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Relationship between Adiposity, Low-Density Lipoprotein Particles Size and Cardiovascular Risk among Adult Obese Cameroonians

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Abstract

Background: Despite the evidence about the increasing prevalence of dyslipidemia among adult obese Cameroonians, little is known about the Low-Density Lipoprotein (LDL) particles which influence lipid metabolism and affect cardiovascular status. The present study aims to assess the relationship between adiposity, LDL particles size and cardiovascular risk (CVR) among adult obese Cameroonians. **Methods:** A cross-sectional study was conducted from September 2015 to March 2016 on apparently healthy adults (n = 1006), aged 20 - 70 years and living in the West and North-West regions of Cameroon. Anthropometric measurements, blood pressure (BP), fasting blood glucose (FBG) and lipid profile markers were analyzed and LDL particle phenotypes (LDL phenotype A; LDL phenotype I; LDL phenotype B) were characterized using small, dense LDL-cholesterol (sdLDL-c) levels. Abdominal fat accumulation (AFA) was defined as waist circumference (WC) ≥ 88 cm (men) and ≥90 cm (women) and the CVR was assessed using Framingham score method. **Results**: In the overall population, 36.6% were overweight, 33.1% were obese and 69.1% were overweight/obese with AFA. The prevalence of LDL phenotype B was 19.8%, 37.5% and 42.8% respectively in normal-weight, overweight and obese. Among the obese, sdLDL and triglycerides levels correlated significantly with WC ($r = 0.768$; $p < 0.05$ and $r = 0.768$; $p < 0.05$ re-

spectively) and body mass index (BMI) ($r = 0.895$; $p < 0.01$ and $r = 0.676$; $p < 0.01$ respectively). The risk of having LDL phenotype B in overweight/obese patients with higher CVR was three times greater in overweight/obese patients with AFA (OR: 3.1; CI 95% (0.8 - 9.1); $p = 0.007$) as compared to those without AFA (OR: 1.6; CI 95% (0.8 - 2.9); $p = 0.021$). **Conclusion:** Among obese Cameroonians, anthropometric markers of adiposity (BMI and WC) were strongly correlated to LDL phenotype B which was associated with high CVR dependently of AFA. SdLDL particles could exacerbate the CVR in obese Cameroonians subjects.

Keywords

Obese Cameroonians, LDL Particle Size, Adiposity Markers, Cardiovascular Risk

1. Introduction

Obesity is a public health problem which affects all social strata. Its progression is accentuated in both developing and developed countries by the nutritional transition commonly observed in these countries [1]. Cameroon hasn't been spared the wave of westernization and urbanization [2]. According to the Food and Agricultural Organisation (FAO), the prevalence of obesity is increasing over the past decade in the adult Cameroonian population from 4.9% in the year 2000 to 9.5% in 2016 [3]. Several epidemiological studies have shown that abdominal obesity, characterized by an excessive accumulation of fat in the intra-abdominal part of the body, more often is a key factor which contributes to the development of several cardiometabolic abnormalities such as insulin resistance, hypertension and dyslipidemia [4] [5]. Clinically, excessive abdominal fat accumulation is diagnosed by the waist circumference (WC) greater than 88 cm in women and greater than 90 cm in men [6]; and this parameter is correlated with waist to hip ratio (WHR) which is thus used as an indicator of morbidity and mortality in a population at risk of cardiovascular disease (CVD) [7]. In fact, increase in WC and/or body mass index (BMI) often affects lipid metabolism, leading to the installation of atherogenic dyslipidemia [8]. This atherogenic dyslipidemia is marked by high triglyceridemia, low HDL-c and increased formation of small-dense Low-Density Lipoprotein (sdLDL) particles which are highly implicated in atherosclerosis process [8] [9]. Meanwhile, over the past two decades, interest was paid to the predictive value of LDL particle size and the determination of LDL particles has been included in the guidelines of the American Association of Clinical Endocrinologists for the prevention of atherosclerosis [10]. SdLDL-c levels are associated with elevated triglyceride (TG) levels and low HDL-c concentrations, which constitutes the "pro-atherogenic lipoprotein phenotype", a common feature of type 2 diabetes mellitus and metabolic syndrome [11]. The potential mechanism of sdLDL phenotype may partly be attribuated to

the lower affinity for LDL receptors and its multiple atherogenic modifications in blood [12]. More often, subjects with higher sdLDL-c levels have been shown to be associated with an increased risk factor for CVD both in cross-sectional and prospective observational studies [13] [14]. However, studies focusing on the relationship between LDL particle phenotypes with adiposity markers and cardiovascular risk among Cameroonians are scarce. Most studies have simply focused on the effect of LDL-c concentration on CVD risk in obese Cameroonians. The objective of this study was to investigate the relationship between adiposity markers (BMI, WC), LDL particles size and cardiovascular risk (CVR) among adult obese Cameroonians, in order to manage intra-abdominal fat accumulation and so prevent early mortality due to cardiovascular diseases in our population.

2. Material and Methods

2.1. Study Design and Setting Population

In a cross-sectional and descriptive survey which took place from September 2015 to March 2016, adult Cameroonians origin of both sex, aged between 20 - 70 years were randomly selected during mass health campaigns on nutritional and cardio-metabolic risk factors surveys. Residing participants in the selected areas made up the study population. The selected areas included urban and rural areas of West and North-West Region of Cameroon. From the western region of Cameroon, Bafoussam, the capital city; Mbouda town, the headquarter of the Bamboutos division; Babadjou, a village situated at about 12 km of Mbouda; Dschang town, located in the Menoua department; Foumban, located in the Noun department; Bafou village, situated in the Menoua division made up the study sites from this region. The study sites from the North West region included Wum, Mbengwi, Ndu towns and Nyen village [15].

2.2. Inclusion and Exclusion Criteria

Inclusion criteria of participants to the study included: 1) subjects aged 20 to 70 years, with no concomitant diseases, and without any treatment; 2) stable body weight (±2 kg) for at least three months before the survey, without any use of medication known or suspected to affect body weight or appetite; 3) no weight loss attempts through dietary intervention over the three months before the survey; 4) no pregnancy. Were excluded from the survey, participants with BMI lower than 18.5 kg/m^2 and those with known metabolic diseases namely hypertension, diabetes, hyperlipidemia, liver and kidney diseases. Based on the above criteria, 1006 consenting volunteers were selected and included in the study.

2.3. Sampling Procedure

The sample size was calculated based on the formula for basic sample size calculation for random sampling [16] [17]. The 95% confidence level and 11.9% prevalence of obesity in Cameroon [18] were used. 314 subjects were required for

the minimum sample size, but 333 obese subjects were included in the study to allow for precision. Knowing that this was a case study, 368 overweight subjects and 305 normal weight subjects were also included. The total sample size required for this study was 1006 subjects. Age, sex, smoking, personal history of hypertension or diabetes, as well as the use of antihypertensive, lipid-lowering, antidiabetic medications and pre-existent medical conditions were assessed by self-report through a face-to-face interview conducted by a well-trained surveyor in data collections sites (health districts, health centers, churches, palace place) using a questionnaire which was conceived from the WHO STEPWISE questionnaire [19] and pre-tested one month prior to the survey.

2.4. Anthropometric Measurements

Height (to the nearest centimetre) was measured with a locally manufactured wall mounted stadiometer calibrated against the Cameroon's Department of National Security identification. Body weight (to the nearest kilogram), was assessed using a Tanita™ BC-418 Segmental Body Composition Analyzer/Scale with participants wearing light clothing. Body mass index (BMI) was computed for all participants as a ratio of body weight to height squared and expressed in kg/m^2 [20]. Participants were classified as obese if they had a body mass index (BMI) ≥ 30 kg/m^2, overweight for a $25 \leq BMI \leq 29.9$ kg/m^2 and normal weight for $18.5 \leq BMI \leq 24.9$ kg/m^2 [21]. Waist circumference (WC) and hip circumference (HC) measurements to the nearest 0.1 cm were assessed according to the World Health organization (WHO) guidelines, using flexible but non-stretchable tapes measured at the mid-point between the last rip and the iliac crest, and at the level of the largest lateral extension of the hips, both in a horizontal plane [22]. WC greater than 88 cm in women and greater than 90 cm in men defined abdominal fat accumulation (AFA) [6]. Waist-to-hip ratio (WHR) was the waist circumference divided by the hip circumference.

2.5. Blood Pressure Assessment

Blood pressure (BP) was measured two times using a mercury sphygmomanometer (Life source™). The first measurement was taken after a ten minutes rest on a sitting position and the following measurements were taken every 5 minutes thereafter. The BP values were the mean of the two measurements. Elevated BP was defined as: systolic blood pressure (SBP) ≥ 140 mmHg, and/or diastolic blood pressure (DBP) ≥ 90 mmHg [21].

2.6. Blood Sampling and Biochemical Analysis

About 5 mL of a 12-hour overnight fasted venous blood was collected on a free anticoagulant tube to each participant. Serum was obtained by collecting whole blood into 5 ml clot activator tube from each participant, samples were allowed to clot for one hour at room temperature and then centrifuged for 10 minutes at 1000 g, 1 mL aliquote of serum was pipetted into labelled cryovials and imme-

diately stored at −70˚C for biochemical assessment.

- **Fasting blood glucose, insulin and insulin resistance assessment**

Fasting blood glucose (FBG) level was measured by the glucose oxidase method [23] using a spectrophotometer (spectrolumb™, X-3456, USA). Elevated FBG was defined as FBG ≥ 100 mg/dl or 5.6 mmol/L and diabetes was defined as FBG ≥ 126 mg/dL [24]. Serum insulin level was quantified 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. Homeostasis assessment model for insulin resistance (HOMA-IR) was used to evaluate insulin resistance (IR) using the following formula: HOMA-IR = Fasting blood glucose (mmol/L) × Fasting insulin (µU/mL)/22.5; with a cut-off value for IR diagnosis HOMA-IR ≥ 2.6 [25].

- **Lipid profile markers assessment**

Serum total cholesterol (TC), HDL cholesterol (HDL-c), and triglycerides (TG) were assessed with standard enzymatic spectrophotometric methods (Kit ChronoLab™) [26] [27] [28]. High total cholesterol (TC) cut-off value was TC ≥ 200 mg/dL; Low HDL cholesterol cut-offs were HDL-c < 40 mg/dL (for men) and < 50 mg/dL (for women) andhypertriglyceridemia (HyperTG) was defined as triglycerides ≥ 150 mg/dL [29]. Serum LDL-cholesterol (LDL-c) was measured by the homogenous assays [30] andLDL-c ≥ 130 mg/dL was defined as high LDL-c [29].

- **Determination of small-dense LDL cholesterol by simple precipitation method**

Small, dense LDL cholesterol (sdLDL-c) was measured quantitatively by heparin-magnesium precipitation procedure according to Hirano *et al.* method [31]. Accordingly, precipitation reagent (0.1 mL) containing 150 U/mL heparin-sodium salt and 90 mmol/L $MgCl_2$, was added to equal volume serum (0.1 mL), mixed and incubated at 37˚C for 10 minutes, and then chilled on ice for 15 minutes followed by centrifugation at 100,000 g for 15 minutes at 4˚C in order to pellet the precipitate. An aliquot of the supernatant was removed for LDL-c analysis. Measured LDL-c content of the supernatant by the homogenous assays [30] was stated as sdLDL-c. The sdLDL-c percentage was determined by the ratio of sdLDL-c to LDL-c (sdLDL/LDL-cx100).

- **LDL particle size estimation**

LDL particle size was estimated using sdLDL concentration according to the protocol described by Srisawasdi *et al.* [32]. A lower concentration of sdLDL (sdLDL < 23.9 mg/dL) predicts the presence of large and buoyant LDL particles (lbLDL) named LDL phenotype A. A concentration of sdLDL range between 24 mg/dL and 34.9 mg/dL (24 mg/dL - 34.9 mg/dL) predicts the presence of the intermediate LDL particles named LDL phenotype I and a higher concentration of sdLDL (sdLDL ≥ 35 mg/dL) predicts the presence of small and dense LDL particles named LDL phenotype B [32].

2.7. Assessment of Cardiovascular Risk

The global cardiovascular risk (CVR) was assessed using Framingham score as described by D'Agostino *et al.* [33]. The Framingham score was computed using some cardiovascular risk factors: age, HDL-c level, total cholesterol levels, systolic hypertension, diabetic status (diabetic and non diabetic) and smooking habits: never (non smoker), past or current (smoker) [34]. Current smoker was defined as a subject who smokes at least one cigarette per day for more than 6 months. According to gender, scores were allocated to each group of participants and total score was computed to estimate percentage of CVR. CVR categories were stratified as follows: Low cardiovascular risk: CVR less than 10%; Moderate cardiovascular risk: CVR from 10% to 20%; High cardiovascular risk: CVR more than 20% [33].

2.8. Ethical Consideration

This study was approved by the National Ethic Committee of research for Human Health of Cameroon (No. 2014/08/488/CE/CNERSH/SP). Authorizations were obtained from local and administrative authorities of each area of the capital city and two regions. Written consent was obtained from each participant who agreed to participate.

2.9. Statistical Analysis

Data were analyzed using the IBM SPSS statistical software package version 20.0. Descriptive analysis included the estimation of mean values and standard deviations for continuous variables. Categorical variables were compared by the Chi square test and continuous variables compared by one way analysis of variance (ANOVA) followed by post hoc LSD. All the data were expressed as means ± standard error of means and frequencies (%). Pearson correlation was used to analyze the correlation between two quantitative variables. Odds Ratio (OR) with a 95% confidence interval (95% CI) was performed by logistic regression analysis to quantify the relationship between two variables. P-value was set at 0.05.

3. Results

3.1. Description of the Study Population

Anthropometric, clinical and biochemical characteristics are recorded in **Table 1**. Out of 1006 participants of this study, 30.3% (n = 305) were normal weight, 36.6% (n = 368) were overweight and 33.1% (n = 333) were obese. The percentage of obesity according to sex was 7.9% (n = 69) and 26.2% (n = 264) respectively in men and women. The mean age was 45.1 ± 1.1, 50.2 ± 0.8 years and 48.1 ± 0.7 years respectively for normal weight, overweight and obese participants. Normal weight participants were significantly younger than overweight (45.1 ± 1.1 years vs. 50.2 ± 0.8 years; $p < 0.05$) and obese (45.1 ± 1.1 years vs. 48.6 ± 0.7 years; $p < 0.05$). The total cholesterol (TC) level was significantly higher among

Table 1. Anthropometric, clinical and biochemical characteristics of participants.

Characteristics	Overall	Normal weight	Overweight	Obese
N (%)	1006	305 (30.3)	368 (36.6)	333 (33.1)
Men n (%)	268 (26.6)	103 (10.2)	96 (9.5)	69 (7.9)
Women n (%)	738 (73.4)	202 (20.1)	272 (27.0)	264 (26.2)
Age (years)	48.1 ± 0.5	45.1 ± 1.1	50.2 ± 0.8§	48.6 ± 0.7‡
BMI (kg/m^2)	29.3 ± 0.3	23.0 ± 0.1	27.3 ± 0.1§	37.7 ± 0.5$^{\ddagger,\Psi}$
WC (cm)	85.0 ± 1.0	66.4 ± 2.0	89.0 ± 1.0§	96.4 ± 1.2$^{\ddagger,\Psi}$
HC (cm)	101.6 ± 0.5	93.1 ± 1.0	102.2 ± 0.7§	108.8 ± 0.8$^{\ddagger,\Psi}$
WHR	0.8 ± 0.2	0.7 ± 0.1	0.9 ± 0.3	0.9 ± 0.4
SBP (mmHg)	137.0 ± 1.0	133.0 ± 2.0	137.0 ± 2.0	141.2 ± 1.5$^{\ddagger,\Psi}$
DBP (mmHg)	84.5 ± 0.8	78.0 ± 2.0	83.8 ± 1.3§	91.5 ± 1.1$^{\ddagger,\Psi}$
Pool (bat/min)	70.3 ± 1.0	66.0 ± 2.0	68.2 ± 1.2	75.8 ± 1.0$^{\ddagger,\Psi}$
FBG (mg/dL)	114.0 ± 1.5	110.5 ± 3.0	107.5 ± 2.3	124.3 ± 2.5$^{\ddagger,\Psi}$
Total cholesterol (mg/dL)	160.1 ± 3.0	151.3 ± 4.3	138.32 ± 3.0§	187.1 ± 5.7$^{\ddagger,\Psi}$
Triglycerides (mg/dL)	116.0 ± 2.0	113.2 ± 4.1	107.2 ± 3.0	128.1 ± 3.3$^{\ddagger,\Psi}$
HDL-cholesterol (mg/dL)	26.4 ± 0.4	26.1 ± 1.0	26.1 ± 1.0	27.0 ± 0.6
LDL-cholesterol (mg/dL)	111.3 ± 2.4	100.91 ± 4.01	114.8 ± 4.0§	116.93 ± 4.4‡
sd LDL-c (mg/dL)	31.0 ± 0.7	25.0 ± 1.0	30.8 ± 1.1§	36.6 ± 1.3$^{\ddagger,\Psi}$
sdLDL %	27.6 ± 1.2	24.5 ± 2.0	26.6 ± 2.1	31.0 ± 2.2$^{\ddagger,\Psi}$
TG/HDL-c ratio	7.8 ± 0.4	7.3 ± 0.6	8.2 ± 0.8	7.8 ± 0.8
Insulin (µL/mL)	21.4 ± 0.4	14.9 ± 0.2	19.5 ± 0.2§	29.4 ± 1.2$^{\ddagger,\Psi}$
HOMA-IR	3.2 ± 0.1	1.4 ± 0.1	2,61 ± 0.1§	5.42 ± 0.2$^{\ddagger,\Psi}$
Smoking				
No (%)	684 (68.0)	247 (81.0)	235 (63.9)	202 (60.7)
Yes (%)	322 (32.0)	58 (19.0)	133 (36.1)	131 (39.3)

SBP: Systolic Blood Pressure, DBP: Diastolic Blood Pressure, BMI: Body Mass Index, WC: waist circumference, HC: Hip circumference, WHR: Waist-to-Hip ratio; FBG: Fasting Blood Glucose, LDL-c: Low density Lipoprotein cholesterol; HDL-c: High Density Lipoprotein cholesterol; sdLDL: small, dense LDL; sdLDL %: small, dense LDL percentage are values of the ratio sdLDL/LDL-c in percentage. ‡Mean values significantly different between obese and normal weight groups. §Mean values significantly different between overweight and normal weight groups. $^{\Psi}$Mean values significantly different between overweight and obese groups.

obese than normal weight subjects (187.1 ± 5.7 mg/dL vs. 151.3 ± 4.3 mg/dL; $p < 0.05$). LDL-cholesterol (LDL-c) levels were significantly higher in obese patients as compared to overweight patients (116.93 ± 4.4 mg/dL vs. 114.8 ± 4.0 mg/dL; $p < 0.05$). Fasting blood glucose (FBG) levels were significantly higher in obese pa-

tients as compared to normal weight (124.3 ± 2.5 mg/dL vs. 110.5 ± 3.0 mg/dL, $p < 0.05$). The small, dense LDL (sdLDL) levels were significantly higher among the obese compared to normal weight (36.6 ± 1.3 mg/dL vs. 25.0 ± 1.0 mg/dL; $p < 0.05$).

3.2. LDL Particle Phenotype Assessment According to Body Mass Index and Waist Circumference Categories

Table 2 shows that the variation of LDL phenotype's frequency depends on BMI and WC values. The evolution of LDL particle size was inversely proportional to the BMI and waist circumference values. In fact according to BMI, frequency of the LDL phenotype A (large and buyant LDL particles) was significantly high in normal-weight subjects group (BMI: 18.5 - 24.9 kg/m^2) compared to overweight (BMI: 25 - 29.9 kg/m^2) (38.6% vs. 34.1%; $p < 0.05$) and obese (BMI ≥ 30) (38.6% vs. 27.3%; $p < 0.05$) whereas the frequency of LDL phenotype B (small and dense LDL particles) was significantly lower in normal-weight subjects group (BMI: 18.5 - 24.9 kg/m^2) compared to overweight (BMI: 25 - 29.9 kg/m^2) (19.8% vs. 37.5%; $p < 0.05$) and obese (BMI ≥ 30) (19.8% vs. 42.8%; $p < 0.05$). According to WC in men group, the frequency of LDL phenotype A was significantly lower in those with abdominal fat accumulation (AFA) (WC ≥ 90 cm) compared with those without AFA (WC < 90 cm) (41.1 vs. 58.9%; $p < 0.05$) whereas the frequency of LDL phenotype B was significantly high in men with AFA (WC ≥ 90 cm) compared with those without AFA (WC < 90 cm) (64.6% vs. 35.4%; $p < 0.05$). Similars results were obtained in women group.

Table 2. LDL phenotype frequency according to BMI and WC categories.

		LDL particles phenotypes % (n)			
		phenotype A	phenotype I	phenotype B	Total
BMI (Kg/m^2)					
18.5 -24.9		38.6 (187)*	27.9 (51)*	19.8 (67)*	30.3 (305)
25 - 29.9		34.1 (165)	41.5 (76)	37.5 (127)	36.6 (368)
≥30		27.3 (132)	30.6 (56)	42.8 (145)	33.6 (333)
Total		(48.1) 484	(18.2) 183	33.7 (339)	1006
WC (cm)					
Men (n = 268)	<90	58.9 (73)*	47.9 (23)*	35.4 (34)*	48.5 (130)
	≥90	41.1 (51)	52.1 (25)	64.6 (62)	51.5 (138)
Total		46.3(124)	17.9 (48)	35.9 (96)	268
Women (n = 738)	<88	52.8 (190)*	44.8 (60)	36.2 (88)*	45.8 (338)
	≥88	47.2 (170)	55.2 (75)	63.9 (155)	54.2 (400)
Total		48.7 (360)	18.3 (135)	33.1 (243)	738

*$p < 0.05$: comparison of LDL phenotype frequency between BMI and WC categories groups.

3.3. Relationship between Lipid Profile Parameters, Small, Dense LDL and Adiposity Markers

In appears from Table 3, that in normal-weight group, no significant correlation was found between anthropometric markers of adiposity (BMI and WC) and lipid profile markers. But important correlations were found between these markers among overweight and obese groups. Indeed among obese, a strongly positive relationship was noted between TG/HDL-c ratio and WC ($r = 0.996$; $p < 0.01$); BMI ($r = 0.906$; $p < 0.01$) and WHR ($r = 0.989$; $p < 0.01$). Among overweight, sdLDL levels were significantly associated with WC ($r = 0.986$; $p < 0.01$) and BMI ($r = 0.956$; $p < 0.01$). Whereas among obese, it was significantly associated with WC ($r = 0.789$; $p < 0.01$) and BMI ($r = 0.895$; $p < 0.05$). On the same hand, significant relationship between adiposity markers and % sdLDL was noticed both in overweight (WC, $r = 0.565$; BMI, $r = 0.246$) and obese (WC, $r = 0.878$ and BMI, $r = 0.991$). TG levels were also positively associated to WC ($r = 0.768$; $p < 0.05$) and BMI ($r = 0.676$; $p < 0.01$) in obese group only with BMI ($r = 0.356$; $p < 0.05$). HDL-c level shows significant relationship with WC (0.392; $p < 0.01$) only among overweight group. BMI was the only adiposity maker with a positive relationship with LDL-c ($r = 0.812$; $p < 0.01$) among the obese subjects group.

3.4. Frequency of Metabolic Risk Factors According to BMI and WC Categories

The results for the frequencies of cardiometabolic risk factors and cardiovascular risk by BMI and waist circumference (WC) categories are reported in Table 4. It was found that the frequencies of insulin resistance (IR) and hyperTG increased as one moves from a normal-weight individual with a low BMI to a high-weight

Table 3. Pearson correlation between anthropometric markers of adiposity, lipid profil and small, dense LDL in normal-weight, overweight and obese subjects.

Parameters	Coefficient of correlation (r)								
	Normal-weight (n = 305)			Overweight (n = 333)			Obese (n = 368)		
	WC	BMI	WHR	WC	BMI	WHR	WC	BMI	WHR
TG	0.005	0.021	0.002	0.041	0.356*	0.289	0.768*	0.676**	0.306
TC	0.087	0.063	0.003	0.053	0.589	0.298	0.465	0.386	0.344
LDL-c	0.003	0.012	0.009	0.512	0.012	0.042	0.349	0.812**	0.219
HDL-c	0.009	0.032	0.023	0.392**	0.382	0.982	0.902	0.382	0.404
TG/HDL-c	0.008	0.062	0.082	0.064	0.086	0.008	0.996**	0.906**	0.989*
sdLDL	0.076	0.056	0.009	0.986**	0.956**	0.396	0.789**	0.895*	0.856
% sdLDL	0.096	0.067	0.041	0.565**	0.246**	0.294	0.878**	0.991*	0.756

TG: Triglycerides; TC: Total cholesterol; sdLDL: small, dense LDL; WC: Waist circumference, BMI: Body mass index; WHR: Waist-to-hip ratio. *p: correlation is signifiacant at the 0.05 (2-tailed), **p: correlation was significant at the 0.01 (2-tailed). $r = 0.689$; $p < 0.01$.

Table 4. Frequency of cardiometabolic factors and cardiovascular risk according to BMI categories.

CMR Factors N (%)	Normal weight 305 (30.3)	Overweight 368 (36.6)	Obese 303 (30.1)	Overweight/obese without AFA 210 (29.9)	Overweight/obese with AFA 491 (69.1)
Elevated BP					
No	237 (77.7)	267 (72.6)	61.0 (203)	76.2 (160)	63.1 (310)
Yes	68 (22.3)	101 (27.4)	39.0 (130)	23.8 (50)	36.9 (181)
Insulinresistance					
No	94.4 (289)	40.8 (150)	9.9 (33)	33.8 (71)	22.8 (112)
Yes	5.6 (16)	59.2 (218)	90.1 (300)	66.2 (139)	77.2 (379)
HyperTG					
No	75.4 (230)	80.4 (296)	47.4 (158)	76.2 (160)	73.7 (362)
Yes	24.6 (75)	19.6 (72)	52.6 (175)	23.1 (50)	26.3 (129)
Hyperglycemia					
No	53.8 (164)	59.8 (220)	44.1 (147)	33.8 (71)	22.8 (112)
Yes	46.2 (141)	40.2 (148)	55.9 (186)	66.2 (139)	77.2 (379)
Low HDL					
No	92.8 (283)	8.4 (31)	3.6 (12)	5.7 (12)	6.3 (31)
Yes	7.2 (22)	91.6 (337)	96.4 (321)	94.3 (198)	93.7 (460)
Cardiovascular risk levels					
Low CVR	51.1 (156)	42.9 (158)	35.1 (117)	56.7 (119)	33.2 (163)
Moderate CVR	18.3 (51)	25.3 (93)	27.6 (92)	22.9 (48)	27.9 (137)
Higher CVR	5.4 (93)	31.8 (117)	37.2 (124)	20.5 (43)	38.9 (191)

CMR: cardiometabolic risk; AFA: abdominal fat accumulation; hyperTG: hyper triglyceridemia.

(overweight and obese) individual with a high BMI. These frequencies vary in the same direction as one moves from an individual with abdominal fat accumulation (overweight/obese with a high waist circumference) to an overweight/obese without abdominal fat accumulation (overweight/obese with a lower waist circumference). As for low HDL, it appears that its frequency was higher in the overweight/obese group with abdominal fat accumilation than in overweight/obese without abdominal fat accumulation (94.3% vs. 93.7%). Considering cardiovascular risk, the high CVR increased proportionally with BMI. As expected, the highest CVR was found in obese subjects (37.2%) followed by overweight (31.8%) and the lowest in normal subjects (5.4%). This was made wores by abdominal fat accumulation (AFA). This was confirmed when the low CVR was calculated otherwise. The lowest CVR was found in normal weight subjects (51.1%) compared to overweight (42.9%) and obese subjects (35.1%) (Table 4).

3.5. Assessment of Odd Ratio of Cardio-Metabolic Abnormalities Related to LDL Phenotype Bamong Participants

The results for the association between cardiometabolic risk factors and LDL phenotype by BMI and WC categories are reported in Table 5. It was found that in obese individuals, LDL phenotype B was associated with insulin resistance (OR = 1.1; 95% CI: 0.7 - 3.2; p = 0.001), Low HDL-c (OR = 4.8; CI (0.4 - 54.6); p = 0.001) and hyperTG (OR = 7.3; 95% CI (4.2 - 12.4); p = 0.003). Dependantly with abdominal fat accumulation, the result showed that in obese patients with abdominal fat accumulation (AFA), LDL-phenotype B was associated with insulinresistance (OR = 1.6; 95% CI (1.0 - 2.6); p = 0.034), hyperTG (OR = 4.9; 95% CI (3.1 - 7.6); p = 0.001) and low HDL-c (OR = 5.0; CI (1.3 - 18.0); p = 0.001), this odd ratio was two folds higher than that obtained in obese/overweight without AFA (OR = 2.6; 95% CI (1.4 - 4.7); p = 0.003). On the same hand, the LDL phenotype B was three time more associated to high CVR (OR = 3.1; 95%

Table 5. Multivariable logistic regression analysis association of risk factors and cardiovascular risk for LDL phenotype B in overweight and obese with and without abdominal fat accumulation.

		Odd Ratio (CI 95%) p-value									
CMRF	Reference (LDL-p A)	Normal weight (n = 305)		Overweight (n = 368)		Obese (n = 333)		Overweight/obese without AFA (n = 210)		Overweight/obese with AFA (n = 491)	
Elevated BP	1	0.5 (0.2 - 1.0)	0.058	0.8 (0.4 - 1.5)	0.510	1.0 (0.5 - 1.9)	0.946	1.3 (0.6 - 2.6)	0.437	1.1 (0.7 - 1.6)	0.711
Hyperglycemia	1	1.0 (0.5 - 1.9)	0.936	1.4 (0.8 - 2.5)	0.156	0.6 (0.3 - 1.3)	0.549	0.5 (0.2 - 1.0)	0.385	1.2 (0.8 - 1.8)	0.409
Insulin-resistance	1	0.6 (0.3 - 1.1)	0.122	1.3 (0.7 - 2.4)	0.251	1.1 (0.7 - 3.2)*	0.001	1.1 (0.5 - 2.0)	0.857	1.6 (1.0 - 2.6)*	0.034
Low HDL-c	1	1.2 (0.4 - 3.8)	0.649	2.1 (0.7 - 9.3)*	0.001	4.8 (0.4 - 54.6)*	0.001	2.6 (1.4 - 4.7)*	0.003	5.0 (1.3 - 18.0)*	0.001
Hyper-LDL	1	0.1 (0.1 - 0.2)	0.001	0.8 (0.3 - 2.2)	0.001	0.8 (0.1 - 4.7)	0.001	0.5 (0.1 - 3.1)	0.000	1.1 (0.5 - 2.4)	0.061
Hyper-TG	1	0.6 (0.2 - 1.5)	0.341	4.3 (2.0 - 9.4)*	0.001	7.3 (4.2 - 12.4)*	0.003	1.6 (0.8 - 3.4)	0.150	4.9 (3.1 - 7.6)*	0.001
		Cardiovascular risk status									
Low CVR	1	1.0 (0.3 - 3.8)*	0.004	0.4 (0.2 - 0.9)	0.081	0.7 (0.4 - 1.4)	0.069	0.5 (0.3 - 1.1)	0.059	0.6 (0.4 - 1.5)	0.078
Moderate CVR	1	0.8 (0.2 - 1.8)	0.087	0.6 (0.2 - 1.3)	0.067	1.1 (0.6 - 1.8)*	0.002	0.9 (0.3 - 1.2)	0.063	0.9 (0.4 - 1.6)	0.098
High CVR	1	0.7 (0.2 - 1.4)	0.676	1.1 (0.5 - 2.3)*	0.003	1.5 (0.5 - 2.4)*	0.004	1.6 (0.8 - 2.9)*	0.021	3.1 (0.8 - 9.1)*	0.007

CMRF: cardiometabolic risk factors; CVR: cardiovascular risk; LDL-pA: LDL phenotype A; Hyper-TG: hypertriglyceridemia; HyperLDL: LDL hypercholesterolemia; AFA: abdominal fataccumulation; BP: blood pressure; OR: Odd Ratio. *significant odd ratio at $p < 0.05$.

CI (0.8 - 9.1); p = 0.007) in overweight/obese with AFA compared to those without AFA (OR = 1.6; 95% CI (0.8 - 2.9); p = 0.021). This result shows that hyperTG is a key factor of the metabolic parthway of small, dense LDL (LDL phenotype B) synthesis, which increases the CVR through abdominal fat accumulation in overweight and obese.

3.6. Assessment of Cardiovascular Risk According to LDL Particle Phenotypes in Overweight/Obese Subjects with AFA and without AFA

It appears in Table 6 that, the LDL phenotype A was significantly associated with Low CVR both in overweight/obese subjects with abdominal fat accumulation and without abdominal fat accumulation (AFA), but the odd ratio was twice as high in overweight/obese subjects without AFA (OR = 3.4; 95% CI (0.9 - 5.5); p < 0.05) compared to those with AFA (OR: 1.7 (0.5 - 2.9); p < 0.05). This result shows that LDL phenotype A was associated with the low CVR independently of AFA.

4. Discussion

The present study was carried out to provide information on the relationship between adiposity markers (BMI, WC), LDL particle size and cardiovascular risk in overweight and obese Cameroonian subjects. In the overall population, the proportion of obese was 33.1% (7.8% for men and 26.2% for women) and that of overweight was 36.6% (9.6% for men and 27.0% for women) based on BMI (Table 1). This result is not in accordance with the report of the study of Simo *et al.* (2021) carried out on selected health areas in a rural health district in Cameroon, where a prevalence of overweight and obesity were 31.1% and 18.9% respectively [35]. The prevalence of obesity of 33.1% reported in this study is about three folds that reported by Aminde *et al.*, 2017 (11.1%) in the semi-urban community of Buea (a city of Cameroon), though they reported a slightly similar prevalence of overweight of 36.5% [36]. The prevalence of obesity in our study is about six folds higher than that reported by Sobngwi *et al.*, 2002 in rural western

Table 6. Odd ratio of having LDL phenotype A by cardiovascular risk categories in overweight/obese subjects with abdominal AFA and without AFA.

	Odd Ratio (CI: 95%)					
	Low CVR		Moderate CVR		Higher CVR	
	overweight/obese with AFA	overweight/obese without AFA	overweight/obese with AFA	overweight/obese without AFA	overweight/obese with AFA	overweight/obese without AFA
LDL-pB (reference)	1	1	1	1	1	1
LDL-pA	1.7 (0.5 - 2.9)*	3.4 (0.9 - 5.5)*	1.3 (0.8 - 2.5)*	0.5 (0.3 - 2.1)	0.8 (0.4 - 1.2)	0.9 (0.5 - 2.8)

*significant odd ratio at p < 0.05; CI: confidence intervalle; AFA: abdominal fat accumulation; CVR: Cardio-Vascular Risk, LDL-pA: LDL phenotype A, LDL-pB: LDL phenotype B.

Cameroon [37]. In the overall overweight/obese population, the prevalence of overweight/obese with abdominal fat accumulation (AFA) based on WC was higher compared to overweight/obese without AFA (69.1% vs. 29.9%). The higher prevalence of AFA in overweight/obese could be due to the fact that the frequencies of obesity and overweight were both higher in women than in men. In fact when women experience menopause, estrogen declines rapidly and follicle stimulating hormone increases. As a result, the accumulation of abdominal fat is exacerbated [38]. Therefore the prevalence of AFA would increase more rapidly in women.

Concerning LDL particles characterization, this is the first study that describes the distribution of LDL phenotypes in overweight and obese adults Cameroonians. According to BMI, the prevalence of LDL phenotype A was 38.6%, 34.1% and 27.3% respectively in normal-weight, overweight and obese whereas the prevalence of LDL phenotype B was 19.8%, 37.5% and 42.8% respectively in normal-weight, overweight and obese; the prevalence of LDL phenotype I was 27.9%, 41.5% and 30.6% respectively in normal-weight, overweight and obese (**Table 2**). This result shows that the formation of large and buoyant LDL particles (LDL phenotype A) decreases from normal-weight to obese passing through the overweight stage whereas the formation of small-dense LDL particles (LDL phenotype B) increases gradually from normal-weight to obese passing through the overweight stage. According to the WC in male group, the frequency of LDL phenotype A was significantly lower in those with abdominal fat accumulation (AFA) (WC ≥ 90 cm) compared to those without AFA (WC < 90 cm) whereas the frequency of LDL phenotype B was significantly higher in men with AFA (WC ≥ 90 cm) compared with those without AFA (WC < 90 cm). Similar results were obtained in the female group. All these results emphasize the fact that, the accumulation of the fat in adipose tissue (diagnosed by BMI and WC) in general and mainly in the abdominal region of the body is responsible to the higher secretion of small, dense LDL. This finding supports the idea of Despres (2012) which reported that the AFA has been associated with hypertriglyceridemia and it had been suggested that the overproduction of triglycerides may act as a mechanism through which sdLDL particles are produced [39]. In fact, sdLDL formation is initiated by the delipidation of triglyceride-rich lipoproteins catalyzed by lipoprotein lipase and hepatic lipase enzymes [40]. Besides, excess intra-abdominal adiposity increases overall cardiometabolic risk partially through alterations in the secretion of a series of biologically active molecules (adipokines). These include increased secretion of free fatty acids which are incorporated in triglyceride and so far responsible for over production of sdLDL particles in blood. In fact, higher triglycerides concentration promote TG transfer from VLDL to HDL and then, the enriched-TG HDL, tranfers TG to LDL and remove cholesterol from LDL. The cholesterol-depleted LDL becomes smaller and denser [41].

The correlation between adiposity markers and lipid profile (**Table 3**) shows that a significant relationship was found between adiposity markers and small-dense

LDL (sdLDL) both in overweight and obese patients; TG also shows a significant relationship with WC and BMI in obese and only with BMI in the overweight group but not in the normal weight group. This result shows that BMI and WC are slightly associated both with sdLDL and TG in subjects with body fat accumulation (overweight and obese). The present study shows that, the circulating sdLDL and TG concentration depend on WC and BMI values. These results support studies which had previously demonstrated the correlation between anthropometric parameters and lipid profil parameters [42] [43]. In the same hand, the study of Xi *et al.* [8] have shown that increase of some ahthropometric parameters value such as WC and BMI is often assoiated with high triglyceridemia, low HDL-c and increased formation of small-dense Low-Density Lipoprotein (sdLDL) particles which are highly implicated in then atherosclerosis process. The contribution of body fat accumulation to the LDL particle size variation has been observed in this study. In fact, the mechanisms of the linkage between fat accumulation and sdLDL in obese and overweight can be explained by the fact that relative weight correlated positively with hepatic lipase [44], which is considered to be a key enzyme for the production of sdLDL [40] and this enzyme is more active in body fat accumulation status. Furthermore, growth hormone also affects LDL particle size. In men with AFA and with blunted growth hormone secretion, growth hormone treatment marginally increased the mean LDL diameter [45]. On the metabolic point of view, serum TG, mainly very low-density lipoprotein (VLDL)-TG, is a major contributor to LDL particle size modification. In the Framingham study, TG was the single most important factor affecting LDL particle size [46].

The association between cardiometabolic risk factors and adiposity markers shows that the frequencies of IR, hyperTG and Low HDL increased in proportion to BMI and WC values. In other words, the frequencies of IR, hyperTG and Low HDL were highest in obese and overweight subjects compared to normal-weight group. However, when a comparison was made between overweight/obese with AFA and without AFA, it was found that the frequencies of IR, hyperTG and Low HDL were highest in overweight/obese with AFA than in overweight/obese without AFA. The assessment of the cardiovascular risk shows that obese subjects present higher CVR (37.2%) compared to overweight (31.8%) and dependently of AFA, the result show that overweight/obese with AFA had highest CVR (38.9%) compared to those without AFA. These result demonstrates that the increase of the adiposity markers (BMI and WC) contribute both to cardiometabolic and cardiovascular health perturbations. Concerning the LDL particle size and other cardiometabolic disorders, our study demonstrated that in overweight/obese with AFA, LDL phenotype B (sdLDL) was associated with Low HDL-c, insulin-resistance and hyper triglyceridemia. These findings may be explained by the fact that body fataccumulation (especially abdominal accumulation fat) is at the center of many metabolic disorders including insulin resistance, hypertriglyceridemia and Low HDL. Those metabolic disorders can be related to the LDL particle size variation. The metabolic mechanisms which

can explain the link between body fat accumulation (especially abdominal accumulation) and metabolic disorders related to LDL particle size have previously been presented in several epidemiological studies elsewhere [47] [48] [49]. In the metabolic point of view, body fat accumulation (especially visceral accumulation fat) is likely to contribute to the release of uncontrolled fatty acid from adipose tissue, especially visceral adipose tissue, through lipolysis, which causes increased delivery of fatty acids to the liver and synthesis of very-low-density lipoprotein (VLDL). Increased levels of free fatty acids can decrease mRNA expression or activity of lipoprotein lipase (LPL) in adipose tissue and skeletal muscle, and increased synthesis of VLDL in the liver can inhibit lipolysis of chylomicrons, which promotes hypertriglyceridemia [50]. The increased triglyceride content in LDL is hydrolyzed by hepatic lipase (HL), leading to the formation of small-dense LDL particles [50]. On other hand, along with triglyceride synthesis in the liver, the increased delivery of free fatty acids to the liver exacerbates insulin resistance, which promotes dyslipidemia (hyper TG, low HDL-c) currently observed in the present study in overweight/obese subjects with AFA and associated to LDL phenotype B.

Concerning cardiovascular risk, our study shows that obese subjects will present higher CVR over ten years as compared to normal-weight according to Framingham score analysis (**Table 5** and **Table 6**). According to LDL particle size, the present study shows that the LDL phenotype A was two times more associated with low CVR in overweight/obese without AFA as compared to those with AFA; whereas its counterpart the LDL phenotype B was significantly associated to higher CVR in overweight/obese subjects. In fact, in obesity state, especially in abdominal obesity, there is an increase in sdLDL due to the large release of free fatty acid which is caused by deleterious effect of AFA on LDL particle size [39]. As a consequence, the sdLDL penetrates easily and gradually into the arterial wall, bind to LDL receptor because of its higher affinity and then develops the arthrosclerosis process, which will contribute to the installation of CVD [51]. However, a higher sdLDL-c level has been reported to be associated with an increased risk of atherosclerotic CVD [51] [52], besides the National Cholesterol Education Program (NCEP) has showed that a higher level of sdLDL-c is currently used as a risk factor for CVD and is considered to be a reliable tool of atherosclerosis predictor than LDL-c [5]. On the other hand, the association observed between LDL phenotype A and low CVR can explain the cardio-protective effect of LDL phenotype A among overweight and obese cameroonians. In fact LDL phenotype A has non-atherogenic properties which cannot allow it to initiate the atherosclerosis process.

5. Conclusion and Recommendations

BMI and WC clearly define adiposity in Cameroonian obese subjects. These adiposity markers are closely associated with small and dense LDL particles (LDL phenotype B) which are implicated in higher CVR among overweight and obese with abdominal fat accumulation. LDL particle phenotype B may be a

useful parameter for the assessment of CVR and help clinicians to diagnose high-risk CVD and prevent early mortality due to CVD in overweight and obese individuals living in these regions of Cameroon. These findings suggest that the measurement of sdLDL should be systematically incorporated in Cameroon's primary healthcare centers for the early detection of high cardiovascular risk in order to prevent high-risk CVD and reduce levels of premature death.

Limitation of the Study

Limitation includes self-reporting of data by participants which made cardiovascular risk assessment difficult.

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Consent for Publication

Not applicable.

Availability of Data and Materials

Data and materials used in this study are available on request.

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Authors' Contributions

JTN, JLN, and BGKA conceived and designed the study. JTN, FRN and GRTN collected data and performed laboratory tests. JTN, FRN and BGKA carried out the statistical analysis. JTN, FRN, GRTN, MF, ENL and JLN drafted and reciewed the manuscript. JEO and JLN supervised the study. All authors read and approved the final manuscript.

Conflicts of Interest

The authors declare that there are no conflicts of interest regarding the publication of this paper.

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Appendix: Questionnaire

NUTRITION AND HEALTH STUDY

CODE: ____ Phone…… Age:…….. Sex:……..

Participant's Name: …….

Place of birth: …….

Ethnic group: ……..

Region of Origin: ……

Division of Origin: ……

Profession: ……..

Religion: …….

Region or Place of Residence: ……..

Hormonal Status:

PARAMETERS OF INTEREST:

Weight (kg): ……. Height (m): …….

BMI (kg/m^2): …… Hip circumference (cm): …….

Waist circumference (cm): …….

CARDIOVASCULAR HISTORY

Do you have familly' history of cardiovascular risk factors (CRF)?

Yes (1) No (2)

Type of cardiovascular risk factors

Obésité (1)

Diabète (2)

Hypertension (3)

Others (4)

Precise it: …….

Member of familly suffering from CRF

Father (1)

Mother (2)

Other (3)

Precise……

- Previous treatment

Are you under medical treatment? Yes (1) No (2); If "yes" mention the type of treatment…….

Do you take nutritional complement? Yes (1) No (2)

Do you take traditional drug? Yes (1) No (2)

- Blood pressure

Systolic blood pressure (mm Hg): …….

Diastolic blood pressure (mm Hg): …….

Pool (bat/min): …….

- Biochemical parameters:

Fasting blood glucose: ……. (mg/dL);

Total cholesterol: ……. (mg/dL);

LDL-cholesterol: ……. (mg/dL)

HDL-cholesterol: ……. (mg/dL);
Triglycerides: ……. (mg/dL);
Small, dense LDL: ……. (mg/dL)
Insulin: ……. (µL/mL)
LDL particle phenpotype:
Phenotype A (1)
Phenotype I (2)
Phenotype B (3)

- Smooking habits

Are you a smoker? Yes (1) No (2)
Are you a past smoker? Yes (1) No (2)

- Conclusion and recommendations