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Full Length Research Paper

Prevalence of insulin resistance in obese Cameroonian women

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Several studies indicate that obesity is closely related to insulin resistance (IR). However, this relationship has not been adequately explored among Africans. This study aims to evaluate the prevalence of insulin resistance among obese Cameroonian women using some indirect methods for assessment of insulin resistance. We also analysed the correlation between some indices of IR with Triglyceride to HDL cholesterol ratio. We examined 230 obese and overweight women. Anthropometric measurements were done for all individuals. Blood lipids parameters, glucose and insulin were assayed after a 10 h fast. The indices McA, HOMA, QUICKI, ISI, FI and FIGR were used to assess insulin resistance. Receiver Operating Characteristic (ROC) curve analysis indicated that HOMA and QUICKI had high sensitivity and specificity for measuring IR compared to other indices. The overall prevalence of IR was 53.9% by HOMA-IR and 55.7% by QUICKI. TG/HDL-C method detected lower number of patients with IR in our study group and was not related to the homeostatic indexes of insulin resistance HOMA and QUICKI. This study indicates that insulin resistance is present in about half proportions of overweight and obese Cameroonian women.

Key words: Insulin resistance, obesity, body mass index, HOMA, QUICKI.

INTRODUCTION

The prevalence of overweight and obesity is rapidly increasing in developing as well as industrialised countries (WHO, 2006). Some studies from urban populations in Cameroon have shown that overweight and obesity are increasingly common (Sobngwi et al., 2002; Pasquet et al., 2003). For example, the estimated prevalence of obesity, based on body mass index (BMI), was 17.1% in women and 5.4% in men in urban Cameroon in 2002 (Sobngwi et al., 2002). Several studies have shown in Africa and particularly in Cameroon that obesity is associated with chronic diseases including diabetes, hypertension and metabolic syndrome which are the accompanying metabolic abnormalities of insulin resistance (IR) (Sobngwi et al., 2002).

Insulin resistance is a state in which a given amount of insulin produces a subnormal biological response (Kahn, 1978). Particularly, it is characterized by a decrease in the ability of insulin to stimulate the use of glucose by muscles and adipose tissue and to suppress hepatic glucose production and output (Matthaei et al., 2000). Furthermore, it accounts for a resistance to insulin action on protein and lipid metabolism as well as on vascular endothelial function and gene expression (Miâdi-Messao et al., 2009). Adipose tissue seems to play a key role in the pathogenesis of insulin resistance through several released metabolites, hormones and adipocytokins that can affect different steps in insulin action (Matsuzawa, 2005). Adipocytes produce non-esterified fatty acids, which inhibit carbohydrate metabolism via substrate competition and impaired intracellular insulin signalling (Matsuzawa, 2005; Randle, 1998).

The euglycemic insulin clamp which was introduced by (DeFronzo et al, 1979) and the intravenous glucose tolerance tests (Bergman et al., 1987) are gold standard methods for measurement of insulin resistance in research, but they are cumbersome in clinical practice and are difficult to perform in population-based research studies. In addition to these standard methods, there are

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various indirect methods for the assessment of IR. The Homeostasis Model Assessments (HOMA) (Matthews et al., 1985) and the Quantitative Insulin Sensitivity Check Index (QUICKI) (Katz et al. 2000) indices are calculated using both the fasting insulin (FI) and fasting blood glucose levels while the McAuley index (McA) and the Insulin Sensitivity Index (ISI) (McAuley, 2001) are calculated using fasting insulin and fasting triglyceride level. Other markers such as fasting insulinemia (FI) (Laakso, 1993) and fasting insulin to glucose ratio (FIGR) (Legro et al., 1998) are also accurate at predicting IR. Recently, the triglyceride to high-density lipoprotein cholesterol (TG/HDL-C) ratio has been reported to be closely related to insulin resistance among nondiabetic individuals (McLaughlin et al., 2005).

The aim of this study was to determine the prevalence and some determinants of the insulin resistance among overweight and obese women living in Yaounde, (the capital city of Cameroon). We have also studied the correlation between the insulin resistance indices like HOMA-IR, QUICKI and FI with TG/HDL-C in predicting insulin resistance in the study population.

MATERIALS AND METHODS

Study population

The study was carried out on 230 women recruited from the city of Yaounde, the capital of Cameroon and surrounding metro region through radio and print media advertisement. Data were collected through exploration of questionnaires. Inclusion criteria for study participation included: (1) women aged 18 to 65 years, diagnosed as having simple obesity through physical examination, with no concomitant diseases and without any pharmacological treatment; (2) stable body weight (± 2 kg) for at least three months prior to study randomization without use of medication known or suspected to affect body weight or appetite; (3) BMI from 25 to 29.9 kg/m^2 for overweight and BMI greater to 30 kg/m^2 for obese women; (4) no weight loss attempts through dietary intervention over the three months prior to trial randomization; (5) non diabetics (6) non-smoker (7) no pregnancy and (8) ability to competently understand and sign the consent form. Excluded were patients with known endocrine particularly hypothyroidism, liver and kidney diseases. Based on the above criteria, 230 consenting volunteers were selected to participate in the study. The study protocol was approved by the Review Committee of the Faculty of Medicine and Biomedical Sciences, University of Yaounde1, Cameroon.

Anthropometric measurements

Height was measured with a locally manufactured wall mounted stadiometer, which was calibrated against the Cameroon's Department of National Security identification scale. Body weight and percent body fat, were assessed using a Tanita™ BC-418 Segmental Body Composition Analyzer/Scale that uses bio-electrical impedance analysis for body composition analysis. Body mass index (BMI) was calculated using the weight and height measurements. Waist circumference measurements to the nearest 0.1 cm were taken at the mid-point between the bottom rib and the hip bone, without restrictive garments using a flexible non expandable tape measure. Waist circumference greater than 88 cm in women defined central obesity (NCEP, 2001).

Laboratory data

At baseline, in the morning after a 10-hour overnight fast, venous blood was sampled for the measurement of the plasma concentration of glucose, total and HDL cholesterol, triglycerides and insulin. Plasma glucose was measured by a glucose-oxidase method. Plasma total cholesterol, HDL cholesterol, and triglycerides were assessed with standard enzymatic spectrophotometric techniques. Plasma LDL cholesterol was calculated using Friedewald's equation, except when triglycerides exceeded 400 mg/dL (Friedwald et al., 1972). Plasma insulin was determined in duplicate using an enzyme-linked immunosorbent assay (ELISA) (Diagnostic Systems Laboratory, Webster, TX. USA).The sensitivity of the assay was 1.5 µU/ml and the variation coefficient inter-assay and intra-assay were 6.29 and 7.67 % respectively.

Data analysis

Six indirect methods were used for the assessment of insulin resistance (homeostasis model assessment (HOMA), quantitative insulin sensitivity check index (QUICKI), MacAuley (McA), fasting insulin/glucose ratio (FIGR), FI (fasting insulinemia) and ISI (insulin sensitivity index). These were calculated using the following equations: HOMA = insulin (mU/m) x [glucose (mmol/L)/22.5]; QUICKI = 1/(log insulin+ log glycemia in mg/dL); McAuley (McA) = exp [2.63 – 0.28 ln (insulin in mU/L) – 0.31 ln (triglycerides in mmol/L)]; Insulin sensitivity index (ISI) = exp [3.29 - 0.25 ln(insulin) - 0.22 ln (BMI) - 0.28 ln (TAG)]. According to different indices, the following thresholds defined insulin resistance state among non-diabetic participants: McA 5.8, HOMA 2.6 and QUICKI 0.33 (DeFronzo, 1999; McAuley et al., 2001). FI level 12 mU/L was considered as insulin resistant in non diabetic populations (DeFronzo, 1999). An ISI 6.3 M.mU/L as well as FIGR of 0.2 (McAuley et al., 2001) . The sensitivity and specificity of insulin resistance indices were estimated by analysis of receiver operating characteristic curve (ROC curve) . All these indices were compared with fasting insulin to evaluate the sensitivity and specificity in predicting insulin resistance.

Statistical analysis was done using statistical package for social sciences (SPSS) Windows version 12.0. Descriptive analysis included the estimation of mean values and standard deviations for continuous variables. Frequencies are expressed in terms of percentage. Categorical variables were compared by the Chi square. The p- values < 0.05 were considered to be statistically significant. The specificity and sensitivity of different indices was assessed using ROC curve. A ROC curve is a plot of the sensitivity (true positive) vs 1−specificity (false positive) for each potential marker tested (Rosner, 2000). The best markers have ROC curves that are shifted to the left with areas under the curve near unity. Non diagnostic markers are represented by diagonals with areas under the ROC curves near 0.5 (McLaughlin et al., 2003; Rosner, 2000).

RESULTS

The subjects' general characteristics of the study population

In the study population, 26% of women were overweight and 74% obese aged 18 - 65 years old. Obese women were of five years older than overweight and their

Table 1. Clinical and metabolic descriptors of overweight women of the study population (N = 60).

Variables	Mean ± SD	Minima	Maximal
Age (years)	35.77 ± 12.18	18	65
Weight (kg)	75.33 ± 7.09	63.20	93.40
BMI (kg/m²)	27.95 ± 1.44	25.1	29.90
Body fat percentage	35.07 ± 2.28	29.8	39.2
Waist (cm)	88.52 ± 8.45	74.00	107.00
Hip (cm)	108.52 ± 9.13	93.00	127.00
WHR	0.81 ± 0.064	0.68	1.00
Triglycerides (mg/dL)	56.28 ± 38.47	22.40	199.10
Total cholesterol (mg/dL)	180.37 ± 49.81	93.10	282.50
HDL-cholesterol (mg/dL)	42.31 ± 21.88	10.8	103.0
LDL-cholesterol (mg/dL)	127.40 ± 51.67	32.20	250.26
Glycemia (mg/dl)	99.16 ± 3.73	77.00	127.00
Insulin (µU/ml)	15.35 ± 0.85	3.27	57.32
HOMAIR	3.68 ± 0.31	0.65	13.44
QUICKI	0.33 ± 0.00	0.27	0.41
MACAULEY	15.36 ± 2.13	3.30	30.58
ISI	8.54 ± 0.55	4.27	12.59
FGIR	0.16 ± 0.00	0.03	0.60
TG/HDL	1.62 ± 0.18	0.40	6.11

anthropometric measurements were higher than in overweight. The mean fasting blood glucose was normal in both overweight and obese participants. The average values of most of the parameters were relatively closed to the threshold of detection of insulin resistance both overweight (Table 1) and obese women (Table 2).

Prevalence of insulin resistance

Almost half proportions of overweight (Figure 1) and obese women (Figure 2) were insulin-resistant considering HOMA and QUICKI. The percentage of IR with FIGR was higher compared to other indices of IR. Furthermore, lower frequency of IR was found when McAuley and TG/HDL were used.

Estimation of best indices of insulin resistance assessment

The ROC curve presented the estimation of best indices of insulin resistance assessment in study population (Figure 3). This figure shows the highest specificity (97.6%) and sensitivity (92.6%) of HOMA and QUICKI to predict insulin resistance when compared with McAuley index, ISI index, fasting insulin/glycemia ratio in this study. McAuley index, ISI index, fasting insulin/glycemia ratio have weak specificity and sensitivity.

Determinants of insulin resistance among obese subjects

The prevalence of insulin resistance in obese with abdominal fat accumulation was higher ($p < 0.05$) compared to obese with global fat (52.6 vs 28.6 %; respectively) (Table 3). It was also observed that the prevalence of IR was significantly higher among 40 – 49 years old women than other age groups (Table 4). Positive correlation was found between QUICKI and weight, BMI as well as with body fatness. It was similar for fasting insulin. However, HOMA wasn't correlated with body fatness. TG/HDL-C ratio was weakly and not significantly associated with HOMA, QUICKI and fasting insulin (Table 5).

DISCUSSION

In this study, we assessed the prevalence and some determinants of insulin resistance among overweight and obese women using indirect methods of assessment of insulin resistance because of the euglycemic insulin clamp which is considered as a gold standard test, is difficult, invasive and impractical for use in large patient groups or epidemiological studies (Mastsude and Defronzo, 1997; Masonori et al., 1999; Matthews et al., 1985).

Results show that half of women (BMI > 25 kg/m^2) have

Table 2. Clinical and metabolic descriptors of obese women of the study population (N=170).

Variables	Mean ± SD	Minima	Maximal
Age (years)	40.85 ± 9.62	18	65
Weight (kg)	96.45 ± 14.00	68.20	134.80
BMI (kg/m²)	36.44 ± 4.74	30.0	48.7
Body fat percentage	44.49 ± 5.48	34.2	60.0
Waist (cm)	106.73 ± 13.76	83.00	180.00
Hip (cm)	124.16 ± 10.99	96.00	155.00
WHR	0.86 ± 0.11	0.65	1.44
Triglycerides (mg/dL)	71.62 ± 29.90	25.00	272.00
Total cholesterol (mg/dL)	192.00 ± 69.24	79.00	400.00
HDL-cholesterol (mg/dL)	39.24 ± 19.70	8.0	110.0
LDL-cholesterol (mg/dL)	140.26 ± 15.63	21.20	379.16
Glycemia (mg/dl)	97.86 ± 6.96	72.00	130.00
Insulin (µU/ml)	13.62 ± 0.24	3.90	66.48
HOMAIR	3.51 ± 0.18	0.74	23.47
QUICKI	0.33 ± 0.03	0.25	0.40
MACAULEY	11.75 ± 2.57	1.79	23.70
ISI	7.50 ± 1.51	3.71	11.01
FGIR	0.14 ± 0.07	0.04	0.46
TG/HDL	2.39 ± 0.17	0.36	16.00

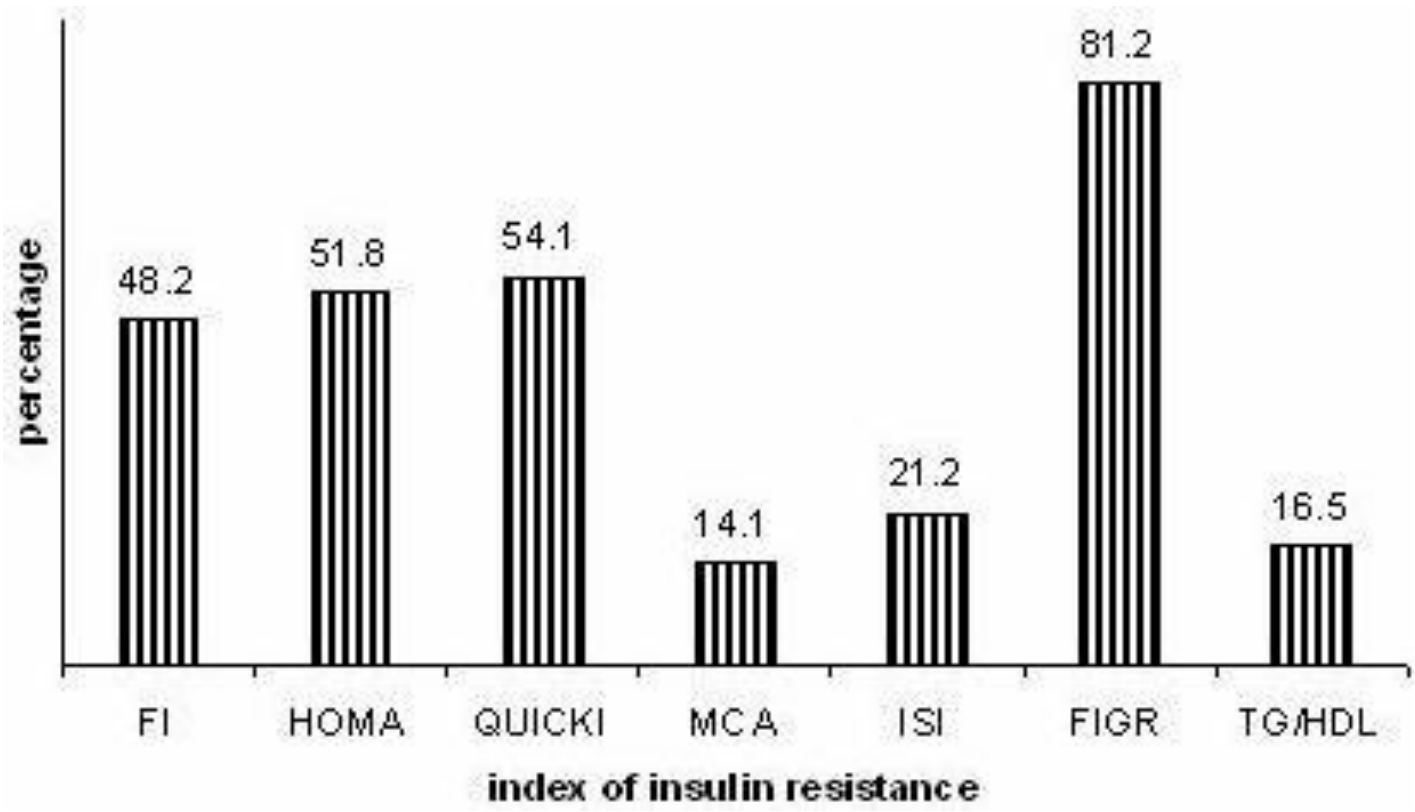

Figure 1. Insulin resistance among overweight women by indirect methods.
Bars represent proportions of insulin resistant obese patients according to the method used (n = 170). Homeostasis model assessment (HOMA), Quantitative insulin sensitivity check index (QUICKI), MacAuley (McA), Fasting insulin/glucose ratio (FIGR), Fasting insulinemia (FI), Insulin sensitivity index (ISI) and Triglyceride/HDL cholesterol ratio (TG/HDL).

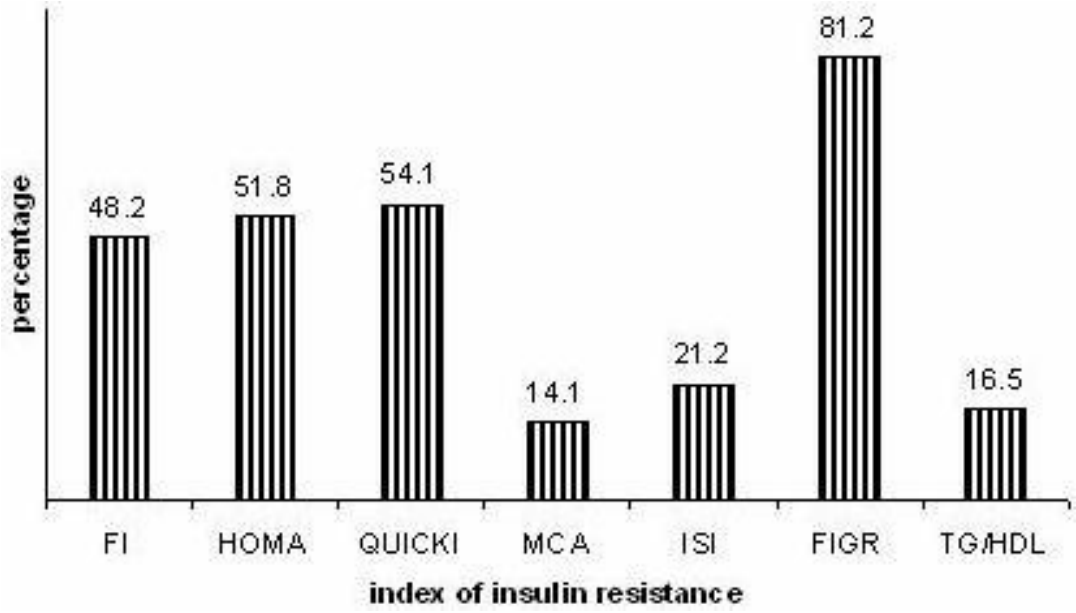

Figure 2. Insulin resistance among obese women by indirect methods. Bars represent proportions of insulin resistant obese patients according to the method used (n = 170). Homeostasis model assessment (HOMA), Quantitative insulin sensitivity check index (QUICKI), MacAuley (McA), Fasting insulin/glucose ratio (FIGR), Fasting insulinemia (FI), Insulin sensitivity index (ISI) and Triglyceride/HDL cholesterol ratio (TG/HDL).

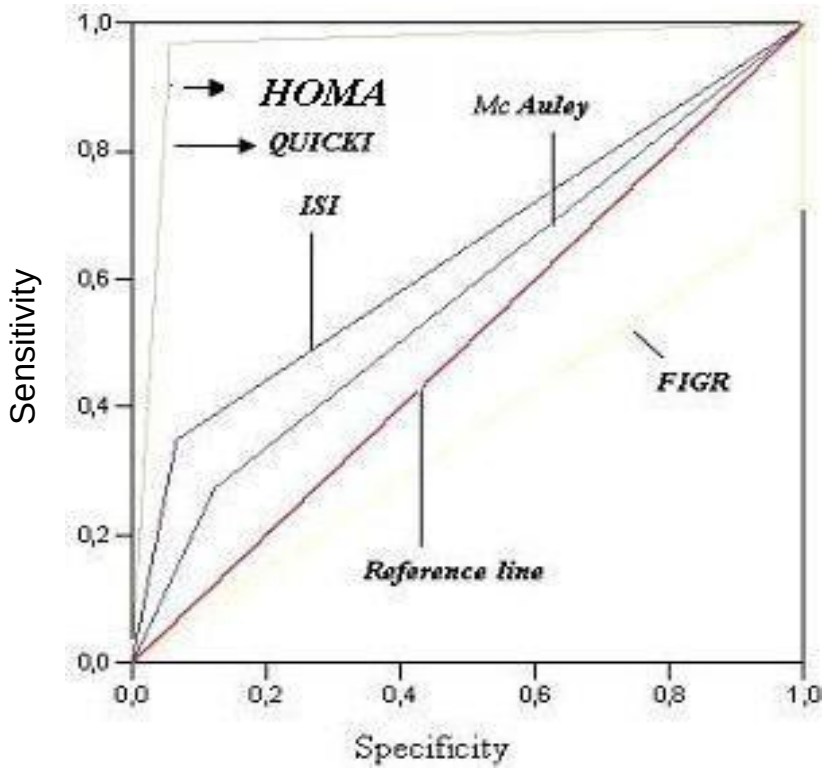

Figure 3. ROC curves for indexes of insulin resistance. Homeostasis model assessment (HOMA), Quantitative insulin sensitivity check index (QUICKI), MacAuley (McA), Fasting insulin/glucose ratio (FIGR), Fasting insulinemia (FI) and Insulin sensitivity index (ISI).

Table 3. Percentage of insulin resistance of obese women according to type of obesity.

	Global obesity (%) (n = 14)	Central obesity (%) (n = 156)
IR using HOMA	4 (28.6)	82 (52.6)[a]
IRusing QUICKI	4 (28.6)	86 (55.1)[a]

a, p<0.01 comparatively to global obesity group.

Table 4. Percentage of insulin resistance of obese women with central obesity following age.

Age groups	IR using HOMA (%)	IR using QUICKI (%)
18 - 29 (n = 22)	10 (45.5)	10 (45.5)
30 - 39 (n = 46)	24 (52.2)	24 (52.2)
40 - 49 (n = 58)	34 (58.6)[b. c]	34 (58.6)[b. c]
50 + (n = 30)	14 (46.7)	14 (46.7)
Total (n = 156)	82 (52.6)	86 (55.1)

b, p < 0.01 comparatively to age group (18 - 29); c, p < 0.01 comparatively to others age group.

insulin resistance using the homeostatic indices HOMA and QUICKI in urbanising Cameroonian population. Several researchers have shown the strong correlation between these indices and the method of reference clamp. The accuracy and precision of HOMA-IR and QUICKI as measures of IR and insulin sensitivity have been determined elsewhere by comparison with euglycemic and hyperglycemic clamps and the intravenous glucose tolerance test (Matthews et al., 1985; Katz et al., 2000) . Among overweight, the prevalence of insulin resistance was estimated to be 60% using HOMA and QUICKI, while among obese, it was 51.8 and 54.1% when evaluated using HOMA and QUICKI respectively.

These proportions indicate presence of insulin resistance in the study population. This finding is consistent with previous studies. In fact, Arslanian et al. (1996) and Bacha et al. (2003) have shown that obesity is a major risk factor for insulin resistance. However, we found a strong heterogeneity of mean plasmatic insulin in patients with the same degree of obesity. Thus, some overweight and obese subjects presented extremely high insulin levels while others have normal values. This variability indicates the importance of factors other than obesity in the regulation of insulin resistance.

These results are remarkably important and consistent with those of Insulin Resistance and Atherosclerosis Study in black adults aged 40 - 69 years who demonstrated evidence of insulin resistance when compared with white adults at a similar body weight, independent of diabetes status (Karter et al., 1996; Haffner et al., 1996). Lovejoy et al. (1996) in a similar study showed that US black women are more insulin resistant despite a lower amount of visceral fat and Merwe et al. (1996) demonstrated *in vitro* a higher degree of insulin resistance in adipose tissue among black women compared to white. In addition, black women had a brisker *in vivo* adipose tissue lipolysis.

The difference in prevalence of insulin resistance observed could be due to the fact that insulin resistance is believed to have both genetic and environmental factors implicated in its aetiology (Matthaei et al., 2000). The genetic component seems to be polygenic in nature, and several genes have been suggested as potential candidates (Matthaei et al., 2000). However, several other factors can influence insulin sensitivity, such as ethnicity, prenatal factors, puberty, sedentary lifestyle and diet.

The high prevalence of IR among women mainly those aged 40 - 49 years as shown in this study can be explained by the role of central obesity, a high percentage of fat and the strong sedentary lifestyle of women in Cameroon. These high values suggest that the setting in of insulin resistance and its importance are linked to the difference in total adiposity and may also be influenced by hormones.

In this study, the prevalence of insulin resistant characterized by central obesity was higher to that observed in global obesity. This finding supports the idea of Stern and Haffner (1986) which reported that central obesity has been associated with hyperinsulinaemia and it has been suggested that the over production of insulin may act as an energy conserving mechanism under conditions of periodic famine and low energy intake. Excess intra-abdominal adiposity increases overall cardio metabolic risk partially through alterations in the secretion of a series of biologically active molecules (adipokines). These include increased secretion of free fatty acids, which can induce insulin resistance in muscle and ß-cell toxicity in the pancreas, inflammatory mediators, such as TNF , IL-6, resistin and PAI-1, and decreased secretion of the cardioprotective adipokine, leptin that contribute to insulin resistance and its complications (Ferroni et al.,

2004; Tataranni , 2005).

One of the objectives of this study was to assess the applicability of the lipid criteria (TG/HDL ratio) as measures of Insulin resistance index in Cameroon. Results obtained have shown that the diagnosis of IR using the TG/C-HDL ratio would be a mistake since we did not find a significant correlation between the TG / HDL ratio and the homeostatic indices HOMA and QUICKI. A similar result was found by Sumner et al. (2005) who demonstrated that the TG/HDL-C ratio is not associated to HOMA and QUICKI in African American. The same observation was made by Bovet et al. (2006) who noticed a stronger association between TG/HDL ratio and insulin resistance among Caucasians than African Americans (Bovet et al., 2006).This could be due to the fact that obese and overweight Cameroonians have a low rate of triglyceride. This is supported by the idea that insulin resistance of Africans is much more at the level of the muscles. Lipoprotein lipase activity has been found to be higher in blacks than in whites, and this may lead to a lower triglyceride concentration in blacks (Despres et al., 2000).

Conclusion

This study showed that homeostatic indexes of insulin resistance (HOMA-IR and QUICKI) can be used as simple and reliable indices of insulin resistance assessment among overweight and obese Cameroonian women. Using these indices, we noticed a high prevalence of IR in the studied population. Despite normal fasting plasma glucose levels and more favourable lipid profile, Cameroonians exhibited high prevalence of insulin resistance, even though the mean of plasmatic insulin is moderate.

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