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Analysis of Oxidized LDL as a Biomarker for the Early Diagnosis of Cardiovascular Diseases in High-Risk Subjects in Brazzaville (Republic of the Congo)

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Abstract

Cardiovascular diseases represent a real public health problem worldwide, and sub-Saharan Africa is experiencing an increasing prevalence of cases of diabetes, high blood pressure, obesity and dyslipidemia that need to be detected early for better management. Comparing the results of traditional monitoring with those of emerging biomarkers such as oxidized LDL could help to adjust surveillance measures, especially screening for these pathologies. Hence the interest of this study, which consisted in analyzing the expression of oxidized LDL within the population of Brazzaville. 533 patients were included in this study, distributed among 326 apparently healthy subjects (of whom 51 were diagnosed with high blood pressure, 25 with T2D and 4 with both), then 207 sick subjects presenting 123 cases of high blood pressure and 84 cases of T2D. Among these subjects who were already ill, we also detected comorbidities, which allowed us to group our study population into 10 groups. The classical lipid profile compared to the expression of oxidized LDL in the subjects tested showed no statistical difference between these groups for the classical lipid profile, whereas the difference was statistically proven for oxidized LDL values. Especially when they had other risk factors. Oxidized LDL appears to be more strongly associated low-grade vascular inflammation; during the formation of atherosclerotic plaques as well as during the classic lipid assessment. They need to be used in preventive monitoring reports on the advent of car-

diovascular pathologies.

Keywords

Oxidized LDL, Cardiovascular Diseases, Vascular Inflammation, Type 2 Diabetes

1. Introduction

Cardiovascular disease (CVD) is one of the major public health problems. They account for the major causes of morbidity and mortality worldwide and in sub-Saharan Africa, where traditional risk factors such as high blood pressure (HBP), diabetes, and dyslipidemia are on the rise among urban populations. There are about 19.8 to 20.5 million annual deaths, or nearly 32% of global deaths where more than 75% of these deaths occur in low- and middle-income countries [1].

In the Republic of the Congo, local epidemiology reveals a growing prevalence of these pathologies among subjects at intermediate risk, characterized by a high prevalence of arterial hypertension (about 15% in rural areas, and much higher in Brazzaville) and an increase in coronary heart disease. Blood pressure, diabetes and obesity are the dominant risk factors [2] [3]. The monitoring and screening of these CVDs in the Congo are carried out using common biomarkers such as transaminases, glycated hemoglobin, fasting blood glucose, standard lipid profiles (triglycerides, total cholesterol, HDL-C, LDL-C) … [4] [5]. Unfortunately, few studies address the issue of biomarkers for predicting the risks associated with these cardiovascular diseases.

Among these emerging biomarkers of cardiovascular risk, oxidized low-density lipoproteins (ox-LDL) have caught our attention, due to their involvement in the formation of atheroma plaques [6] [7]. They are central actors and initiators of atherosclerosis, transforming LDL cholesterol into a toxic inflammatory agent. They penetrate the arterial wall, trigger chronic inflammation, and are captured by macrophages to form foam cells, constituting the lipid core of plaque, promoting its progression and rupture. Vascular inflammation plays an important role in the pathophysiological progression of cardiovascular disease (CVD).

Thus, the analysis of this inflammatory process via ox-LDL could constitute an analytical strategy of interest in the early detection and management of these cardiovascular diseases. It is in this context that this study was conducted to assess the variations in plasma concentrations of ox-LDL within the population of Brazzaville exposed to risks of underlying vascular inflammation.

2. Generality

2.1. Involvement of Ox-LDL in the Pathophysiology of Atheromatous Plaques

The pathophysiology of atheromatous plaque is a chronic inflammatory process

of the arterial intima, starting with endothelial dysfunction often due to leakage of LDL-cholesterol at the subendothelial level, promoting infiltration of immune cells (foamy macrophages), followed by lipid accumulation, and the formation of a fibrous cap, which can lead to stenosis or thrombosis [8].

2.2. Mechanism of Formation (Atherogenesis) [9]

Plate formation evolves in several stages over decades:

- Endothelial dysfunction: due to risk factors such as smoking, blood pressure, diabetes, the inner wall of the artery; endothelium becomes permeable.
- Accumulation of lipids: LDL-cholesterol penetrates the intima and oxidizes.
- Inflammatory reaction: monocytes penetrate the wall, turn into macrophages and absorb ox-LDL, becoming "foam cells", because they are laden with fat.
- Formation of the fibrous cap: smooth muscle cells migrate to the area and produce collagen, creating a fibrous cap that covers the lipid core.
- Evolution and complications (stenosis): the plaque gradually grows, narrowing the lumen of the artery and limiting blood flow (angina pectoris, claudication).
- Rupture and Thrombosis: the fibrous screat can become thinner and ruptured, releasing the prothrombogenic lipid content. This triggers the rapid formation of a clot (thrombus), which is responsible for acute accidents such as myocardial infarction (MI) or stroke.

2.3. Locations and Risks of Atheroma

Atheroma mainly affects large and medium-sized arteries (aorta, coronary, carotid, lower limb arteries). Their weakening is favored by local inflammation.

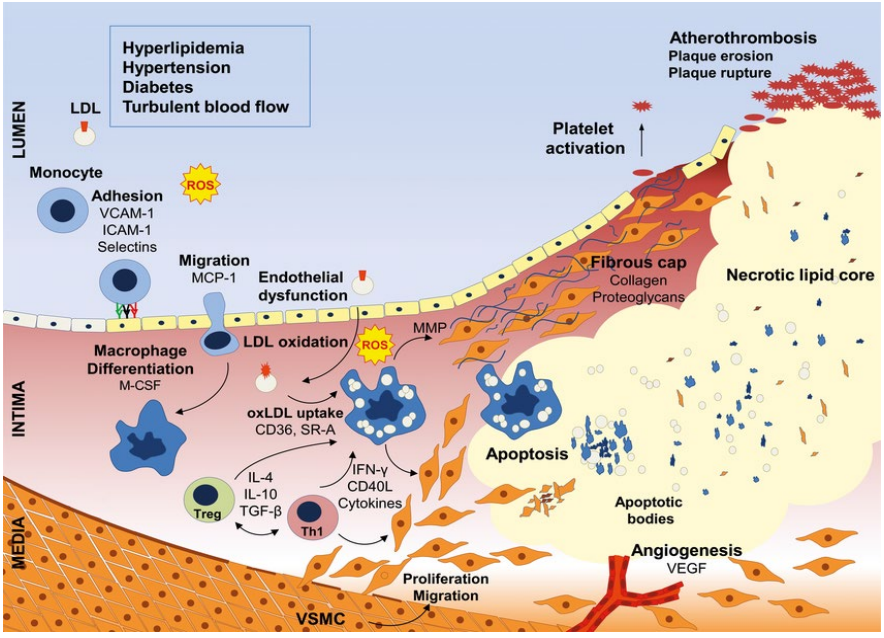

Figure 1. Schematic overview of key mechanisms of atherosclerosis in an evolving atherosclerotic plaque. Source [10].

Figure 1 illustrates the pathophysiological mechanism of atherosclerotic plaque formation, highlighting the involvement of LDL-cholesterol oxidation and other inflammatory markers.

Continuous exposure to risk factors causes endothelial dysfunction, leading to the retention of lipids in the intima, induction of endothelial expression of adhesion molecules, and secretion of chemotactic substances promoting leukocyte recruitment, adhesion, and transmigration into the vessel wall. Monocytes differentiate into macrophages and internalize modified lipoproteins, resulting in foam cell formation. Interaction of macrophages, T cells, and other leukocytes further contributes to the atherogenic process by releasing reactive oxygen species (ROS), growth factors, and cytokines, thereby perpetuating inflammation. VSMC dedifferentiate from a contractile to a proliferating phenotype, migrate from the media into the intima, and secrete extracellular matrix proteins, thereby forming the fibrous cap. Apoptosis of plaque resident cells leads to the formation of a necrotic core. Hypoxia-induced neovascularization and secretion of MMP by macrophages can destabilize the fibrous plaque, leading to plaque rupture. Exposure of plaque content to blood initiates platelet activation and aggregation, resulting in thrombus formation. ICAM-1, intracellular adhesion molecule 1; IFN-c, interferon-gamma; IL, interleukin; LDL, low-density lipoprotein; M-CSF, macrophage colony-stimulating factor; MCP-1, monocyte chemoattractant protein 1; MMP, matrix metalloproteinase; oxLDL, oxidized LDL; SR-A, scavenger receptor A; TGF-b, transforming growth factor beta; VCAM-1, vascular adhesion molecule 1; VEGF, vascular endothelial growth factor; VSMC, vascular smooth muscle cells.

3. Methodology

This was a cross-sectional analytical study conducted in March 2025 in the Department of Metabolic Diseases at the Brazzaville University Hospital Center (CHU-B) and at the TRIOS Foundation's Health Center; patient inclusion sites and biomedical analyses as well as in the Training, Research, and Biomedical Analysis Laboratory of the Faculty of Health Sciences of Brazzaville, Republic of the Congo for the determination of ox-LDL.

3.1. Epidemiological Investigation

Were eligible for the population of this study, patients having voluntarily agreed to participate, the inclusion criteria were as follows: to be a black African, residing in Brazzaville, at least 16 years old, without a history of liver disease and fever at baseline (temperature at 37˚C was required). The data were collected using a direct interview questionnaire from patients who had given their written informed consent with the mention "read and approved". This work has been ethically authorized by the Research Ethics Committee in Health Sciences (CERSSA) under number No. 092-24/MESRIT/DGRST/CERSSA/-24 of January 10, 2024.

3.2. Description of the Sampling Process

For sampling, we used a probabilistic method by systematic sampling. A screening campaign for type 2 diabetes and hypertension was conducted in a working-class neighborhood of Brazzaville for five (5) days. 617 Black subjects were enrolled and screened, allowing us to classify them into different groups based on biomedical and clinical results. Of these, 533 met our criteria, and the others were excluded from this study due to factors such as fever, liver disease, having other race than the black race, or being Black men who had lived in Africa for less than three months. Furthermore, patients undergoing statin treatment were not included in this study. The 533 subjects included in this study were distributed as shown in Figure 2.

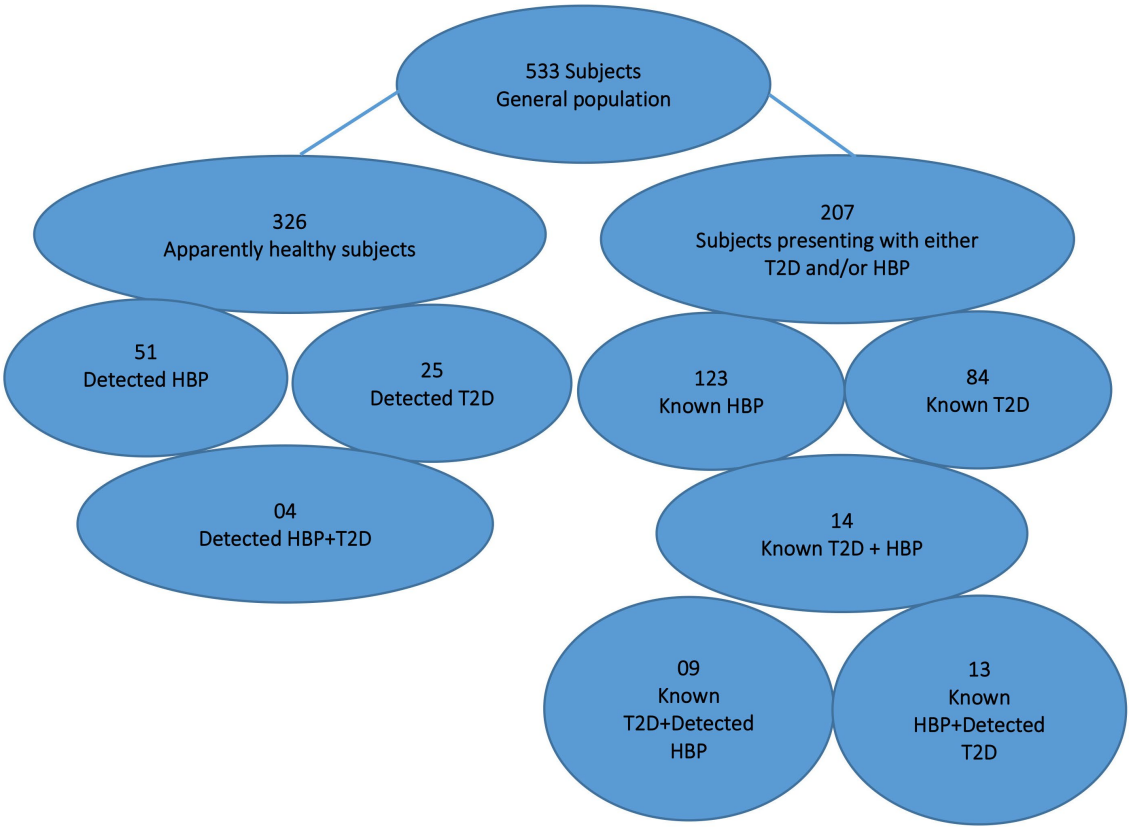

Figure 2. Distribution of the study's general population.

T2D and HBP were used separately as dependent variables in the logistic regression. Age integrated as a continuous variable in years, BMI (Body Mass Index) introduced as a continuous variable (value calculated in Kg/m^2). Smoking included as a categorical variable (non-smoker, former smoker, current smoker). Alcohol consumption: Taken into account via frequency categories (abstinent, occasional consumption, regular or risky consumption). Sedentary behavior treated as a continuous variable (hours per day spent sitting or in front of a screen).

3.3. Biological Investigation

The analytical conditions were met for the determination of our various biomarkers. For each patient fasting for at least 8 hours, 5 ml of blood was drawn via venipuncture at the elbow and collected respectively in dry tubes for the following parameters: creatinine, total cholesterol, triglycerides, HDL and LDL cholesterol, the tubes containing sodium fluoride for the determination of venous blood glucose and in the tubes containing Ethylene Diamine Tetra-Acetic (EDTA) for the determinations of glycated hemoglobin and ox-LDL.

The collected samples were centrifuged at 3000 revolutions per minute for 15 minutes to obtain serum (dry tube) and plasma (EDTA and sodium fluoride tube). Measurements of creatinine, total cholesterol, triglycerides, HDL cholesterol and LDL cholesterol were carried out using the Abbott ARCHITECT c4000 brand automatic analyzer which uses spectrophotometric methods (Beer-Lambert law). The determination of glycated hemoglobin was carried out on a Finecare brand immunochromatographic analyzer using the FIA (Fluorescence immunoassay) solid-base technique with whole blood collected in an EDTA tube and hemolyzed according to the manufacturer's recommendations according to the manufacturer's recommendations. A biomarker of subclinical inflammation, the high sensitive C-reactive Protein (hsCRP), was also measured using Finecare. The ox-LDL were made with plasma using the "sandwich" type enzymelinked immunosorbent assay (ELISA) and the optical densities were read by the automatic reader of the PHOmoLUmoAutobio ELISA plates. The PARS BIOCHEM kit (Human ox-LDL Elisa kit) was used for the ox-LDL assay technique. This kit allows for the measurement of human ox-LDL levels in plasma and other samples. It uses a purified human ox-LDL antibody that coats the wells of the microtiter plate. The HRP-labeled ox-LDL antibody forms an antibody-antigen-enzyme-antibody complex. After thorough washing, the TMB (3,3',5,5'-tétraméthylbenzidine) substrate solution is added to the complex, which turns blue. Once catalyzed by the HRP (Horseradish peroxidase) enzyme, the reaction is stopped by the addition of sulfuric acid, and the color change is measured using an optical reader at a wavelength of 450 nm. Ox-LDL concentrations are then determined based on their respective optical densities. The detection thresholds provided by the factory PARS BIOCHEM kit (Human ox-LDL Elisa kit) were 0.3 to 8 ng/ml. A dilution of 1/30th (*i.e.*, 1 volume of plasma to 29 volumes of distilled water) was recommended at the start of the assays of any sample, and a sample that showed a value higher than that of the standard was further diluted by half.

3.4. Statistical Analyses

Data were collected and analyzed using the software Epi info version 7.1.5 and the software Excel 2017 coupled with XLSTAT 2020. The Chi-square test was used for crossing qualitative variables. The analysis of variance was performed by the T-student test and the non-parametric ANOVA SUMMARY test or the Kruskal Wallis test. The collinearity curves were compared using the Pearson test (r). The

Wald test and the LR test were used for logistic regression. A value of $p < 0.05$ was considered significant.

4. Results

Table 1 presents the various average ages for the groups studied. Although the ages are unevenly distributed, the average age across all these groups falls within the 41 - 62 years, significant range bracket for studying cardiovasculars risk.

Table 1. Age averages of the population studied.

Population studied (number)	Average age ± standard deviation (in year)	Median	Minima	Maxima
Apparently healthy subjects (326)	**41 ± 18**	**39**	**16**	**88**
Detected HBP (51)	55 ± 9	58	18	74
Detected T2D (25)	41 ± 7	41	19	61
Detected HBP + T2D (04)	53 ± 5	54.5	41	61
Known subjects (T2D and/or HBP) (207)	**45 ± 10**	**46**	**16**	**80**
Known T2D (84)	57 ± 10	58	37	79
Known HBP (123)	52 ± 10	52	23	84
Known T2D + detected HPB (09)	61 ± 4	61	53	71
Known T2D + HBP (14)	56 ± 8	58.5	37	72
Known HBP + detected T2D (13)	62 ± 9	55	47	84

Note: HBP = High Blood Pressure, T2D = Type 2 diabetes.

Table 2. Average blood pressure and BMI.

Population studied	Vascular Risk Variables		
	BMI	Systolic	Diastolic
Apparently healthy subjects (326)	24 ± 10	120.5 ± 38.2	73 ± 24
Detected HBP (51)	26 ± 10.5	162 ± 61	89 ± 29
Detected T2D (25)	24 ± 9.2	127 ± 45	77 ± 26
Detected HBP+DT2 (04)	24 ± 8.2	159 ± 79	94 ± 47
Known subjects (T2D and/or HBP) (207)	25 ± 8	145 ± 35	80 ± 32
Known T2D (84)	27 ± 13	137 ± 67	78 ± 37
Known HBP (123)	26 ± 14	144 ± 90	83 ± 49
Known T2D + detected HBP (09)	26 ± 10	152 ± 76	77 ± 38
Known T2D + HBP (14)	26 ± 12	139 ± 68	78 ± 37
Known HBP + detected T2D (13)	27 ± 8.2	154.5 ± 58	83 ± 34
p-value (<0.05)	0.29	0.04	0.03

Body mass indices (BMI) showed no significant differences, indicating that the various groups had nearly identical body types. In contrast, blood pressure readings showed distinct average differences ranging from 120/70 mmHg to 160/90 mmHg, allowing for a clear separation between hypertensive individuals and the rest of the group.

Table 3. Averages of different biomarkers.

Biomarkers	Different groups studied (number)										p-value
	Apparently healthy subjects (326)	Detected HBP (51)	Detected T2D (25)	Detected HTA + T2D (04))	**Known subjects (T2D and/or HBP) (207)**	Known T2D (84)	Known HBP (123)	Known T2D + detected HBP (09)	Known T2D + HBP (14)	Known HBP + detected T2D (13)	
Glycaemia (g/l)	0.98 ± 0.5	1.07 ± 0.87	1.82 ± 1	1.79 ± 1	1.08 ± 0.9	1.77 ± 1.2	1.13 ± 1.10	1.79 ± 1.14	1.77 ± 1.15	1.58 ± 0.93	0.002
HbA1C (%)	6.0 ± 0.7	5.8 ± 2	7.5 ± 3	9.5 ± 4	9.0 ± 5	7.3 ± 2	6.5 ± 1.5	7.6 ± 2	7.0 ± 1.6	8.5 ± 2.5	0.003
TC (g/l)	1.75 ± 0.93	1.90 ± 0.96	1.89 ± 0.98	2.11 ± 1.2	1.84 ± 1.3	1.8 ± 0.94	1.82 ± 0.88	1.86 ± 0.91	1.73 ± 0.91	1.98 ± 0.94	0.06
TG (g/l)	0.99 ± 0.64	1.12 ± 0.67	1.16 ± 0.63	1.19 ± 0.6	1.06 ± 0.78	1.04 ± 0.6	1.06 ± 0.66	1.03 ± 0.52	1.01 ± 0.57	1.24 ± 0.63	0.07
HDL-C (g/l)	0.45 ± 0.3	0.42 ± 0.21	0.45 ± 0.22	0.45 ± 0.2	0.43 ± 0.4	0.42 ± 0.2	0.43 ± 0.23	0.43 ± 0.2	0.41 ± 0.19	0.45 ± 0.22	0.55
LDL-C(g/l)	1.10 ± 0.61	1.26 ± 0.69	1.21 ± 0.71	1.42 ± 0.8	1.14 ± 1.2	1.17 ± 0.6	1.18 ± 0.57	1.23 ± 0.61	1.12 ± 0.61	1.28 ± 0.63	0.13
PAI (TG/HDL-C)	2.2 ± 0.46	2.7 ± 0.24	2.58 ± 1.4	2.64 ± 2	2.46 ± 0.6	2.48 ± 1.7	2.47 ± 0.75	2.4 ± 0.14	2.46 ± 1.8	2.75 ± 0.25	0.56
Ox-LDL* (ng/ml)	**176 ± 74**	**179 ± 45**	**181 ± 71**	**221 ± 85**	**144 ± 88**	**124 ± 77**	**124 ± 75**	**140 ± 72**	**124 ± 78**	**161 ± 71**	**0.03**
HsCRP (mg/l)	0.44 ± 2.8	0.6 ± 1	0.59 ± 0.63	1.04 ± 0.6	4.5 ± 3	0.41 ± 0.5	0.64 ± 3.2	0.32 ± 0.26	0.42 ± 0.52	0.96 ± 0.56	0.005

*Usual values of ox-LDL: <100 low risk, 100-140 medium risk and ≥200 high risk. Source: https://www.optimaldx.com/research-blog/author/odx-research.
Notes: PAI: plasma atherogenicity index, TC: Total cholesterol, TG: Triglycérides, HDL-C: High Density Lipoprotein of cholesterol, LDL-C: Low Density Lipoprotein of cholesterol, HsCRP: high sensibility C-protein reactive and ox-LDL: oxidized Low Density Lipoprotein.

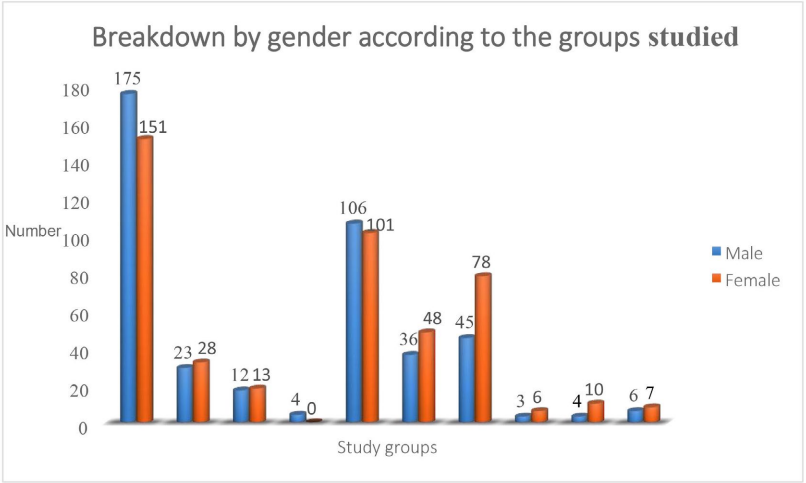

Figure 3. Gender distribution within the study population.

Sex ratio, shows us a slight predominance of the female gender at 51.3%.

Table 4. Univariate and multivariate logistic regression analysis of biomarkers associated with the early detection of high blood pressure (N = 533).

Biomarkers (Units)	univariate OR	CI à 95%	p-value	Adjusted OR (aOR)	CI à 95%	p-value
Glycaemia (g/l)	0.95	[0.62; 1.81]	0.070	2.15	[1.32; 3.69]	0.010
HbA1C (%)	0.74	[0.54; 1.76]	0.260	0.77	[0.55; 1.79]	0.410
TC (g/l)	1.05	[0.85; 1.20]	0.060	1.80	[1.01; 3.50]	0.020
TG (g/l)	1.08	[0.90; 2.50]	0.070	1.10	[0.90; 2.10]	0.060
6HDL-C (g/l)	0.54	[0.25; 0.75]	0.003	0.79	[0.74; 0.95]	0.003
LDL-C(g/l)	0.89	[0.78; 1.26]	0.090	0.95	[0.68; 1.06]	0.090
PAI (TG/HDL-C)	0.94	[0.60; 2.90]	0.080	1.00	[0.70; 1.25]	0.130
Ox-LDL* (ng/ml)	**2.90**	**[1.60; 5.40]**	**0.001**	**3.50**	**[2.60; 8.00]**	**0.002**
HsCRP (mg/l)	1.17	[0.70; 2.00]	0.140	1.70	[0.90; 2.40]	0.250

Note: OR = Odds Ratio; CI = Confidence Interval. The multivariate model simultaneously incorporates all the biomarkers listed above for mutual adjustment. The multivariate models were adjusted for age, gender, BMI, history of hypertension, history of dyslipidemia, history of diabetes mellitus, current smoking, current alcohol consumption, history of coronary heart disease (CHD), atrial fibrillation, pulmonary infection, intravenous thrombolysis treatment, previous ischemic stroke.

Univariate logistic regression analyses reveal that only HDL-cholesterol (OR = 0.54; 95%; CI [0.25; 0.75]; p = 0.003) and oxidized LDL (OR = 2.90; 95% CI [1.60; 5.40]; p = 0.001) are significantly associated with the risk of early-onset hypertension.

Following overall multivariate adjustment in a sample of 533 subjects, four biomarkers emerge as independent predictors of high blood pressure.

- Oxidized LDL remains the strongest risk factor, increasing the risk by a factor of 3.50 for each additional unit (aOR = 3.50; 95% CI [2.60; 8.00]; p = 0.002).
- A one-unit increase in fasting blood glucose more than doubles the risk of hypertension (aOR = 2.15; 95% CI [1.32; 3.69]; p = 0.010).
- Total cholesterol maintains an independent and significant deleterious association (adjusted OR = 1.80; 95% CI [1.01; 3.50]; p = 0.020).
- Conversely, HDL cholesterol confirms its independent protective role, significantly reducing the risk of developing hypertension by 21% (adjusted OR = 0.79; 95% CI [0.74; 0.95]; p = 0.003).
- Other clinical parameters evaluated (HbA1c, triglycerides, PAI, and hs-CRP) show no statistically significant association in the final adjusted model (p > 0.05).

Table 5. Average ox-LDL based on CVD risk factors.

Population studied	Risk Factor		
	Alcohol consumption	Tobacco consumption	Sedentary lifestyle
Apparently healthy subjects (326)	**154 ± 90**	**135 ± 65**	**149 ± 78**
Detected HBP (51)	**197 ± 80**	**168 ± 40**	**205 ± 94**
Detected T2D (25)	**173 ± 72**	**182 ± 47**	**247 ± 54**
Detected HBP+DT2 (04)	**191 ± 10**	-	**200 ± 5**

Continued

Known subjects (T2D and/or HBP) (207)	**165 ± 41**	**164 ± 19**	**153 ± 19**
Known T2D (84)	**176 ± 53**	**179 ± 18**	**155 ± 25**
Known HBP (123)	**152 ± 28**	**149 ± 22**	**150 ± 12**
Known T2D + detected HBP (09)	**173 ± 43**	-	**152 ± 14**
Known T2D + HBP (14)	**149 ± 12**	**133 ± 09**	**145 ± 16**
Known HBP + detected T2D (13)	**210 ± 35**	-	**187 ± 42**
p-value (<0.05)	**<0.001**	**<0.05**	**<0.05**

Table 5 summarizes the distribution of oxLDL levels according to these three risk factors; groups with a diagnosis of type 2 diabetes (T2D), high blood pressure (HBP), or both exhibit the highest values (T2D/HBP).

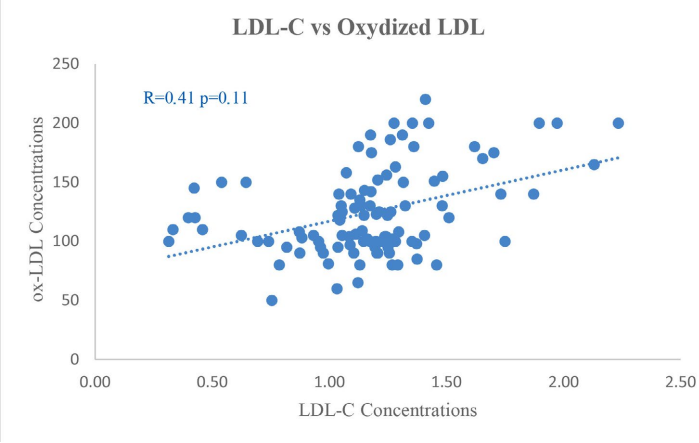

Figure 4. Low correlation between LDL-C and oxidized LDL.

Figure 4 shows a weak correlation between LDL-C and ox-LDL with r = 0.41 and p = 0.11 this lack of specificity, which appears to be a lipid paradox. However, this demonstrates that circulating LDL-C levels do not necessarily reflect the lipid status at the sub-endothelial level.

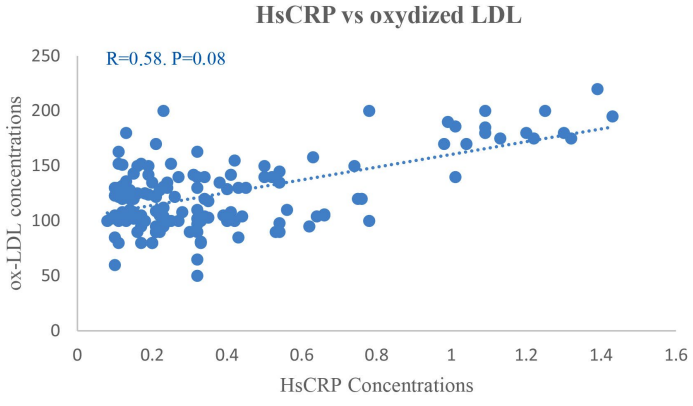

Figure 5. Average correlation between HsCRP and oxidized LDL.

There is a weak correlation between high-sensitivity CRP and ox-LDL (r = 0.58, p = 0.08), demonstrating the complexity of diagnosing low-grade inflammation.

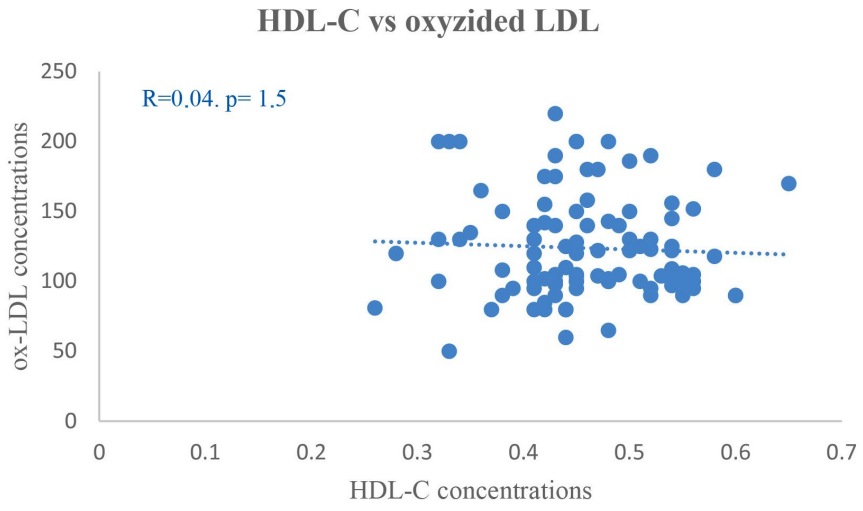

Figure 6. No correlation between HDL and oxidized LDL.

No correlation was found between HDL-C and ox-LDL. Les valeurs de r = 0.04; p = 1.5 montre le caractère antagoniste entre ces deux biomarqueurs l'un est pro-athérogénique et pro-inflammatoire (ox-LDL) et l'autre est anti-inflammatoire (HDL-C).

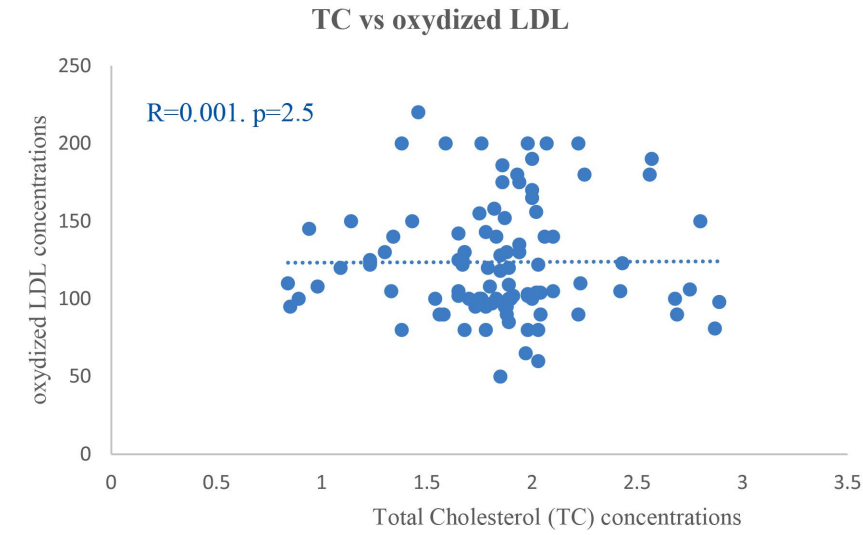

Figure 7. No correlation between total cholesterol and oxidized LDL.

There is no correlation between total cholesterol and ox-LDL, reflecting the independent nature of ox-LDL and its independent involvement in low-grade inflammation.

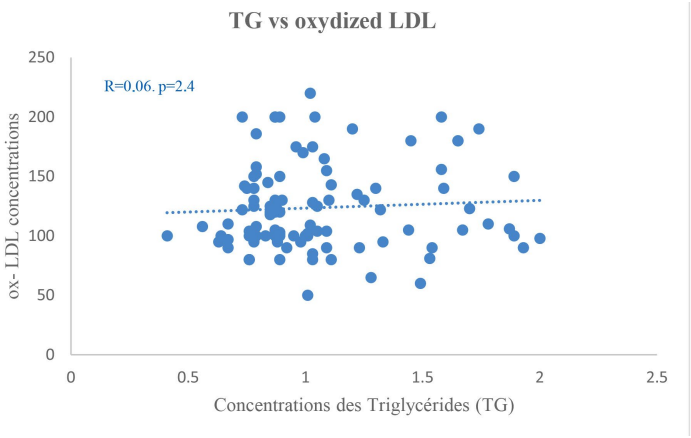

Figure 8. No correlation between triglycerides and oxidized LDL.

There is no correlation between Triglycerides and ox-LDL, reflecting the independent nature of ox-LDL and its independent involvement in low-grade inflammation.

5. Discussion

5.1. Limitations of This Study

The non-prospective nature, potential effect of age heterogeneity and unequal gender distribution among subgroups were the limitations of this study. Furthermore, failing to conduct an in-depth analysis of the study population's dietary habits could introduce bias, given that in an African context, the population frequently consumes various spices with antioxidant properties.

Nevertheless, this study, among the first in our country to analyze oxidized LDL in the preventive monitoring of cardiovascular diseases, has opened up a perspective for reorganizing the choice of biomarkers for screening, diagnosis or monitoring of cardiovascular diseases among the black population of the Congo Basin.

5.2. Analysis of the Results Obtained

The distribution of ages within each group, shown in **Table 1**, shows us, in general, a well-represented population with ages ranging from 16 to 88 years. These extreme ages allowed us to have a fairly broad overview for the detection of cardiovascular diseases. Of course, the literature tells us which age groups are likely to be vulnerable to these diseases at least 35 years old [11] [12]. But, it is not uncommon to see the least aged than those being victims of these pathologies, especially with the influence of bad hygienic-dietary behaviors such as tobacco consumption, alcohol and other narcotics, sedentary lifestyle without forgetting stress [13] [14]. Especially in the context of screening for cardiovascular diseases in the aftermath of the COVID-19 pandemic, it was necessary to look at this age interval for underlying predispositions to these pathologies.

Figure 3, which divides the sex ratio, shows us a slight predominance of the female gender at 51.3%.

Table 2 did not show a significant difference in the Body Mass Index. This can justify the well-mixed character of the study population, but blood pressure was significantly different within these different groups, thus justifying the presence or absence of high blood pressure.

Table 3 summarises the mean values of the various biomarkers measured in this study, where we observe a significant difference in blood glucose levels between the different groups studied, which enabled us to clearly classify these patients into groups. However, the most striking finding is the lack of a significant difference in the biomarkers of the standard lipid profile within these different groups, whilst we observe a clear and significant difference in oxidized LDL and hsCRP. Furthermore, the groups with at least one of the conditions (T2D or HBP) detected, presented us with concentrations of ox-LDL and hsCRP at risk. Given that screening is carried out at an asymptomatic stage, we can state that ox-LDL and low concentrations of hsCRP can be considered biomarkers of an underlying manifestation of cardiovascular disease. This is also reported by Markus *et al.*, in 2025 [15].

Table 4, summarizes a univariate and multivariate analysis of the various cardiovascular risk biomarkers, and this logistic regression, show us a clear implication of ox-LDL, hsCRP and PAI in the onset of cardiovascular diseases, as also reported by Coulibaly Djibril Mamadou *et al.*, in relation to ox-LDL [16].

Table 5, highlights high-risk levels of ox-LDL in subjects, particularly those diagnosed with T2D or HBP, who did not adhere to a strict healthly lifestyle. This may be explained by low-grade inflammation that had likely recently been triggered. This argument is also described by A. Sarr *et al.*, in Senegal on 2012 [17], also by Oumar Sangho *et al.*, in Mali on 2024 [18].

Figures 4-8 illustrate the collinearity between the various biomarkers studied, and we note here that ox-LDL shows no correlation with the biomarkers of the standard lipid profiler apart from a weak correlation with LDL-cholesterol, which may be explained by the fact that ox-LDLs are indeed LDL cholesterol particles, but their infiltration at the subendothelial level does not necessarily depend on hyperactivity of LDL-cholesterol at the peripheral level. This is what distinguishes ox-LDL from the biomarkers of the standard lipid profile. This means that the standard or conventional lipid profile may appear normal, whilst, underlying this, these particles are already present, triggering low-grade inflammatory signaling which is the starting point for vascular damage and the formation of atherosclerotic plaques. A moderate correlation was nevertheless observed between oxidized LDL and hsCRP, which could be evidence of low-grade inflammation. This indicates that oxidized LDL resulting from oxidation that causes oxidative stress would be a better marker than standard lipid biomarkers. Our results highlight the critical and predominant role of lipid oxidative stress (oxidized LDL) and carbohydrate metabolism (fasting blood glucose), while HDL cholesterol confirms its protective function. The strongest association revealed by our multivariate model involves oxidized LDL, with a 3.50-fold increased risk (aOR = 3.50; p =

0.002). Physiologically, oxidized LDL is not merely a passive marker of the lipid profile but a direct driver of endothelial dysfunction. It penetrates the arterial intima, activates leukocyte adhesion molecules, and reduces the bioavailability of nitric oxide (NO), a potent vasodilator. This early arterial stiffening explains why this biomarker outperforms standard lipid fractions (such as native LDL-cholesterol) in the early detection of HBP. Louis Raymond Nguele *et al.*, they worked in Cameroon in 2016, highlighting the influence of oxidative stress on lipid metabolism [19].

One of the most notable findings of our study is that fasting blood glucose (aOR = 2.15) and total cholesterol (aOR = 1.80) become statistically significant only after multivariate adjustment certainly caused by the masking effect (or suppression effect).

Regarding fasting blood glucose: in univariate analysis, the specific effect of blood glucose on blood pressure is masked by the high variance of other collinear confounding factors (such as lipid profile or hs-CRP). Once the model adjusts for and stabilizes variations in HDL cholesterol and oxidized LDL, the direct relationship between blood glucose levels and HBP is revealed. Clinically, chronic hyperglycemia even at subclinical stages (prediabetes) stimulates the renin-angiotensin-aldosterone system (RAAS) and increases tubular sodium reabsorption, thereby directly raising blood pressure.

Regarding total cholesterol: it encompasses both atherogenic (LDL) and protective (HDL) fractions. In univariate analysis, these opposing effects cancel each other out, rendering the result non-significant ($p = 0.060$). Once the model isolates the protective effect of HDL cholesterol, the remainder of the total cholesterol exerts its full deleterious impact on arterial compliance (adjusted OR = 1.80; $p = 0.020$).

Conversely, HDL cholesterol retains a robust and independent protective role in the adjusted model (aOR = 0.79; $p = 0.003$). Beyond reverse cholesterol transport, HDL possesses direct antioxidant properties capable of specifically inhibiting the oxidation of LDL particles. This biological synergy supports the consistency of our multivariate model (**Table 4** and **Figure 6**). In Gabon, a similar study was conducted by F.O. Abessolo *et al.*, correlating LDL oxidation time with Paraoxonase-1 (PON-1) expression. Study that also addresses the influence of oxidative stress on lipid metabolism [20].

The lack of a linear correlation between total LDL cholesterol and oxidized LDL (ox-LDL) concentrations presents a paradox that warrants analysis. Specifically, a normal or low blood LDL-C level does not guarantee a low level of oxidized LDL, and vice versa. This is because LDL-C is a quantitative marker measuring the amount of circulating cholesterol, whereas ox-LDL reflects the extent of oxidative stress—particularly relevant in the context of developing nations—and systemic inflammation.

LDL itself is not toxic; it becomes truly atherogenic—capable of infiltrating the arterial wall—only after undergoing an oxidation cascade triggered by free radicals. Ox-LDL levels depend on the overall balance between pro-oxidant factors

(such as smoking, inflammation, hyperglycemia, and a diet high in saturated fats) and the body's antioxidant defense system. Consequently, the total amount of LDL does not determine the proportion that becomes oxidized, as reported by Rabizadeh *et al.* (2023) [21]. This study demonstrates that simply measuring standard LDL cholesterol is insufficient and may mask the true risk, as oxidative stress renders LDL particles more vulnerable by attacking their unsaturated fatty acids. This oxidation transforms normal LDL into a harmful particle that infiltrates and accumulates in arterial walls, initiating the development of atherosclerotic plaques.

Ox-LDL particles are therefore direct initiators of atheroma plaque formation and the degradation of blood vessel walls. Measuring LDL-C provides no information regarding the potential danger posed by the particles themselves. An individual with low LDL-C but high ox-LDL levels faces significant underlying cardiovascular risk, as their LDL particles are more aggressive. Certain standard treatments, such as statins, significantly reduce LDL-C levels but do not always have a direct, proportional impact on lowering ox-LDL. Clinical management is therefore shifting toward a holistic approach to protecting cardiovascular health: on the one hand, reducing the number of target particles (lowering LDL); on the other, combating oxidative stress (through antioxidant intake, physical activity, and lifestyle choices).

6. Conclusions

This study, which compared the levels of conventional lipid biomarkers (total cholesterol, triglycerides, HDL-cholesterol and LDL-cholesterol) with those of oxidized LDL in the prediction of cardiovascular disease (High Blood pressure), has shown us a more active expression of the latter biomarker, particularly in subjects who have been diagnosed with at least one cardiovascular condition or type 2 diabetes. This indicates activation of this biomarker during low-grade inflammation, which is a key trigger for vascular diseases. Oxidized LDL might therefore be useful in preventive screening programmes for the onset of cardiovascular diseases.

In conclusion, early detection of high blood pressure becomes more accurate when fasting blood glucose monitoring is combined with oxidative stress markers such as oxidized LDL. These findings support a comprehensive metabolic management approach rather than the isolated analysis of traditional risk factors.

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Author Contributions

Conceptualization, R.I.O. and F.O.K.; methodology, R.I.O.; software, R.I.O.; J.B.P.;

and J.F.; validation, EM, B.L-M., and HGM.; formal analysis, R.I.O. and N.A.; investigation, R.I.O.; B.T.N and F.E.B.G.; resources, R.I.O.; F-O.K.; N.A.; data curation, R.I.O.; writing—original draft preparation, R.I.O.; writing—review and editing, R.I.O.; visualization, E.M. and H.G.M.; supervision, E.M. and H.G.M.; project administration, R.I.O.; funding acquisition, Nil. All authors have read and agreed to the published version of the manuscript.

Conflicts of Interest

The authors have reported no conflicts of interest.

References

[1] World Health Organization (WHO) (2026) Maladies Cardiovasculaires. https://www.who.int/fr/health-topics/cardiovascular-diseases

[2] Ossou-Nguiet, P.M., Doungou, N.D., Mpandzou, G.A., *et al.* (2019) Troubles cognitifs et diabète de type 2 au Congo. *Asian Journal of Neurosurgery*, **38**, 1992-2647. https://ajns.paans.org/troubles-cognitifs-et-diabete-de-type-2-au-congo/

[3] Ikama, S.M., Nsitou, B.M., Bouénizabila, E. and Monabéka, H.G. (2014) Prévalence des lésions d'athérosclérose chez les patients diabétiques à Brazzaville, Congo. *Journal des Maladies Vasculaires*, **39**, 443-444. https://www.researchgate.net/publication/265295828

[4] Monabéka, H.G., Kimbally-Kaky, G., Gombet, T., *et al.* (2012) Syndrome métabolique et prévalence des cardiopathies ischémiques au CHU de Brazzaville, Congo. *Médecine des Maladies Métaboliques*, **6**, 75-79. https://doi.org/10.1016/s1957-2557(12)70365-6

[5] Eboka-Loumingou Sakou, R.F., Longo-Mbenza, B., Nkalla-Lambi, M., Mokondjimobe, E., Monabeka, H.G., Moukassa, D., *et al.* (2021) Inflammatory Biomarkers and Prediction of Insulin Resistance in Congolese Adults. *Heliyon*, **7**, e06139. https://doi.org/10.1016/j.heliyon.2021.e06139

[6] Gao, S., Zhao, D., Wang, M., Zhao, F., Han, X., Qi, Y., *et al.* (2017) Association between Circulating Oxidized LDL and Atherosclerotic Cardiovascular Disease: A Meta-Analysis of Observational Studies. *Canadian Journal of Cardiology*, **33**, 1624-1632. https://doi.org/10.1016/j.cjca.2017.07.015

[7] Bonnefont-Rousselot, D. (2020) Les marqueurs de l'oxydation des lipides. *Revue Francophone des Laboratoires*, **2020**, 47-55. https://doi.org/10.1016/s1773-035x(20)30162-3

[8] Azouaou, L., Ballouti, W., Arab, M., Benyahia, A. and Rayane, T. (2018) Le LDLox serait-il un facteur de risque majeur des complications cardiovasculaires chez l'IRC? *Néphrologie & Thérapeutique*, **14**, 337-338. https://doi.org/10.1016/j.nephro.2018.07.197

[9] Tedgui, A. (2022) Actualités dans la physiopathologie de l'athérosclérose. *Revue Francophone des Laboratoires*, **2022**, 26-34. https://doi.org/10.1016/s1773-035x(22)00212-x

[10] Wilck, N. and Ludwig, A. (2014) Targeting the Ubiquitin-Proteasome System in Atherosclerosis: Status Quo, Challenges, and Perspectives. *Antioxidants & Redox Signaling*, **21**, 2344-2363. https://doi.org/10.1089/ars.2013.5805

[11] Kekäläinen, T., Ahola, J., Reinilä, E., Savikangas, T., *et al.* (2025) Associations cumulatives entre les comportements liés à la santé, le bien-être mental et la santé sur 30 ans. *Annals of Medicine*, **57**, Article 2479233.

[12] Bonaldi, C., Grave, C., Serra, D., Rogel, A., *et al.* (2026) Les décès attribuables au tabagisme. Mise à jour des estimations pour l'année 2023. Édition national publiée le 16 février 2026. Santé Publique France. https://bdoc.ofdt.fr/index.php?lvl=notice_display&id=88869

[13] Produits du tabac, produits connexes, arômes pour cigarettes: Les mineurs sont de plus en plus exposés au risque d'intoxication. Vigil Anses du 21 novembre 2023. Site internet Agence nationale de sécurité de l'alimentation, de l'environnement et du travail. Maisons-Alfort (France). https://vigilanses.anses.fr/sites/default/files/VigilAnses_N21_article_tabac.pdf

[14] Portail national d'information pour les personnes âgées et leurs proches. Il n'y a pas d'âge pour arrêter de fumer. Site internet: Pour-les-personnes-agees.gouv. https://www.pour-les-personnes-agees.gouv.fr/preserver-son-autonomie/preserver-son-autonomie-et-sa-sante/tabagisme-des-personnes-agees-il-n-y-a-pas-d-age-pour-arreter-de-fumer

[15] Markus, M.R.P., Ittermann, T., Mariño Coronado, J., Schipf, S., Bahls, M., Könemann, S., *et al.* (2025) Low-Density Lipoprotein Cholesterol, Lipoprotein(a) and High-Sensitivity C-Reactive Protein Are Independent Predictors of Cardiovascular Events. *European Heart Journal*, **46**, 3863-3874. https://doi.org/10.1093/eurheartj/ehaf281

[16] Mamadou, C.D., Ibrahim, D.B.S., Adama, K., *et al.* (2024) Évaluation du Stress Oxydatif par le Dosage Plasmatique des LDL Oxydées chez les Diabétiques de Type 2 au Mali. *Health Research in Africa*, **2**, 110-115. https://ams-hra.org/index.php/hra/article/view/5663/4324

[17] Sarr, A., Lopez-Sall, P., Ndour-Mbaye, N.M., *et al.* (2012) Fréquence du syndrome plurimétabolique et des anomalies associées au sein d'une population noire au Sénégal. *Médecine des Maladies Métaboliques*, **6**, 238-243. https://doi.org/10.1016/s1957-2557(12)70405-4

[18] Sangho, O., Camara, D., Togo, A., *et al.* (2024) Clinical and Epidemiological Aspects of the Lipid Profile in Type 2 Diabetic Patients in Commune I of Bamako, Mali. *Mali Sante Publique*, **14**, 9-15. https://doi.org/10.53318/msp.v14i1.3001

[19] Nguele, L.R., Fokunang, C.N., Etoundie, C., Chakokan, R.M., Ngondi, J.L., Tembe, E.A., *et al.* (2017) Utilisation des espèces du genre Aframomum (Aframomum Aulacocorpus, A. citratum, A. daniellii) pour le contrôle du poids, le profil lipidique et le statut antioxydant chez les rats Wistar nourris avec une diète athérogène. *International Journal of Biological and Chemical Sciences*, **10**, Article 2575. https://doi.org/10.4314/ijbcs.v10i6.14

[20] Abessolo, F., Bekale, S., Jose, F., Moutsinga, Y. and Milama, E. (2012) Profil lipidique, cinétique d'apparition des diènes conjugués et activité paraoxonase-1 chez le diabétique de type 2 à Libreville au Gabon. *International Journal of Biological and Chemical Sciences*, **6**, 572-581. https://doi.org/10.4314/ijbcs.v6i2.2

[21] Rabizadeh, S., Seyedi, S.A., Nabipoorashrafi, S.A., Omidvar Siahkalmahalleh, M., Yadegar, A., Mohammadi, F., *et al.* (2023) The Lack of Association between Different LDL-C Levels and Oxidized LDL in Patients with Type 2 Diabetes. *Chronic Diseases and Translational Medicine*, **9**, 329-335. https://doi.org/10.1002/cdt3.84