g/l) were preselected to be included in the study. Amongst them, 103 subjects effectively participated to the study. Prior to their participation, written consent forms were obtained from all subjects.

Exclusion criteria for subject recruitment

Diabetic subjects (subjects known diabetic and those diagnosed diabetic during the screening), pregnant women and subjects taking medication which could interfere with glucose metabolism were excluded from the study.

Clinical data collection

A questionnaire was used to collect data on medical story and lifestyle. Physical examination was performed and clinical variables such as, height, weight, waist and hip circumferences were measured. BMI (body mass index) was determined by dividing the weight (kg) by the square of height (m^2).

Oral glucose tolerance test (OGTT)

After an overnight fasting period of 12 hours, an oral glucose tolerance test was carried out. A venous blood sample was collected and subjects were orally administrated 75g of glucose. At 30, 60, 120 and 180 minutes of the OGTT, blood samples were also collected for the determination of glucose and insulin levels.

Glucose tolerance status were defined according to fasting blood glucose and blood glucose levels during OGTT as follows:

- Normal glucose tolerance (NGT): fasting blood glucose < 1.1g/l and blood glucose level at 2 hours of OGTT <1.4g/l
- Isolated impaired fasting glucose (IFG): fasting blood glucose value between 1.1g/l and 1.26g/l and blood glucose level at 2 hours of OGTT <1.4g/l
- Isolated impaired glucose tolerance (IGT): fasting blood glucose < 1.1g/l and blood glucose level at 2 hours of OGTT between 1.4g/l and 2g/l.
- Combined glucose intolerance (CGI): association of IFG and IGT, fasting blood glucose value between 1.1g/l and 1.26g/l and blood glucose level at 2 hours of OGTT between 1.4g/l and 2g/l.

(Hanefeld et al., 2003**)**

Determination of blood glucose and insulin levels

Blood glucose levels were measured by glucose oxidase assay on plasma obtained after blood centrifugation. Plasma insulin levels were measured by radioimmunoassay (RIA) using insulin CT kit (CISBIO Bioassays, France) according to the manufacturer instructions.

Insulin sensitivity/resistance indexes and β cell function

Homeostatic model assessment of insulin resistance (HOMA-IR) was calculated using fasting plasma glucose (mmol/l) and fasting plasma insulin (µU/ml) levels as follows:

$$\text{HOMA-IR} = \text{fasting plasma glucose} \times \frac{\text{fasting plasma insulin}}{22.5}$$

(Maniyappa et al., 2008**)**

MATSUDA insulin sensitivity index (MATSUDA - ISI) was determined as followed:

$$= \frac{10000}{\sqrt{[(\text{mean OGTT plasma glucose} \times \text{mean OGTT plasma insulin}) \times (\text{fasting plasma glucose} \times \text{fasting plasma insulin})]}}$$

(Matsuda and DeFronzo, 1999)

β cell function assessment

β cell function was assessed by determining the insulinogenic index (IGI) which is the ratio of the incremental insulin to glucose response over the first 30 minutes of the OGTT ($\Delta I0\text{-}30/\Delta G0\text{-}30$). **(**Abdul-Ghani et al., 2007**)**

Statistical analysis

Values are expressed as means ± SEM or as proportions. Comparison of proportions between groups was made by Chi-squared test (χ^2) and that of mean values by Student t test. Significant difference was set for $p<0.05$.

RESULTS

Subjects characteristics

The main characteristics of subjects included in the study are presented in table 1.

Fifty four of the 103 subjects were women (52.4%) and 49 were men (47.6%). Subjects were aged from 20 to 50 years and the mean age was 30.25 ± 0.76 years.

Mean body weight and BMI of subjects were respectively 71.43 ± 1.47 kg and 25.89 ± 0.52 kg/m^2. On BMI basis, 4 subjects (3.9%), who were all men, were underweighted, 51 subjects (49.5%) had a normal BMI, 24 (23.3%) were overweighed and 24 (23.3%) were obese. Obesity prevalence was, at least, twofold higher in women than in men (31.5% versus 14.3%, p<0.05). An increased obesity prevalence was also observed with age. It raised from 7.1% in subjects under 25 years, to 23.3% between 25 and 30 years, to 27.3% between 30 and 35 and to 39.1% in subjects over 35 years.

Mean value of waist circumference was 86.06 ± 1.18 cm. Abdominal obesity (elevated waist circumference higher than 94 cm in men and 80 cm in women) was observed in 46 subjects (44.6%). Abdominal obesity prevalence was at least three times higher in women than in men (66.67% versus 20.41%; p<0.001).

Glucose tolerance

Of the 103 subjects of the study, 55 (53.40%) had normal glucose tolerance (NGT). Isolated impaired fasting glucose (IFG), isolated impaired glucose tolerance (IGT) and combined glucose intolerance (IFG and IGT) were observed in 2 (1.94%), 37 (35.92%) and 9 subjects (8.74%) respectively.

Table 1: Subjects characteristics

Variables	Means ± SEM (n=103)
Sex (M/F)	54/49
Age (years)	30.25±0.76
Body weight (kg)	71.43±1.47
BMI (kg/m2)	25.89±0.52
Waist circumference (cm)	86.06±1.18
Hip circumference (cm)	98.59±1.12
Ratio waist/hip	0.87±0.01
Fasting glucose (mmol/l)	5.43±0.07
Fasting insulin (µU/ml)	29.57±1.86

BMI: Body mass index

Table 2: Characteristics of normal glucose tolerance subjects and of subjects with impaired fasting glucose and or impaired glucose tolerance

	NGT (n=55)	IFG/IGT (n=48)
Sex (M/F)	26/29	23/25
Age (years)	30.38±1.09	30.1±1.09
Body weight (kg)	70.44±1.88	72.57±2.34
BMI (kg/m^2)	25.37±0.65	26.49±0.83
WC (cm)	84.93±1.42	87.4±1.96
HC (cm)	97.33±1.45	100.04±1.75
Ratio waist/hip	0.88±0.01	0.87±0.01
FBG (mmol/l)	5.21±0.09	5.68±0.09*
Fasting Insulin (µU/ml)	29.42±2.81	29.81±2.3
HOMA-IR	6.87±0.72	7.47±0.57
MATSUDA-ISI	1.95±0.13	1.47±0.1*
Insulinogenic Index	50.3±5.31	42.5±4.42

NGT: Normal glucose tolerance; IFG/IGT: Impaired fasting glucose and or impaired glucose tolerance; BMI: Body Mass Index; WC: Waist Circumference; HC: Hip Circumference; FBG: Fasting Blood Glucose. Values are means ± SEM. *Significantly different from NGT group value, p<0.01

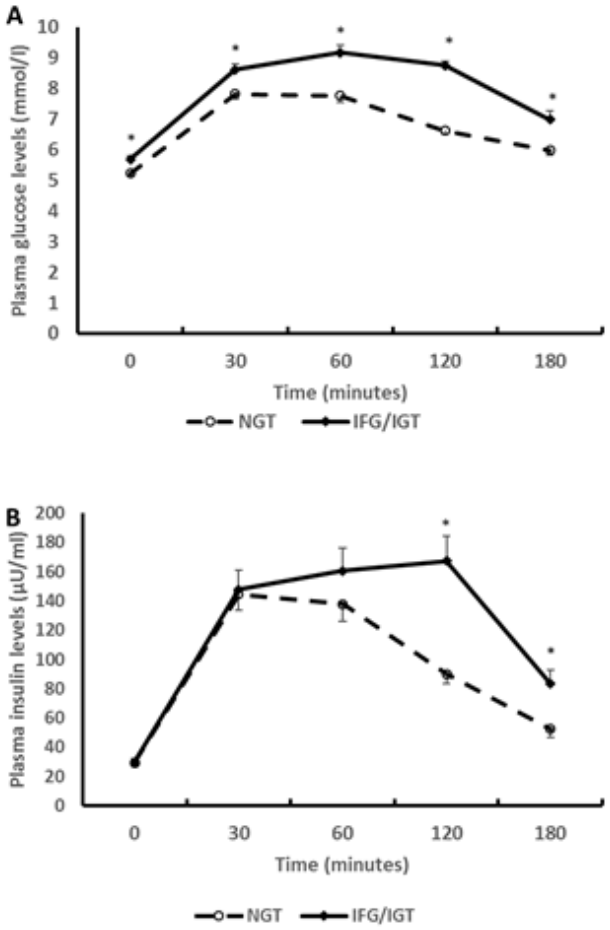

Figure 1: Plasma glucose (A) and insulin levels (B) during oral glucose tolerance test (OGTT). Values are means ± SEM; n = 49 in NGT group and 37 in IFG/IGT group
*significantly different from NGT value, p<0.05

Abnormal glucose tolerance, either IFG and or IGT (IFG/IGT), was thus observed in 48 subjects (46.60%). Table 2 presents the characteristics of NGT subjects and of IFG/IGT subjects. As shown by table 2, there were no significant differences in body weight, BMI, waist circumference and fasting blood insulin levels between NGT subjects and IFG/IGT subjects. However, in both men and women, IFG/IGT prevalence was higher in obese subjects (66.67%) than in people with normal BMI (41.15%). In addition, in men, IGT was observed in 3 of the 4 underweighted subjects.

There was no significant effect of age on glucose tolerance prevalence. In young subjects (20-24 years), a high prevalence of IFG/ITG (46.42%) was observed.

Plasma insulin levels

Fasting plasma insulin levels and insulin levels during OGTT have been measured in 88 subjects. Fasting hyperinsulinemia has been observed in 81.81% of the subjects.

Proportion of subjects with fasting hyperinsulinemia increased with BMI as it was 33.3% in underweighted subjects, 73% in subjects with normal BMI, 85.7% in overweighed subjects and 95.5% in obese people.

Plasma glucose and insulin profile during OGTT

Evolution of plasma glucose and insulin levels during OGTT is presented in figure 1. In subjects with normal glucose tolerance, the peak of glucose level during OGTT was observed 30 minutes after the beginning of the test while this peak occurred at 60 minutes in IFG/IGT subjects. At all points of the OGTT, plasma glucose levels were higher in IFG/IGT subjects than in NGT subjects.

As for plasma insulin profile, the peak of insulin levels, like that of glucose levels, appeared early at 30 minutes in NGT subjects but this peak occurred latter at 120 minutes of the OGTT in IGT subjects. In IFG/IGT subjects, plasma insulin levels, at 30 minutes, were not significantly different from those in NGT subjects.

Insulin sensitivity and resistance indexes

HOMA-IR

Mean value of HOMA-IR in NGT subjects was 6.87 ± 0.72 in NGT subjects and 7.47 ± 0.57 in IFG/IGT subjects but there was no significant difference between these values. HOMA-IR value was significantly higher ($p< 0.01$) in obese subjects (9.74 ±1.53) than in subjects with normal BMI (6.01 ± 0.28).

Matsuda insulin sensitivity index

Mean value of Matsuda-ISI was significantly lower in IFG/IGT subjects than in NGT subjects (1.47± 0.1 versus 1.95 ± 0.13, $p<0.01$). This value was also significantly lower in obese than in subjects with normal BMI (1.33 ± 0.12 versus 1.93 ± 0.13, $p<0.01$)

Insulinogenic index

Insulinogenic index was 50.3 ± 5.31 $\mu U.ml^{-1}/mM.l^{-1}$ in NGT subjects and 42.5 ± 4.42 $\mu U.ml^{-1}/mM.l^{-1}$ in IFG/IGT subjects, values which were not significantly different.

DISCUSSION

In the present work, glucose intolerance is observed in a high proportion of non-diabetic subjects. The 46% prevalence of IFG/IGT found in our study is far higher than all those reported in other sub Saharan African countries (Christensen, 2009; Evaristo-Netto 2010; Peer, 2012). It is a worrying situation especially since a high prevalence of prediabetes (46.6%) was already observed in young people aged 20 to 24 years. Without preventive measure, a progression to diabetes could occur in a high proportion of these subjects with IFG/IGT. Indeed, it has been estimated that up to 70% of subjects with prediabetes eventually develop diabetes (Nathan et al. 2007). In addition, incidence rate of diabetes in subjects with prediabetes has been estimated to 6.1%, 7% and 14% for respectively subjects with IGT, IFG and combined IGT and IFG (Gerstein et al. 2007). In Mauritius population, over 11 years (from 1987 to 1998), 38% of subjects with impaired fasting glucose and 46% of those with impaired glucose tolerance developed diabetes (Söderberg et al, 2004).

Alteration of tissue insulin sensitivity and deterioration of pancreatic beta cell function are the two main factors involved in the development of abnormal glucose tolerance. Insulin resistance and sensitivity were assessed by HOMA-IR and MATSUDA-ISI. The high mean value of HOMA-IR observed suggest a high degree of insulin resistance in subjects included in the study. These data are consistent with those previously published showing a greater insulin resistance in black African and in African Americans when compared to subjects of other ethnicities, especially Caucasians (Haffner et al. 1996, Rasouli et al. 2007, Goedecke et al. 2009). Reduction of hepatic insulin sensitivity could therefore be one of the factors involved in the development of abnormal glucose tolerance in our study as HOMA-IR, based on fasting parameters, explores especially hepatic insulin resistance. However, the mean value of HOMA-IR in IFG/IGT subjects was not significantly different from that observed in NGT subjects suggesting that decreased hepatic insulin sensitivity alone is not sufficient to explain development of glucose intolerance.

In contrast to HOMA-IR, MATSUDA insulin sensitivity index was significantly lower in IFG/IGT subjects than in NGT subjects indicating that IFG/IGT subjects had a lower sensitivity to insulin compared to subjects with normal glucose tolerance. As MATSUDA explores both hepatic and muscle insulin sensitivity, and because the mean HOMA-IR value was not significantly different in IFG/IGT and in NGT subjects, the difference in insulin sensitivity between IFG/IGT and NGT subjects could be related to a decreased muscle insulin sensitivity in IGF/IGT subjects. Moreover, it has been reported that the predominant site of insulin resistance in IGT subjects was muscle and that in IFG subjects was liver (Abdul-Ghani et al. 2006). In our study, impaired glucose tolerance was present in 95.83% of subjects with glucose tolerance disorders. These data suggest that a marked muscle insulin resistance is probably present in these subjects.

MATSUDA-ISI also appears as an index which allows a better assessment of insulin sensitivity in black African subjects than HOMA-IR, which is in line with previously published studies.

Indeed, it has been shown that in sub Saharan African, fasting insulin sensitivity indices are modest predictors of insulin sensitivity measured by hyperinsulinemic-euglycemic clamp (Sobngwi Et al., 2014). In addition, it has also been reported that Matsuda-ISI, in contrast to HOMA-IR, was significantly correlated with insulin sensitivity measured by hyperinsulinemic-euglycemic clamp in African American (Pisprasert et al., 2013).

In addition to insulin sensitivity, pancreatic beta cell function is also important for glucose homeostasis. In our study, in response to the development of insulin resistance, fasting hyperinsulinemia was observed in 82% of subjects and it allowed to maintain normal fasting blood glucose in most of them. This is in agreement with previous published data which reported that hyperinsulinemia and appropriated increased insulin secretion were associated with insulin resistance in African American and in black African (Haffner et al. 1996; Rasouli et al. 2007, Goedecke et al. 2009). Study of the early response of beta cell to a glucose load was carried out by the determination of the insulinogenic index during OGTT. The mean value of this index in IFG/IGT subjects was not significantly different from that of NGT subjects. These data suggest that the early response of beta cells was not altered in IFG/IGT subjects. Analysis of the curves of blood glucose and insulin levels during OGTT suggests that beta cell function was also maintained during late stages of the OGTT test. Indeed, after the first 30 minutes, as blood glucose levels remained high in subjects with IFG/IGT, blood insulin levels continued to rise along with blood glucose levels up to 120 minutes before dropping following lower blood glucose levels at 180 minutes.

These data suggest that the abnormal glucose tolerance observed in the subjects of our study, seems to be more related to a marked insulin resistance, especially at muscle level, than to deterioration of beta cells function.

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