Hindawi Publishing Corporation
Journal of Obesity
Volume 2012, Article ID 740854, 10 pages
doi:10.1155/2012/740854

Research Article

Four-Year Trends in Cardiometabolic Risk Factors according to Baseline Abdominal Obesity Status in West-African Adults: The Benin Study

Charles Sossa,[1,2] Hélène Delisle,[1] Victoire Agueh,[2] Michel Makoutodé,[3] and Benjamin Fayomi[4]

[1] *TRANSNUT, WHO Collaborating Centre on Nutrition Changes and Development, Department of Nutrition, Faculty of Medicine, University of Montreal, CP 6128 succursale centre-ville, Montréal, QC, Canada H3C 3J7*
[2] *Departement of Health promotion, Regional Institute of Public Health, 01 BP 918 Cotonou, Benin*
[3] *Departement of Health and Environment, Regional Institute for Public Health, 01 BP 918 Cotonou, Benin*
[4] *Institute of Applied Biomedical Sciences, 01 BP 862 Cotonou, Benin*

Correspondence should be addressed to Hélène Delisle, helene.delisle@umontreal.ca

Received 15 August 2011; Revised 22 October 2011; Accepted 26 October 2011

Academic Editor: Ivana Zavaroni

The study examined whether abdominal obesity (AO) according to waist circumference was associated with more unfavourable changes in other cardiometabolic risk (CMR) factors in sub-Saharan Africans. The study included 541 randomly selected and apparently healthy subjects (50% women) aged 25–60 years. Complete data at baseline, 24, and 48 months later was available in 366 subjects. AO was associated with higher CMR at baseline and over the follow-up period, except for high blood pressure. A significantly higher incidence of high ratio of total cholesterol : HDL-cholesterol (TC/HDL-C) was associated with AO. Controlling for WC changes, age, baseline diet, and lifestyles, the relative risk (RR) of low HDL-C and high TC/HDL-C was 3.2 (95% CI 1.06–9.61) and 7.4 (95% CI 2.01–25.79), respectively, in AO men; the RR was not significant in women. Over a four-year period, AO therefore appeared associated with an adverse evolution of cholesterolemia in the study population.

1. Introduction

Obesity is an independent risk factor for chronic diseases, such as diabetes, cardiovascular disease (CVD), and some cancers [1, 2], which are the leading causes of deaths worldwide [3]. According to the World Health Organization [4], 44% of diabetes, 23% of ischemic heart disease, and 7% to 41% of some cancers are attributable to overweight and obesity [3]. The rising incidence of chronic diseases associated with obesity does not spare developing countries (DCs) even low-income ones [5]. WHO estimates that if appropriate preventive measures are not taken, 20 million deaths by 2015 will be due to CVD and 80% of this mortality burden will occur in DCs [6]. Changes in lifestyle and diet that characterize the ongoing nutrition transition contribute to the outbreak of obesity in DCs [7].

Abdominal obesity (AO) is a more potent risk factor than gynoid or general obesity [8]. Several studies have shown that Africans and peoples of African descent have less visceral fat than whites, regardless of gender, age, total body fat, and BMI [9–12]. The prevalence of metabolic syndrome (MetS) is reportedly lower among African Americans than in Caucasians [13] while the prevalence of CVD and insulin resistance is higher [14, 15]. In a nested study of African descent Haitians living in Montreal and white subjects paired for age, sex, and BMI, Haitians had significantly less visceral fat than Whites for the same waist circumference (WC) [16]. In the large INTERHEART study in 52 countries, the association of myocardial infarction risk with abdominal obesity was higher in Europeans than in Blacks [17] due to less visceral fat in the latter than the former. The relationship between obesity and other cardiometabolic risk (CMR) factors in Africans is poorly documented. The prohibitive cost of treatment of obesity-related diseases, both for the patient and the health system, mainly in developing countries, requires

that increased attention be paid to CMR assessment. The challenge of addressing CMR factors is especially complex in sub-Saharan settings where epidemiologic data related to risk factors are not readily or reliably available at present. The purpose of our study was to assess changes in CMR factors in sub-Saharan Africans according to baseline obesity status and taking account of diet and lifestyle. We hypothesized that obesity as currently defined by standard anthropometric criteria is weakly associated with unfavourable changes of other CMR factors.

2. Methods and Materials

2.1. Subjects and Study Design. This prospective observational study was conducted among Benin (West Africa) adults who were initially in apparent good health. Subjects ($n = 541$) aged 25–60 years were randomly selected by multistage sampling and were enrolled in baseline study on CMR factors and nutrition transition. Subjects were selected in Cotonou, the largest urban city ($n = 200$), Ouidah, a small-size city located 45 km away from Cotonou ($n = 171$), and the rural area surrounding Ouidah ($n = 170$) using location data (urban) or after enumeration of compounds (semi-urban and rural). Eligible participants were Beninese-born subjects having lived in the study area for at least six months. Subjects with a prior diagnosis of hypertension, diabetes or cardiac condition were excluded, but those diagnosed for high blood pressure or diabetes during the baseline study or at followup remained in the cohort, whether or not they received medical treatment. Details of sample size determination and sampling process were published elsewhere [18, 19]. After the baseline study (T0), subjects were followed up after 24 months (T1) and 48 months (T2). A final sample size of 350 was deemed sufficient to achieve 80% statistical power with a significance level (alpha) of 0.05 using logistic regression on blood pressure as main dependent variable [20].

2.2. Data Collection Procedure. Data were collected between June 2005 and October 2010 by the same investigator team as the baseline study. Only physicians collected anthropometric data and measured blood pressure after standardization of techniques. A laboratory technician handled the blood samples. Before each biochemical data collection, participants were informed on the practical arrangements that were prior to blood sampling: (1) an overnight fast of at least 12 hours, (2) no alcohol for at least 48 hours, and (3) no intense physical activity on the previous day. We made sure that these conditions were met, otherwise another appointment was set. Subjects were organized into self-help groups by area of residence to facilitate followup and for active prevention support once the followup study would be over. Apart from contacts for formal followup data collection, self-help groups were encouraged to meet every three months for weight and blood pressure checks, and for standard general health advice. Anthropometric and blood pressure data that were collected during these meetings were not used in the followup study reported here.

2.3. Study Variables

2.3.1. Anthropometric Parameters. Anthropometric variables included body weight, height, and WC that were measured according to standard procedures. WC was used as index of abdominal obesity [21]. It was measured to the nearest 0.1 cm at the midpoint between the lower rib margin and the iliac crest while subjects were standing and breathing normally [21]. We used a flexible nonstretch measuring tape at baseline survey and at the first followup. A flexible nonstretch measuring tape (Gulick) equipped with an estimator of the applied tension during measurement was used in the last followup. Two measures of WC were taken for each subject and the mean of the two readings was used in the analyses. Generic cutoff values for AO (80 and 94 cm for women and men, resp.) were used, as recently recommended by several organizations for sub-Saharan Africans, in the absence of specific values [22]. Body weight was measured on subjects in fasting with light clothing and without shoes to the nearest 0.1 kg using a mechanical scale that had maximum capacity of 150 kg (SECA, Germany). Height was measured once at baseline to the nearest 0.1 cm with a commercial stadiometer (SECA, Germany). Body mass index (BMIs) cutoffs for underweight (<18.5), overweight (25–29.9), and obesity (≥30) were as defined by WHO [23].

2.3.2. Blood Pressure. Blood pressure was measured using a mercury sphygmomanometer. Systolic and diastolic blood pressure was measured on the right arm of seated subjects after a 10-minute rest. Two readings of systolic and diastolic blood pressure were taken. The interval of time between the first and the second reading was at least 20 minutes [24]. The mean of the two readings was used in the analyses. High blood pressure was defined as systolic blood pressure (SBP) ≥130 mmHg and or diastolic blood pressure (DBP) ≥85 mmHg [25].

2.3.3. Biochemical Parameters. Blood samples were collected in the morning after a 12-hour overnight fast and were centrifuged within two hours in the biochemistry laboratory of the Institute of Applied Biomedical Sciences in Cotonou. Using standard enzymatic laboratory methods, fasting glycemia, and serum concentrations of total cholesterol, HDL-cholesterol (HDL-C) and triglycerides were determined. The ratio of total cholesterol/HDL-C (TC/HDL-C) was computed. Abnormal values were defined as: elevated triglycerides (>1.70 mmol/L); high fasting glycemia (≥5.6 mmol/L); and low HDL-cholesterol (≤1.29 mmol/L in women and ≤1.03 mmol/L in men) [22]. Fasting insulin was assayed using ELISA method with IBL kits (IBL, Hamburg). Insulin resistance cutoff was the 75th centile of HOMA-IR (homeostasis model assessment, insulin resistance) in the whole sample of subjects [26]. The selected cutoffs for high TC/HDL-C were 4 for men and 5 for women [27].

2.3.4. Cardiometabolic Risk Factors. CMR factors considered in the study along with AO according to WC or high BMI (overweight/obesity) were high blood pressure, high fasting glycemia, insulin resistance (based on HOMA-IR), low

HDL-C, high TC/HDL-C, and high triglycerides. In counting CMR factors, high fasting glycemia or (newly diagnosed) diabetes, or insulin resistance were considered as one risk factor. Similarly, high blood pressure included medical treatment for hypertension. Dyslipidemia as one risk factor was defined as low HDL-C, high TC/HDL-C, or high triglyceride concentration.

2.3.5. Socioeconomic Variables. Socioeconomic data were collected in personal interviews. Education and Socioeconomic status (SES) were the main Socioeconomic factors considered. SES was assessed at baseline using a household amenity score as proxy of household income, much like in several demographic and health surveys in Africa. The items include type of latrine; paid domestic help; ownership of land, motorcycle, car, television, mobile phone, land line phone, and refrigerator; electricity, running water in the house; type of fuel used for cooking; wall and floor materials. The SES score was computed separately in each site and tertiles were used in analyses. Details of items and coding are published elsewhere [18, 19].

2.3.6. Diet Quality. Dietary intake was computed at baseline on the basis of two or three nonconsecutive 24-hour food recalls conducted over an average period of one month. Diet quality was appraised with several scores including a micronutrient adequacy score which was significantly associated with CMR in the cross-sectional study (unpublished data) and is therefore used in the present study. The micronutrient score was based on adequacy of intake of 14 micronutrients (vitamins A, B6, B12, C, and E, thiamine, riboflavin, niacin, pantothenic acid, folic acid, magnesium, calcium, iron, and zinc) according to WHO/FAO recommended dietary intakes for age and sex [4].

2.3.7. Lifestyle Variables. Subjects were questioned at baseline about their habitual drinking and smoking patterns. Alcohol consumption is expressed in the present study as mean quantity of pure alcohol per day. Regarding smoking, we distinguished three categories: smokers, ex-smokers, and nonsmokers. For alcohol and tobacco, the questionnaire was based on the STEPwise tool developed by the WHO [28].

Baseline physical activity was assessed through two or three nonconsecutive 24-hour recalls [18]. Participants were asked about all their previous day's activities, from the time they got up to the moment they went to bed. Time spent in bed, in various modes of transportation, for main and secondary occupations, for house chores, and for leisure activities was computed from the daily estimated schedule. The intensity level of each activity was estimated in metabolic equivalents (METS). Based on WHO guidelines for the prevention of chronic diseases [29], we classified subjects as active ($\geq$3 MET, $\geq$30 min/day) and inactive ($\geq$3 MET, <30 min or <3 MET, any duration).

2.4. Statistical Analyses. Data were analyzed using SPSS, version 16.0 (SPSS Inc., Chicago, IL). The differences in CMR factors at baseline and at last followup according to anthropometric status were assessed using chi^2 test. The incidence rate was computed as the number of new cases during the four years divided by the number of subjects without the risk factor at baseline. The relative (RR) risk of developing one or the other CMR factor during the course of the followup was assessed using multiple logistic regression models while controlling for baseline age, diet (micronutrient intake adequacy score), and lifestyle (alcohol consumption and physical activity), as well as WC change between T0 and T2. The level of statistical significance for all tests was $P = 0.05$.

2.5. Ethical Considerations. The study was approved by the Ethics Committee of the Faculty of Medicine (University of Montreal) and by the Ministry of Health in Benin. Written informed consent was obtained from each participant before enrolment. The participants were all informed individually of their blood pressure and the results of laboratory tests. Those with abnormal values were referred to a physician for diagnosis and treatment. The first medical consultation and prescription was paid by the research project.

3. Results

3.1. Study Subjects. Out of 622 eligible subjects, a total of 541 completed the study, giving a response rate of 86.9%. The participation rates were 76.7% (415 out of 541) in first followup and 76.9% (416 out of 541) in the second followup; 67.7% (366 out of 541) had complete followup data.

Baseline characteristics of subjects who attended both followups and those who missed one or the other (Table 1) were not significantly different except for age, anthropometric status (in men only), and place of residence. Average age was higher by roughly two years in men and women with complete data compared to those with incomplete data. A lower proportion of subjects were available for followup in the large city than other locations. Obesity-related parameters were significantly higher in men with complete followup than in those with incomplete data. Socioeconomic status, diet, and lifestyle were not significantly different in the two groups, however. Statistical analyses were performed on subjects with complete data ($n = 366$) (Figure 1).

3.2. Baseline Obesity Status and Other Cardiometabolic Risk Markers. At baseline, 39.3% of subjects with complete data were overweight or obese according to BMI. AO reached 33.6%. Obesity was much more frequent in women than in men. Indeed, one man for three women had AO. Similarly, one man for two women was obese or overweight based on BMI.

Table 2 gives the prevalence rate of CMR factors at baseline according to anthropometric status of subjects. Initial prevalence of CMR factors was generally higher in obese or overweight subjects compared to those with normal anthropometric status at onset of study, whether on the basis of WC or BMI. The prevalence of hypertriglyceridemia and fasting hyperglycemia was low and not significantly different in obese and nonobese subjects. In AO subjects, only 16.6% of women and 6.2% of men were free from other CMR factors. Similarly, only 19% of women and 13.6%

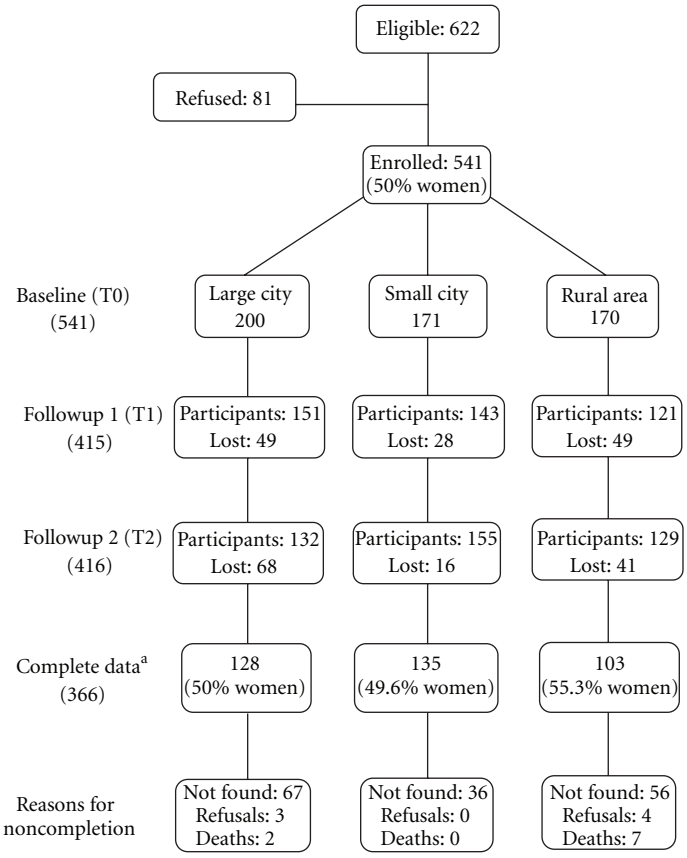
Table 1: Baseline characteristics of study subjects according to followup completion.

Characteristics	Complete data ($n = 366$)	Incomplete data ($n = 175$)	P value[a]
Age years (mean ± SD)			
Men	37.8 ± 10.2	36.0 ± 9.9	**0.041**
Women	39.5 ± 9.9	37.0 ± 10	**0.007**
Sex (%)			
Men	48.6	53.1	0.326
Women	51.4	46.9	
Location (%)			
Large city	35.0	41.1	
Small city	36.9	20.6	**0.001**
Rural area	28.1	38.3	
Socioeconomic level (%)			
Low	33.3	36.6	
Medium	35.6	37.1	0.498
High	31.1	26.3	
Education (%)			
No formal education	24.8	26.8	
Elementary school	34.9	33.7	0.880
High school and above	40.2	39.4	
Physical activity (%)			
Active (≥30 min/d moderate/vigorous activity)	83.1	80.6	
Inactive (<30 min/d moderate/vigorous activity)	16.9	19.4	0.276
Alcohol consumption (%)			
Binge drinking	8.2	8.0	
Regular high	6.8	10.9	
Regular moderate	35.5	33.7	0.462
None	49.5	47.7	
Smoking (%)			
Current smokers	4.4	4.0	0.539
Former smokers	7.7	5.1	
Nonsmokers	88.0	90.9	
Diet quality indicator (mean ± SD)			
Micronutrient adequacy score	10.4 ± 2.6	9.9 ± 2.7	0.706
Body mass index (mean ± SD)			
Men	22.8 ± 4.0	21.2 ± 3.1	**<0.001**
Women	25.8 ± 5.8	26.3 ± 6.6	0.588
Waist circumference cm (mean ± SD)			
Men	83.9 ± 11.0	79.0 ± 8.0	**0.001**
Women	87.5 ± 12.7	88.0 ± 14.4	0.993
Number of cardiometabolic risk factors[b]	1.0 ± 0.8	0.9 ± 0.9	0.706

[a] P value for unpaired t-test or χ^2 test as appropriate.
[b] Risk factors other than abdominal obesity or overall overweight/obesity.

of men who were overweight/obese (BMI >25) at baseline presented no additional CMR factor. In both men and women, the proportion of subjects without any of the CMR factors considered was significantly higher in the nonobese or overweight. The difference was particularly marked in men, with at least a threefold increase in the proportion of CMR-free subjects among the nonobese or overweight.

3.3. Four-Year Changes in Cardiometabolic Risk Factors. Table 3 shows that the prevalence of most CMR factors increased significantly over the four-year followup in both men and women, even hypertriglyceridemia which was low and remained so. Of note, subjects who went on medical treatment for high blood pressure ($n = 30$) are included in the prevalence figures, and those being treated for diabetes



[a]: missing subjects in at least one followup are considered as having incomplete data

Figure 1: Study subjects.

with insulin or oral hypoglycemiants ($n = 3$) are included in "high fasting glycemia" and "insulin resistance." It is reminded that at baseline, already diagnosed cases of hypertension or diabetes were excluded from the cohort. There was over the four-years a significant decrease of high blood pressure prevalence. The rate of general overweight/obesity tended to increase, and that of AO increased significantly, but only in women. In men, only an upward trend for overweight/obesity was observed. Figure 2 provides more detail on the evolution of BMI and WC status. In women, overall and abdominal obesity increased significantly. In men, overall obesity and AO rates remained low over the followup period; a nonsignificant upward trend was only observed for overweight, and not for overall obesity.

Compared to the nonobese, AO subjects exhibited a significantly higher incidence rate of high TC/HDL-C ($P = 0.002$), as shown in Table 4. The incidence rate of insulin resistance tended to be higher in AO subjects ($P = 0.051$). Subjects who had to go onto medical treatment for high blood pressure or diabetes are included in the incidence figures. At the end of the followup period, the total number of new cases was 81 for low HDL-C, 58 for high TC/HDL-C, 83 for insulin resistance, 59 for high fasting glycemia, and 11 for diabetes, while there were 19 new cases of high blood pressure, including the subjects under medical treatment (data not shown).

In Table 5, the relative risk (RR) of onset of CMR factors during the followup period according to obesity status is shown separately for men and women, before and after adjusting for WC changes, age, diet, and lifestyles. RR could not be computed for other CMR factors because of too small numbers. In women, the RR was not significantly higher in those with AO. In contrast, in men, the RR of onset of dyslipidemia was three- to four-fold in subjects exhibiting initial AO compared to the nonobese in the nonadjusted model. Controlling for WC changes and initial diet and lifestyle profile further increased the likelihood of dyslipidemia up to seven times.

4. Discussion

The study examined the evolution of cardiometabolic risk (CMR) factors according to baseline obesity status, while controlling for confounding factors, as well as for obesity change over the followup period. We had to combine overweight and overall obesity (BMI $\geq$ 25) because of the small number of men with overall obesity (BMI $\geq$ 30). Furthermore, results were examined primarily for AO using WC cutoffs as suggested by several organizations (in an attempt to harmonize the definition of MetS) in the absence of specific values for sub-Saharan Africans [22], since AO is considered to be associated with higher risk than general

Table 2: Baseline cardiometabolic risk factors according to anthropometric status ($n = 366$).

Risk factors (%)	All	Abdominal obesity			Overall overweight/obesity		
		Yes	No	P value	Yes	No	P value[a]
Women ($n = 188$)		($n = 91$)	($n = 97$)		($n = 100$)	($n = 88$)	
High blood pressure	36.7	46.2	27.8	**<0.001**	44.0	28.4	**0.026**
High fasting glycemia	10.0	13.2	7.2	0.132	9.1	11.8	0.426
Insulin resistance	33.0	42.9	23.7	**0.005**	41.0	21.6	**0.004**
Low HDL-C	31.4	37.4	28.5	0.060	41.0	20.5	**0.002**
High TC/HDL-C	19.1	25.3	13.4	**0.030**	29.0	8.0	**<0.001**
High TG	1.1	1.1	1.0	0.732	1.0	1.1	0.718
No CMR factor[b]	28.2	16.6	39.2	**<0.001**	19	38.6	**<0.001**
Men ($n = 178$)		($n = 32$)	($n = 146$)		($n = 44$)	($n = 134$)	
High blood pressure	35.1	65.6	28.8	**<0.001**	52.3	29.9	**0.006**
High fasting glycemia	7.9	12.5	6.8	0.228	6.7	11.4	0.243
Insulin resistance	19.1	34.4	15.8	**0.015**	36.4	13.4	**<0.001**
Low HDL-C	21.1	37.5	18.5	**0.020**	38.6	16.4	**0.003**
High TC/HDL-C	10.1	34.4	4.8	**<0.001**	29.5	3.7	**<0.001**
High TG	2.8	6.2	2.1	0.220	6.8	1.5	0.098
No CMR factor[b]	37.1	6.2	43.8	**<0.001**	13.6	44.8	**<0.001**

[a] P value for χ^2 test.
[b] Cardiometabolic risk factors (CMR) other than abdominal or overall overweight/obesity.

Table 3: Evolution of prevalence of cardiometabolic risk factors during the four-year followup period ($n = 366$).

Risk factors (%)	Women ($n = 188$)			Men ($n = 178$)		
	Baseline	T2 (4 years)	P value[a]	Baseline	T2 (4 years)	P value[a]
High blood pressure[b]	36.7	25.0	**0.014**	35.1	18.0	**<0.001**
High fasting glycemia[c]	10.1	17.0	**0.050**	7.9	23.6	**<0.001**
Insulin resistance[c]	33.0	38.2	0.281	19.1	29.2	**0.025**
Low HDL-C	31.4	47.3	**<0.001**	21.1	30.3	**<0.001**
High TC/HDL-C	19.1	29.1	**0.022**	10.1	18.0	**0.032**
High triglycerides	1.1	5.9	**0.011**	2.8	12.2	**<0.001**
Overweight/obesity	53.2	62.8	0.060	24.7	33.1	0.079
Abdominal obesity	48.4	68.1	**<0.001**	18.0	20.2	0.589

[a] P values for χ^2 test.
[b] Including 30 subjects under treatment for high blood pressure.
[c] Subjects under treatment for diabetes are included ($n = 3$).

Table 4: Four-year incidence[a] rate of cardiometabolic risk factors according to baseline abdominal obesity status ($n = 366$).

Risk factors	Overall incidence (%)	Specific incidence		
		Obese (%)	Non obese (%)	P value[b]
High BP[c]	8.1	13.3	6.3	0.149
High fasting glycemia[d]	17.7	22.4	15.5	0.121
Diabetes[d]	3.0	4.9	2.1	0.236
Insulin resistance[d]	30.7	39.7	27.4	0.051
Low HDL-C	30.2	36.4	27.7	0.164
High TC/HDL-C	18.6	29.2	14.3	**0.002**
High triglyceride	8.4	10.8	7.1	0.229

[a] Number of new cases divided by the total number of subjects without the risk factor at baseline.
[b] P-values for χ^2 test.
[c] Includes new cases under treatment for hypertension ($n = 7$).
[d] Includes new cases under treatment for diabetes ($n = 2$).

Table 5: Onset of cardiometabolic risk factors over the followup period before and after adjusting for WC changes, age, diet, and lifestyle ($n = 366$).

	Not adjusted				Adjusted[a]			
	Women		Men		Women		Men	
	RR	CI 95%	RR	CI 95%	RR	CI 95%	RR	CI 95%
High fasting glycemia	1.90	0.79–4.75	2.35	0.97–5.68	1.90	0.79–4.75	1.33	0.48–3.72
Insulin resistance	1.38	0.65–2.91	1.76	0.58–5.30	1.40	0.62–3.20	2.22	0.85–5.78
Low HDL-C	0.79	0.40–1.62	**3.01**	**1.12–8.23**	0.47	0.19–1.10	**3.18**	**1.06–9.61**
High TC/HDL-C	1.57	0.71–3.29	**4.70**	**1.70–13.1**	1.61	0.66–3.93	**7.45**	**2.01–25.79**

[a]Adjusted for WC (waist circumference) changes between T0 and T2 and baseline age, physical activity, alcohol intake, and dietary micronutrient adequacy score.

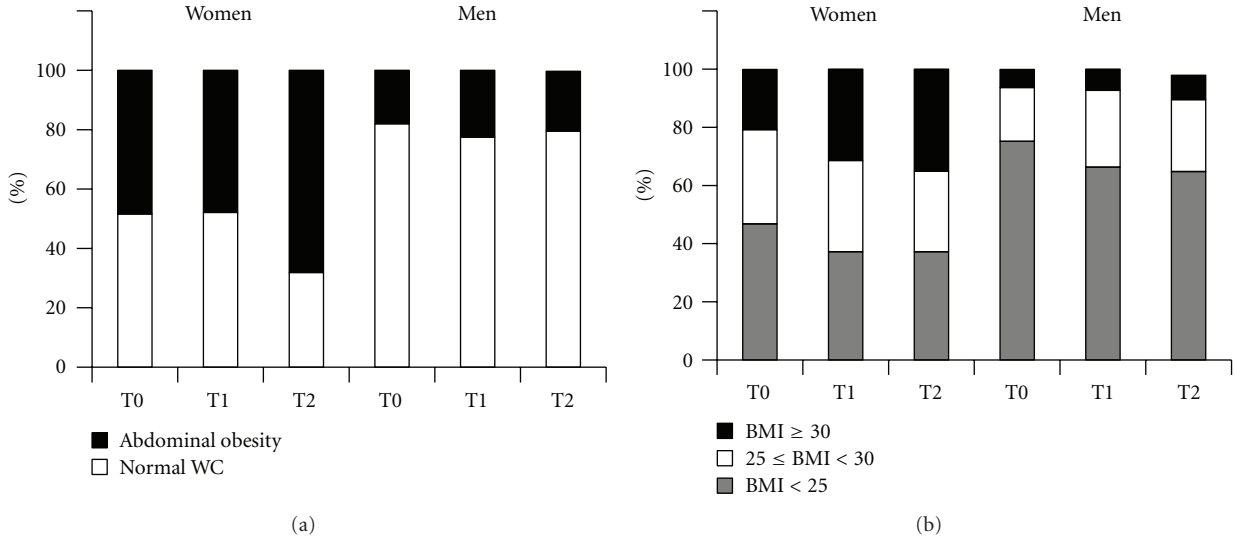


Figure 2: Changes in obesity status over the four-year followup period ($n = 366$).

obesity [8]. However, we observed the same trends when considering overweight/obesity based on BMI instead of AO based on WC (data not shown). One practical implication is that until specific cutoff points for obesity (general or abdominal) against chronic disease endpoints are validated in sub-Saharans Africans, BMI cutoff for overweight and WC cutoffs of 80 cm (for women) and 94 cm (for men) for AO might be used interchangeably for screening.

Only 16.6% of women with abdominal obesity at baseline had no other CMR factor, and the proportion was still lower among AO men (6.2%). It therefore proved impossible to assess the evolution of uncomplicated obesity. Nonetheless, we were able to verify whether baseline AO was associated with a more adverse evolution of other CMR factors, at least during a followup period of four years.

The CMR factors studied, except for the high TC/HDL-C ratio, are usually examined together as the metabolic syndrome (MetS), which is a clustering of some of the following: high blood pressure, dysglycemia, dyslipoproteinemia, and abdominal obesity [22]. However, as we wanted to isolate obesity and assess its impact on the evolution of other CMR factors, we considered the risk factors separately. Another justification for not collapsing the CMR factors into the MetS is that MetS phenotypes vary widely according to race-ethnicity [16]. High triglyceride concentrations, for instance, are not common among Africans while hypertension is highly prevalent. In populations at risk for CVD, it was reported that TC/HDL-C ratio would identify more at-risk subjects than Framingham risk scores [30]. Furthermore, TC/HDL-C showed the largest area under the curve of HOMA-IR compared with other lipid components or ratios in Indian diabetics [31]. We therefore also examined TC/HDL-C.

Control variables were those found to be significantly associated with one or the other CMR factor in the baseline study: age, physical activity, alcohol consumption, and dietary micronutrient adequacy as the only significant diet quality score [18, 19, 24, 32].

The study confirmed that obesity, which is widespread among women, was associated with elevated CMR in this sub-Saharan population. Except for high blood pressure, an upward trend of CMR markers was observed over the four-year followup both in obese and non obese individuals. The incidence of high TC/HDL-C was significantly higher in AO than nonobese subjects. The significantly higher relative risk of onset of dyslipoproteinemia in AO men (not significant in women) over the followup period, even in the adjusted model, suggests that AO as currently defined using

nonspecific cutoffs may be associated with higher CMR in men than women. This is consistent with a recent cross-sectional study in Senegal that reported less favourable CMR profiles in men than women in general, with lower HDL-C concentrations [33]. According to a recent review, higher HDL-C may be considered a gender-specific protective factor in women [34]. However, longitudinal studies of longer duration and in larger cohorts, using not only CMR factors but also disease end points would be required to confirm the findings. Because of the small number of new cases of high blood pressure and high hypertriglyceridemia, for instance, we were unable to perform logistic regression on these risk factors.

The higher observed prevalence of obesity among women than men is consistent with other studies in sub-Saharan Africa [35]. This may be partly explained by the on-going nutrition transition process in DCs with major shifts in diet and lifestyle patterns under the influence of urbanization, globalization, and economic growth [36, 37]. The observed downward trend for WC among obese subjects, at least in women, whereas the trend was positive in the nonobese, may reflect this nutrition transition process, with normal subjects progressively moving towards more abdominal adiposity; or else, these opposite trends reflect the phenomenon of regression to the mean.

In our study, the baseline proportion of insulin-resistant subjects was significantly higher in obese compared with non obese whereas no difference was observed for high fasting glycemia. Furthermore, the incidence of hyperglycemia was not different in obese and non obese while the incidence of insulin resistance tended to be higher in the obese. Indeed, under normal conditions, the pancreatic islet β-cells increase insulin release to overcome the reduced efficacy of insulin action, thereby maintaining normal glucose tolerance [38]. For obesity and insulin resistance to be associated with high fasting glycemia, β-cells must be unable to compensate fully for decreased insulin sensitivity [39]. Several studies reported the association of obesity and insulin resistance in obese Africans based on BMI, waist/hip ratio, or WC [40–42]. However, the duration of obesity is reportedly critical [43, 44], but we did not have this information.

Low HDL-C was the most prevalent CMR factor in the last followup, in both men and women (47.3% and 30.3%, resp.). Concurrently, the incidence rate of high TC/HDL-C was markedly higher in obese subjects. Triglycerides were low and remained so irrespective of obesity status. This is consistent with studies showing that low HDL-C is a major dyslipoproteinemia phenotype in obese Africans [33, 45]. In fact, studies show a gradual decrease in HDL-C levels in Africa, over the last three decades [45–47]. This decreasing trend in HDL-C levels can be attributed to the urbanization of African populations and changes in diet and physical activity. As suggested by some authors, a longer life expectancy and more access to abundant food may contribute to increased prevalence of low HDL-C in developing countries [48].

The downward trend of blood pressure means as observed in our study over four years, even excluding subjects under treatment for hypertension, cannot be fully explained, although some authors reported a weak link between obesity and high blood pressure in Africans [40, 49] while others observed an association, for instance in rural communities in south Nigeria [50]. Several cross-sectional studies in non-Africans reported the deleterious effect of obesity on blood pressure [51, 52]. The etiologic mechanism linking obesity and hypertension is the stimulation of sympathetic tone and the activation of the renin-angiotensin-aldosterone system mediated by adipocyte hormones [53]. Since Blacks had less visceral fat, this relationship between obesity and blood pressure may be weaker than in Caucasians. Another possible explanation for the reduction of mean blood pressure during the study is that individuals became more aware of their blood pressure as it was checked in the self-help groups, making them more receptive to the general health advice given to them. However, when controlling for participation in group meetings, the results were unchanged (data not shown) so that self-help groups may not be considered as confounders. Nevertheless, this does not exclude that subjects adopted a more preventive diet and lifestyle, which cannot be verified. We cannot totally exclude either technical error measurements since blood pressure was not measured by the same medical team throughout the study, although measurement techniques were standardized.

Our study has some limitations. First, only 68% of baseline participants had complete data and were included in the analysis, since significant differences in age, anthropometric status (BMI and WC) and location were observed between subjects lost to followup and those with complete followup at T1 and T2. However, this reduction in sample size did not affect statistical power substantially. The small number of obese men was nevertheless a limitation. Secondly, the relatively short followup period did not allow collecting sufficient data on hard endpoints (incidence of diabetes, CVD, or cancer) to fully assess the risk associated with obesity status. Thirdly, we did not have information on whether weight loss, when it occurred, was intentional or not. Finally, the study was only conducted in the southern part of Benin and therefore, extrapolation of the findings to other population groups requires caution. These limitations notwithstanding, the study has major strengths, including that it is the first prospective population-based study in sub-Saharan Africa which addresses the issue of cardiometabolic risk factor changes according to AO. Additionally, we were able to control for several lifestyle variables in order to explore the independent effects of baseline AO status in men and women.

5. Conclusion

Abdominal obesity as defined by high waist circumference (generic cutoffs suggested for sub-Saharan Africans) was associated with higher CMR at baseline and over the four-year followup period, except for high blood pressure, in initially apparently healthy Benin adults. Based on our findings, there was a worsening trend of CMR factors during the followup period among the abdominally obese subjects, and particularly for cholesterol profile among men. These findings support the need for obesity prevention measures. The lack of association between baseline obesity and blood pressure

changes suggests that the effect of obesity on the latter may not be important in the study population. Larger longitudinal studies of longer duration in sub-Saharan Africans are needed to confirm our findings.

Conflict of Interests

The authors declare no competing interests.

Authors' Contributions

H. Delisle designed the study. C. Sossa developed the protocol and collected the data under supervision of H. Delisle, B. Fayomi, M. Makoutodé, and V. Agueh. C. Sossa and H. Delisle analysed the data and wrote the first draft of the paper. All coauthors contributed to the revision and the finalisation of the paper.

Acknowledgments

The authors thank Professor A. Sanni from Applied Biomedical Sciences Institute for performing laboratory analyses. They would also like to express their gratitude to field workers and participants in the study. The study was funded by the Canadian International Development Agency (CIDA).

References

[1] E. E. Calle, "Obesity and cancer," *British Medical Journal*, vol. 335, no. 7630, pp. 1107–1108, 2007.

[2] K. M. V. Narayan, J. P. Boyle, T. J. Thompson, E. W. Gregg, and D. F. Williamson, "Effect of BMI on lifetime risk for diabetes in the U.S," *Diabetes Care*, vol. 30, no. 6, pp. 1562–1566, 2007.

[3] A. R. Dyer, J. Stamler, D. B. Garside, and P. Greenland, "Long-term consequences of body mass index for cardiovascular mortality: the Chicago heart association detection project in industry study," *Annals of Epidemiology*, vol. 14, no. 2, pp. 101–108, 2004.

[4] WHO/FAO Expert consultation, *Vitamin and mineral requirements in human nutrition*, Worl Health Organization, Geneva, Switzerland, 2nd edition, 2005.

[5] C. J. Girman, T. Rhodes, M. Mercuri et al., "The metabolic syndrome and risk of major coronary events in the Scandinavian Simvastatin Survival Study (4S) and the Air Force/Texas Coronary Atherosclerosis Prevention Study (AFCAPS/TexCAPS)," *American Journal of Cardiology*, vol. 93, no. 2, pp. 136–141, 2004.

[6] L. H. Lindholm and S. Mendis, "Prevention of cardiovascular disease in developing countries," *Lancet*, vol. 370, no. 9589, pp. 720–722, 2007.

[7] B. M. Popkin, "An overview on the nutrition transition and its health implications: the Bellagio meeting," *Public Health Nutrition*, vol. 5, no. 1, pp. 93–103, 2002.

[8] J. P. Després, B. J. Arsenault, M. Côté, A. Cartier, and I. Lemieux, "Abdominal obesity: the cholesterol of the 21st century?" *The Canadian Journal of Cardiology*, vol. 24, supplement D, pp. 7D–12D, 2008.

[9] S. M. Camhi, G. A. Bray, C. Bouchard et al., "The relationship of waist circumference and BMI to visceral, subcutaneous, and total body fat: sex and race differences," *Obesity*, vol. 19, no. 2, pp. 402–408, 2011.

[10] P. Deurenberg, M. Yap, and W. A. Van Staveren, "Body mass index and percent body fat: a meta analysis among different ethnic groups," *International Journal of Obesity*, vol. 22, no. 12, pp. 1164–1171, 1998.

[11] S. Lee, J. L. Kuk, T. S. Hannon, and S. A. Arslanian, "Race and gender differences in the relationships between anthropometrics and abdominal fat in youth," *Obesity*, vol. 16, no. 5, pp. 1066–1071, 2008.

[12] M. C. Désilets, D. Garrel, C. Couillard et al., "Ethnic differences in body composition and other markers of cardiovascular disease risk: study in matched Haitian and white subjects from Quebec," *Obesity*, vol. 14, no. 6, pp. 1019–1027, 2006.

[13] Y. W. Park, S. Zhu, L. Palaniappan, S. Heshka, M. R. Carnethon, and S. B. Heymsfield, "The metabolic syndrome: Prevalence and associated risk factor findings in the US population from the Third National Health and Nutrition Examination Survey, 1988–1994," *Archives of Internal Medicine*, vol. 163, no. 4, pp. 427–436, 2003.

[14] L. T. Clark, K. C. Ferdinand, J. M. Flack et al., "Coronary heart disease in African Americans," *Heart Disease*, vol. 3, no. 2, pp. 97–108, 2001.

[15] S. M. Haffner, R. D'Agostino, M. F. Saad et al., "Increased insulin resistance and insulin secretion in nondiabetic African-Americans and Hispanics compared with non-Hispanic whites: the Insulin Resistance Atherosclerosis Study," *Diabetes*, vol. 45, no. 6, pp. 742–748, 1996.

[16] H. Delisle, M.-C. Désilets, E. R. Vargas, and D. Garrel, "Metabolic syndrome in three ethnic groups using current definitions," *Applied Physiology, Nutrition and Metabolism*, vol. 33, no. 2, pp. 356–360, 2008.

[17] K. Steyn, K. Sliwa, S. Hawken et al., "Risk factors associated with myocardial infarction in Africa: the INTERHEART Africa Study," *Circulation*, vol. 112, no. 23, pp. 3554–3561, 2005.

[18] G. Ntandou, H. Delisle, V. Agueh, and B. Fayomi, "Physical activity and socioeconomic status explain rural-urban differences in obesity: a cross-sectional study in benin (West Africa)," *Ecology of Food and Nutrition*, vol. 47, no. 4, pp. 313–337, 2008.

[19] R. Sodjinou, V. Agueh, B. Fayomi, and H. Delisle, "Obesity and cardio-metabolic risk factors in urban adults of Benin: relationship with socio-economic status, urbanisation, and lifestyle patterns," *BMC Public Health*, vol. 8, article no. 84, 2008.

[20] J. Cohen, *Statistical Power Analysis for the Behavioural Sciences*, Lawrence Erlbaum, Hillsdale, NJ, USA, 1988.

[21] J. P. Després, I. Lemieux, and D. Prud'homme, "Treatment of obesity: need to focus on high risk abdominally obese patients," *British Medical Journal*, vol. 322, no. 7288, pp. 716–720, 2001.

[22] K. G. M. M. Alberti, R. H. Eckel, S. M. Grundy et al., "Harmonizing the metabolic syndrome: a joint interim statement of the international diabetes federation task force on epidemiology and prevention; National heart, lung, and blood institute; American heart association; World heart federation; International atherosclerosis society; And international association for the study of obesity," *Circulation*, vol. 120, no. 16, pp. 1640–1645, 2009.

[23] World Health Organisation, *Consultation on obesity Classification according to B. M. I.*, Geneva, Switzerland, 1993.

[24] G. Ntandou, H. Delisle, V. Agueh, and B. Fayomi, "Abdominal obesity explains the positive rural-urban gradient in the prevalence of the metabolic syndrome in Benin, West Africa," *Nutrition Research*, vol. 29, no. 3, pp. 180–189, 2009.

[25] K. G. M. M. Alberti, P. Zimmet, and J. Shaw, "The metabolic syndrome—a new worldwide definition," *Lancet*, vol. 366, no. 9491, pp. 1059–1062, 2005.

[26] D. R. Matthews, J. P. Hosker, and A. S. Rudenski, "Homeostasis model assessment: insulin resistance and β-cell function from fasting plasma glucose and insulin concentrations in man," *Diabetologia*, vol. 28, no. 7, pp. 412–419, 1985.

[27] J. Millán, X. Pintó, A. Muñoz et al., "Lipoprotein ratios: physiological significance and clinical usefulness in cardiovascular prevention," *Vascular Health and Risk Management*, vol. 5, pp. 757–765, 2009.

[28] World Health Organization, "WHO STEPS Instrument Question-by-Question," 2009, http://www.who.int/chp/steps/STEPS_QbyQ_Guide.pdf.

[29] Organisation Mondiale de la Santé, "Régime alimentaire, nutritition et prévention des maladies chroniques," Rapport d'une consultation OMS/FAO d'experts, in Série de rapports techniques 2003.

[30] T. P. Bersot, G. M. Pépin, and R. W. Mahley, "Risk determination of dyslipidemia in populations characterized by low levels of high-density lipoprotein cholesterol," *American Heart Journal*, vol. 146, no. 6, pp. 1052–1060, 2003.

[31] S. Tangvarasittichai, P. Poonsub, and O. Tangvarasittichai, "Association of serum lipoprotein ratios with insulin resistance in type 2 diabetes mellitus," *Indian Journal of Medical Research*, vol. 131, no. 5, pp. 641–648, 2010.

[32] H. Delisle, G. Ntandou, V. Agueh et al., "Urbanization, nutrition transition and cardiometabolic risk: the Benin study," *British Journal of Nutrition*. In press.

[33] A. Fontbonne, A. Cournil, C. Cames et al., "Anthropometric characteristics and cardiometabolic risk factors in a sample of urban-dwelling adults in Senegal," *Diabetes and Metabolism*, vol. 37, no. 1, pp. 52–58, 2011.

[34] O. Evangelista and M. A. McLaughlin, "Review of cardiovascular risk factors in women," *Gender Medicine*, vol. 6, no. 1, pp. 17–36, 2009.

[35] S. Dalal, J. J. Beunza, J. Volmink et al., "Non-communicable diseases in sub-Saharan Africa: what we know now," *International Journal of Epidemiology*, vol. 40, no. 4, pp. 885–901, 2011.

[36] B. M. Popkin, "Global nutrition dynamics: the world is shifting rapidly toward a diet linked with noncommunicable diseases," *American Journal of Clinical Nutrition*, vol. 84, no. 2, pp. 289–298, 2006.

[37] A. Drewnowski, "Nutrition transition and global dietary trends," *Nutrition*, vol. 16, no. 7-8, pp. 486–487, 2000.

[38] S. E. Kahn, R. L. Prigeon, R. S. Schwartz et al., "Obesity, body fat distribution, insulin sensitivity and islet β-cell function as explanations for metabolic diversity," *Journal of Nutrition*, vol. 131, no. 2, pp. 354S–360S, 2001.

[39] S. E. Kahn, "Clinical, review 135: the importance of β-cell failure in the development and progression of type 2 diabetes," *Journal of Clinical Endocrinology and Metabolism*, vol. 86, no. 9, pp. 4047–4058, 2001.

[40] A. E. Schutte, H. W. Huisman, J. M. Van Rooyen et al., "Should obesity be blamed for the high prevalence rates of hypertension in black South African women?" *Journal of Human Hypertension*, vol. 22, no. 8, pp. 528–536, 2008.

[41] A. P. Doumatey, K. S. Lashley, H. Huang et al., "Relationships among obesity, inflammation, and insulin resistance in African Americans and West Africans," *Obesity*, vol. 18, no. 3, pp. 598–603, 2010.

[42] B. K. Jacobsen, K. H. Bonaa, and I. Njolstad, "Cardiovascular risk factors, change in risk factors over 7 years, and the risk of clinical diabetes mellitus type 2: the Tromsø study," *Journal of Clinical Epidemiology*, vol. 55, no. 7, pp. 647–653, 2002.

[43] A. Abdullah, J. Stoelwinder, S. Shortreed et al., "The duration of obesity and the risk of type 2 diabetes," *Public Health Nutrition*, vol. 14, no. 1, pp. 119–126, 2011.

[44] S. G. Wannamethee and A. G. Shaper, "Weight change and duration of overweight and obesity in the incidence of type 2 diabetes," *Diabetes Care*, vol. 22, no. 8, pp. 1266–1272, 1999.

[45] A. E. Sumner, J. Zhou, A. Doumatey et al., "Low HDL-cholesterol with normal triglyceride levels is the most common lipid pattern in West Africans and African Americans with Metabolic Syndrome: Implications for cardiovascular disease prevention," *CVD Prevention and Control*, vol. 5, no. 3, pp. 75–80, 2010.

[46] A. R. P. Walker and B. F. Walker, "High high-density-lipoprotein cholesterol in African children and adults in a population free of coronary heart disease," *British Medical Journal*, vol. 2, no. 6148, pp. 1336–1337, 1978.

[47] R. W. Evans, C. H. Bunker, F. A. M. Ukoli, and L. H. Kuller, "Lipoprotein (a) distribution in a Nigerian population," *Ethnicity and Health*, vol. 2, no. 1-2, pp. 47–58, 1997.

[48] E. Windler, M. Schöffauer, and B. C. Zyriax, "The significance of low HDL-cholesterol levels in an ageing society at increased risk for cardiovascular disease," *Diabetes and Vascular Disease Research*, vol. 4, no. 2, pp. 136–142, 2007.

[49] L. Fezeu, B. Balkau, E. Sobngwi et al., "Waist circumference and obesity-related abnormalities in French and Cameroonian adults: the role of urbanization and ethnicity," *International Journal of Obesity*, vol. 34, no. 3, pp. 446–453, 2010.

[50] G. I. Ahaneku, C. U. Osuji, B. C. Anisiuba, V. O. Ikeh, O. C. Oguejiofor, and J. E. Ahaneku, "Evaluation of blood pressure and indices of obesity in a typical rural community in eastern Nigeria," *Annals of African Medicine*, vol. 10, no. 2, pp. 120–126, 2011.

[51] K. C. Zalesin, B. A. Franklin, W. M. Miller, E. D. Peterson, and P. A. McCullough, "Impact of obesity on cardiovascular disease," *Endocrinology and Metabolism Clinics of North America*, vol. 37, no. 3, pp. 663–684, 2008.

[52] P. W. Sullivan, V. H. Ghushchyan, and R. Ben-Joseph, "The impact of obesity on diabetes, hyperlipidemia and hypertension in the United States," *Quality of Life Research*, vol. 17, no. 8, pp. 1063–1071, 2008.

[53] J. E. Hall, D. A. Hildebrandt, and J. Kuo, "Obesity hypertension: role of leptin and sympathetic nervous system," *American Journal of Hypertension*, vol. 14, no. 6, pp. 103S–115S, 2001.