ISSN No:-2456-2165 https://doi.org/10.38124/ijisrt/26may2007

High Concentrations of Phospholipase-A2-Associated Lipoproteins in Subjects Screened for Type 2 Diabetes and/or Hypertension in Brazzaville, Republic of Congo

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Publication Date: 2026/06/09

Abstract: Cardiovascular diseases represent a significant public health problem worldwide, and sub-Saharan Africa is experiencing increasing prevalence of diabetes and hypertension, necessitating early diagnosis for improved management. Comparing traditional monitoring results with those of emerging biomarkers such as Lp-PLA2 could help refine surveillance measures, particularly for screening for these conditions. This explains the importance of this study, which analysed Lp-PLA2 expression in the population of Brazzaville. 533 patients were included in this study, divided into 326 apparently healthy subjects (51 of whom were diagnosed with hypertension, 25 with diabetes, and 4 with both hypertension and diabetes) and 207 subjects with pre-existing conditions (123 with hypertension and 84 with diabetes). Among these subjects with pre-existing conditions, we also screened for comorbidities, which allowed us to group our study population into 10 groups. The standard lipid profile compared to Lp-PLA2 levels in screened subjects showed no statistically significant difference between these groups for the standard lipid profile, while a statistically significant difference was found for Lp-PLA2 values, especially when these subjects had other risk factors. Lp-PLA2 appears to be better expressed during low-grade vascular inflammation, specifically during the formation of atherosclerotic plaques, than in conventional lipid profiles. Its use in preventive monitoring of the onset of cardiovascular disease is warranted.

Keywords: *Lp-PLA2, Cardiovascular Diseases, Vascular Inflammation, Type 2 Diabetes and Hypertension.*

How to Cite: Rod Ibara Okabande; Fylla-Onanga Koumou; Nestor Andzouana; Ferdinand Brel Emaniel Got; Berge Tsiba Ngokana; Junior Batsi Poati; Judicael Foundikila; Etienne Mokondjimobe; Benjamin Longo-Mbendza; Henri Germain Monabeka (2026) High Concentrations of Phospholipase-A2-Associated Lipoproteins in Subjects Screened for Type 2 Diabetes and/or Hypertension in Brazzaville, Republic of Congo. *International Journal of Innovative Science and Research Technology*, 11(5), 3759-3767. https://doi.org/10.38124/ijisrt/26may2007

I. INTRODUCTION

Preventive medicine is widely considered a better approach to addressing the public and individual health needs of a population. By acting before the onset of disease (primary prevention) or through early detection (secondary prevention), it constitutes an essential tool for monitoring quality of life. Cardiovascular diseases, primarily hypertension and diabetes, which is more deadly due to cardiovascular complications, are major causes of morbidity

ISSN No:-2456-2165 https://doi.org/10.38124/ijisrt/26may2007

and mortality worldwide. They account for approximately 19.8 to 20.5 million deaths annually, representing nearly 32% of global deaths, with over 75% of these deaths occurring in low- and middle-income countries [1].

In the Republic of Congo, local epidemiology reveals a growing prevalence of these diseases among individuals at intermediate risk, characterized by a high prevalence of hypertension (approximately 15% in rural areas, and significantly higher in Brazzaville) and an increase in coronary heart disease. Hypertension, diabetes, and obesity are the dominant risk factors. [2, 3].

Monitoring and screening for these MCAs are ensured with common biomarkers such as glycated haemoglobin, fasting blood glucose, standard lipid profile (triglycerides, total cholesterol, HDL-C, LDL-C) … [4, 5]. Unfortunately, few studies address the issue of biomarkers for predicting risks related to these cardiovascular diseases.

When discussing these emerging biomarkers for predicting cardiovascular diseases, Phospholipase-A2-associated lipoproteins (Lp-PLA2) have caught our attention. They are involved in the formation of atherosclerotic plaques. [6-7]. Lp-PLA2 circulates in the blood as a lipoprotein complex. Due to their activity on platelet-activating factor (PAF), they can hydrolysed oxidized phospholipids from lipids, primarily oxidized phosphatidylcholines, generating pro-inflammatory mediators: lysophosphatidylcholines.

(lyso-PC) and oxidized fatty acids. A vicious cycle is established because the selection of inflammatory cells within atherosclerotic plaques leads to further production of Lp-PLA2 by activated macrophages and foam cells. Oxidized LDL, as well as oxidized fatty acids and lyso-PCs generated by the action of Lp-PLA2, indicate vascular inflammation; a crucial step in the development of atherosclerotic plaques [8-11].

Vascular inflammation has a very important role in the pathophysiology progression of cardiovascular diseases (CVDs). Therefore, analysing this inflammatory process via Lp-PLA2 could be a valuable analytical strategy for the early diagnosis of these diseases [12-14]. It is in this context that this study was carried out to evaluate the variations of Lp-PLA2 within an at-risk population in Brazzaville.

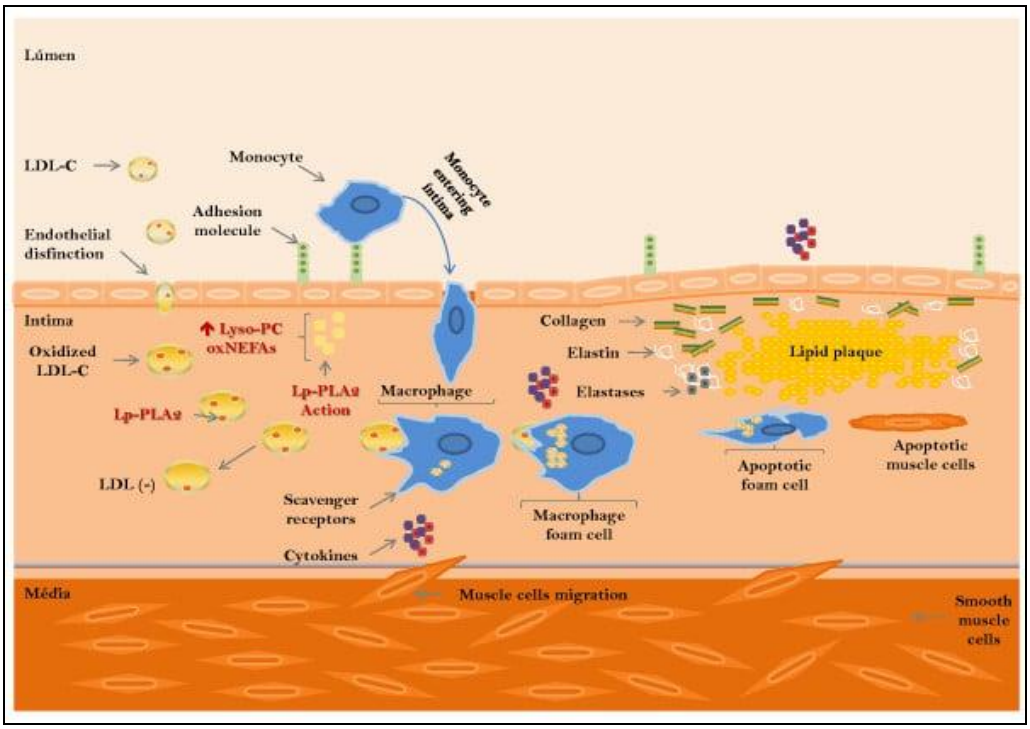

Fig 1 Possible Actions of Lp-PLA2 in the Atherosclerotic Process.
Source: [15]

II. METHODOLOGY

This was a cross-sectional analytical study, conducted in March 2025, in the metabolic diseases department of the University Hospital Center of Brazzaville (CHU-B) and at the TRIOS Foundation Health Center; locations for patient inclusion and biomedical analyses, as well as in the Training, Research and Biomedical Analysis Laboratory of the Faculty of Health Sciences of Brazzaville, Republic of Congo for the Lp-PLA2 assay.

➢ *Epidemiological Survey*

Patients who provided written consent were included in the study population. Inclusion criteria were as follows: being Black Congolese, at least 35 years old, with no history of liver disease and no fever at inclusion (temperature of 37°C was required).

ISSN No:-2456-2165 https://doi.org/10.38124/ijisrt/26may2007

Data were collected using a questionnaire via direct interviews with patients. 533 subjects, whose data were collected randomly, were selected, and the distribution after screening from the general population was as follows:

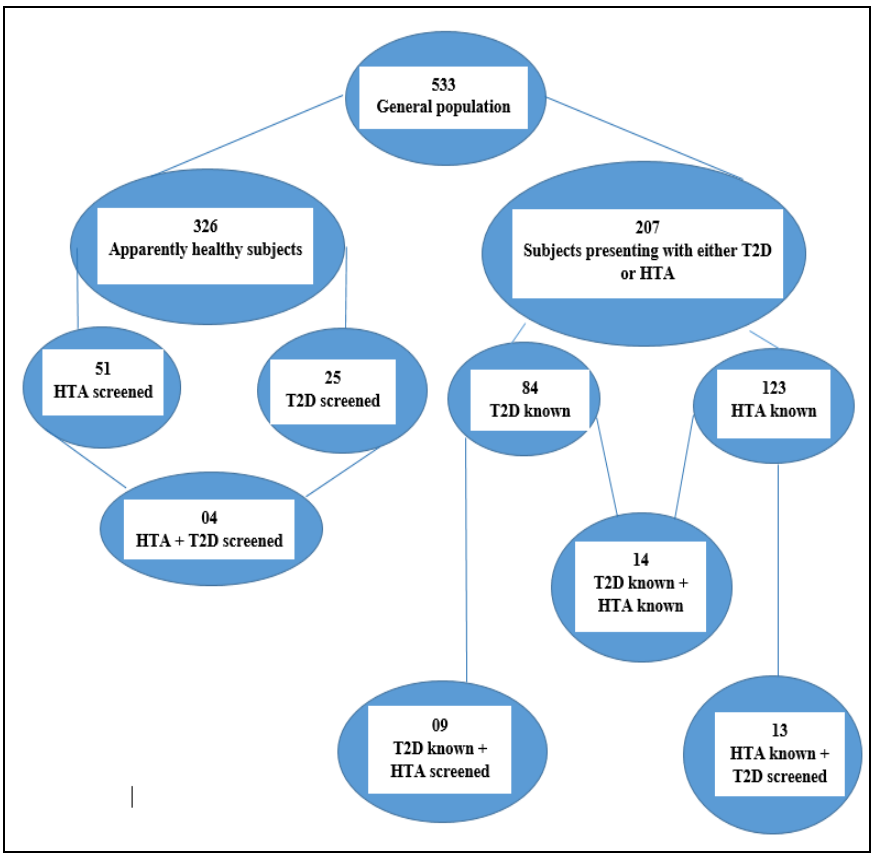

Fig 2 Distribution of the General Population of the Study

➢ *Biological Investigation*

The analytical conditions were respected for the measurement of our different biomarkers. For each patient who had been fasting for at least 8 hours, 5 ml of blood was taken by venipuncture at the bend of the elbow and collected respectively in dry tubes for the measurement of the following parameters: creatinine, total cholesterol, triglycerides, HDL and LDL cholesterol, in tubes containing sodium fluoride for the measurement of venous blood glucose and in tubes containing Ethylene Diamine Tetraacetic Acid (EDTA) for the measurements of glycated hemoglobin and Lp-PLA2.

The collected samples were centrifuged at 3000 rpm for 15 minutes to obtain serum (dry tube) and plasma (EDTA and sodium fluoride tube). Creatinine, total cholesterol, triglycerides, HDL cholesterol, and LDL cholesterol levels were measured using an Abbott ARCHITECT c4000 automated analyzer, which employs photometric methods (Beer-Lambert law).

Glycated hemoglobin (HbA1c) levels were measured using a Finecare immunochromatographic analyzer with solid-based fluorescence immunoassay (FIA) on whole blood collected in an EDTA tube and hemolyzed according to the manufacturer's instructions. Lp-PLA2 levels were measured using plasma with a sandwich enzyme-linked immunosorbent assay (ELISA), and optical density values were read using a PHOmoLUmoAutobio automated ELISA plate reader. The PARS BIOCHEM Human Lp-PLA2 ELISA kit was used for the Lp-PLA2 assay.

➢ *Statistical Analyzes*

Data were collected and analyzed using Epi Info version 7.1.5 and Excel 2017 coupled with XLSTAT 2020. The chi-square test was used for crossover of categorical variables. Analysis of variance was performed using the Student's t-test and the non-parametric ANOVA summary test or the Kruskal-Wallis test. Collinearity was compared using Pearson's correlation coefficient (r). A p-value < 0.05 was considered statistically significant. Wald's test and the LR test were used for logistic regression.

III. RESULTS AND COMMENTARY

➢ *Analysis of the Results Obtained*

The age distribution within each group, shown in Table I, generally indicates a well-represented population with ages ranging from 16 to 88 years. This age range allowed us to gain a fairly broad perspective for the detection of cardiovascular diseases. Indeed, the literature identifies age groups likely to be vulnerable to these diseases, at least from age 35 [16, 17]. However, it is not uncommon to see younger people suffering from these conditions, especially due to poor lifestyle habits such as tobacco, alcohol and other drug use, a sedentary lifestyle, and stress [18, 19]. Especially in

ISSN No:-2456-2165 https://doi.org/10.38124/ijisrt/26may2007

the context of cardiovascular disease screening following the COVID-19 pandemic, it was necessary to investigate underlying predispositions to these pathologies in this age group.

Figure 3, showing the sex ratio, reveals a slight predominance of females at 51.3%.

Table II did not show a significant difference in Body Mass Index. This may explain the mixed nature of the study population, but blood pressure was significantly different within these different groups, thus justifying the presence or absence of the pathology of arterial hypertension.

Table III, which summarizes the average values of the different biomarkers measured in this study, shows a significant difference in blood glucose levels between the different study groups, allowing us to clearly classify these patients. However, the most remarkable finding is the lack of significant difference in biomarkers from the standard lipid profile within these groups, while we observed a clear and significant difference in Lp-PLA2 and hs-CRP. Furthermore, the groups with at least one of the pathologies (T2D or hypertension) screened for presented with high-risk Lp-PLA2 and hs-CRP concentrations. Given that the screening was performed at an asymptomatic time, we can affirm that Lp-PLA2 and low hs-CRP concentrations can be considered biomarkers of an underlying manifestation of cardiovascular disease. This is also reported by François Paillard in 2024 [20].

Tables IV and V, summarizing a univariate and multivariate analysis of the different cardiovascular risk biomarkers and this logistic regression, show us the clear involvement of Lp-PLA2, hs-CRP and IAP in the advent of cardiovascular pathologies, as also reported by other authors [21-23].

Table VI highlights high-risk Lp-PLA2 values in subjects, particularly those screened for type 2 diabetes or hypertension, who did not maintain a strict lifestyle. This may be explained by low-grade inflammation that had likely just been detected. We can thus observe that poor lifestyle habits can trigger underlying inflammatory reactions, which can be key factors in vascular damage.

Figures 4, 5, 6, 7, and 8 illustrate the collinearity between different biomarkers studied. We observe that Lp-PLA2 does not correlate with biomarkers from a standard lipid profile, which can be explained by the fact that Lp-PLA2 is independent of other biomarkers used to monitor cardiovascular disease. Its infiltration at the subendothelial level does not necessarily depend on its hyperactivity; a single cholesterol particle found at this subendothelial level is sufficient to trigger a low-grade inflammatory response. This means that a standard lipid profile may appear normal, while underlying lipid particles are already present, triggering low-grade inflammatory signalling that is the starting point for vascular damage and the formation of atherosclerotic plaques. A moderate correlation was nevertheless observed between Lp-PLA2 and hs-CRP, which could be evidence of low-grade inflammation that is particularly atherogenic.

- *Limitations of the Study*

The non-prospective nature, the potential effect of age heterogeneity and the unequal distribution of sexes between subgroups constituted the limitation of this study.

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

IV. CONCLUSION

This study, which compared the levels of classic lipid biomarkers (total cholesterol, triglycerides, HDL cholesterol, and LDL cholesterol) with those of Lp-PLA2 in predicting cardiovascular disease, showed a more active expression of Lp-PLA2, particularly in subjects screened for at least one cardiovascular condition or type 2 diabetes. This demonstrates activation of this biomarker during low-grade inflammation, a true starting point for vascular pathologies. Lp-PLA2 should perhaps be used in preventive monitoring of the onset of cardiovascular diseases.

- *Conflict of Interest*

The authors reported no conflicts of interest.

ACKNOWLEDGEMENTS

Our sincerest thanks go to the TRIOS Foundation for their many contributions. We also extend our gratitude to all the patients who agreed to participate in this study, and to all our fellow researchers who have shown a particular interest in this topic.

- *Lists of Tables:*

Table 1 Means Ages of the Studied Population

Study population (size)	Mean age ± standard deviation in year	Median	Minimum	Maximum
Apparently healthy subjects (326)	**41 ± 18**	**39**	**35**	**88**
HTA screened (51)	55 ± 9	58	18	74
T2D screened (25)	41 ± 7	41	19	61
HTA+T2D screened (04)	53 ± 5	54.5	41	61
Subjects (T2D or HTA) known (207)	**45 ± 10**	**46**	**16**	**80**

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T2D knowns (84)	57 ± 10	58	37	79
HTA knowns (123)	52 ± 10	52	23	84
T2D knowns + HTA screened (09)	61 ± 4	61	53	71
T2D knowns + HTA knowns (14)	56 ± 8	58.5	37	72
HTA knowns + T2D screened (13)	62 ± 9	55	47	84

Table 2 Means of Blood Pressure and BMI

Study population (size)	**Vascular risk parameters**		
	BMI	**Systolic**	**Diastolic**
Apparently healthy subjects (326)	24±10	120.5±38,2	73±24
HTA screened (51)	26±10.5	162±61	89±29
T2D screened (25)	24±9.2	127±45	77±26
HTA+T2D screened (04)	24±8,2	159±79	94±47
subjects T2D or HTA knowns (207)	25±8	145±35	80±32
T2D knowns (84)	27±13	137±67	78±37
HTA knowns (123)	26±14	144±90	83±49
T2D knowns + HTA screened (09)	26±10	152±76	77±38
T2D knowns + HTA knowns (14)	26±12	139±68	78±37
HTA knowns + T2D screened (13)	27±8.2	154.5±58	83±34
P-value (<0,05)	0.29	0.04	0.03

Table 3 Means of the Different Biomarkers

Biomarkers	**Different groups studied (number of people)**										**P-value**
	Apparently healthy subjects (326)	**HTA screened (51)**	**DT2 screened (25)**	**HTA+T2D Screened (04)**	**subjects T2D or HTA known (207)**	**T2D Knowns (84)**	**HTA Knowns (123)**	**DT2 Knowns + HTA screened (09)**	**DT2 knowns + HTA knowns (14)**	**HTA knowns + DT2 screened (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
Total Cholesterol (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
Triglycerides (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-Cholesterol (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-Cholesterol (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
IAP (TG/HDL-C)	0.22±0.46	0.27±0.24	0.26±1.4	0.16±2	0.25±0.6	0.25±1.7	0.25±0.75	0.24±0.14	0.25±1.8	0.27±0,25	0,56
Lp-PLA2* (ng/ml)	274±174	245±150	131±71	258±85	124±88	124±77	124±75	140±72	124±78	161±71	0,03
CRP-us (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

*Lp-PLA2 values < 180 indicate low risk, 180-200 moderate risk, and ≥ 200 high risk. Source: PARS BIOCHEM kit (Human Lp-PLA2 ELISA kit) with a dilution factor of 1/30. IAP: Plasma atherogenicity index. AIP = Log (TG/HDL-C) with IAP < 0.11: low risk, IAP = (0.11-0.24): moderate risk, IAP > 0.24: high risk. HDL-C: High-Density Lipoprotein cholesterol, LDL-C: Low-Density Lipoprotein cholesterol.

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Table 4 Univariate Analysis of Logistic Regression of Biomarkers in Relation to Cardiovascular Diseases

Independent variables	OR (Odds Ratio)		IC 95%	P-value
Glycaemia (g/l)	0,52		0,22 – 1,61	0,14
HbA1C (%)	0,74		0,54 – 1,76	0,26
Total Cholesterol	1,05		0,85 - 2	0,06
Triglycerides	1,08		0,9 – 2,5	0,07
HDL-Cholesterol	0,54		0,25 – 0,75	0,003
LDL-Cholesterol	0,89		0,78 – 1,26	0,09
IAP (TG/HDL-C)	2,4		1,5 – 2,9	0,03
Lp-PLA2 (ng/ml)	2,9		1,6 - 5	0,001
Hs-CRP (mg/l)	1,7		1,6 - 2	0,04

Table 5 Multivariate Analysis of Logistic Regression of Biomarkers in Relation Cardiovascular Diseases

Independent variables	OR (Odds Ratio) Adjust	IC 95%	P-value
Glycaemia (g/l)	0,55	0,32 – 1,69	0,18
HbA1C (%)	0,77	0,55 – 1,79	0,41
Total Cholesterol	1,8	1,65 – 2,9	0,02
Triglycerides	1,10	0,9 – 2,1	0,07
HDL-Cholesterol	0,79	0,74 – 0,95	0,003
LDL-Cholesterol	1,05	0,88 – 1,46	0,09
IAP (TG/HDL-C)	2,0	1,7 – 3,2	0,03
Lp-PLA2 (ng/ml)	4,5	2,6 - 8	0,001
Hs-CRP (mg/l)	2,0	1,9 – 2,8	0,03

Table 6 Means of Lp-PLA2 According to CVD Risk Factors

Study population (size)	Risk factors		
	Alcohol	Cons. Tabaco	Sedentary lifestyle
Apparently healthy subjects (326)	154±90	135±65	149±78
HTA screened (51)	197±80	237±40	225±94
T2D screened (25)	263±72	220±47	247±54
HTA+T2D screened (04)	271±10	-	200±5
subjects T2D or HTA knowns (207)	165±41	164±19	153±19
T2D knowns (84)	176±53	179±18	155±25
HTA knowns (123)	152±28	149±22	150±12
T2D knowns + HTA screened (09)	173±43	-	152±14
T2D knowns + HTA knowns (14)	149±12	133±09	145±16
HTA knowns + T2D screened (13)	210±35	-	187±42
P-value (<0,05)	<0.001	<0.05	<0.05

➢ *Lists of Figures*

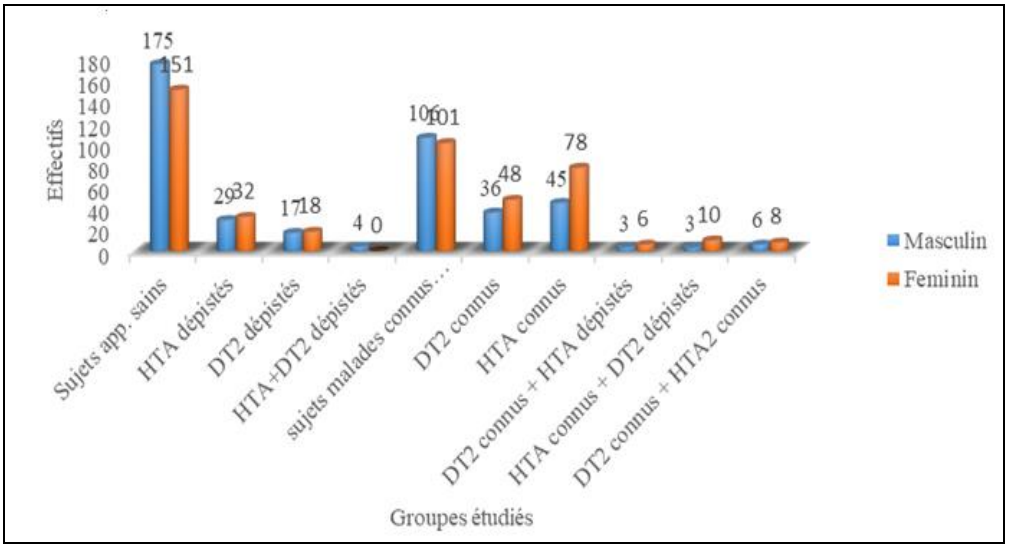

Fig 3 Sex Distribution within the Studied Population

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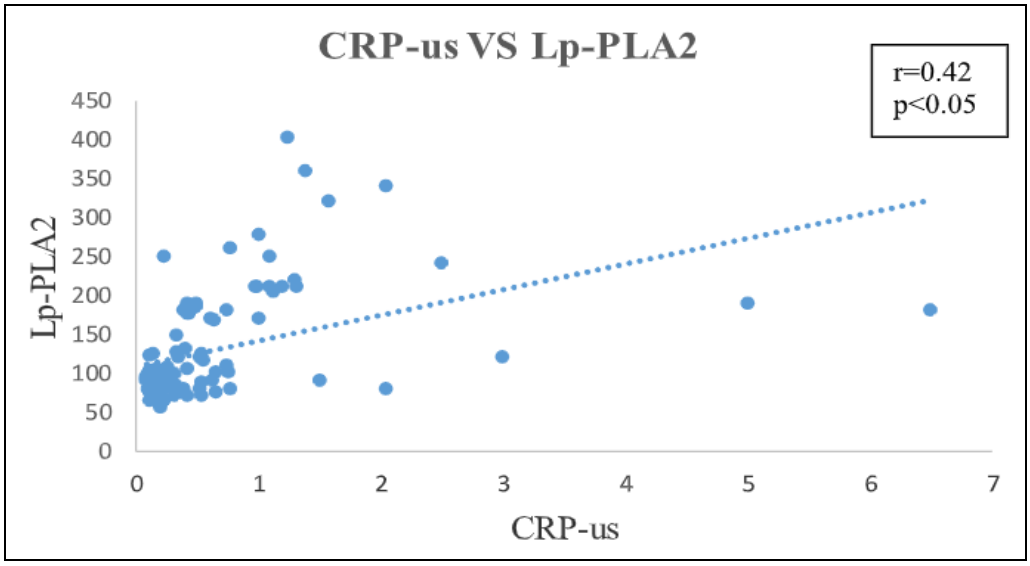

Fig 4 Low Correlation between hs-CRP and Lp-PLA2

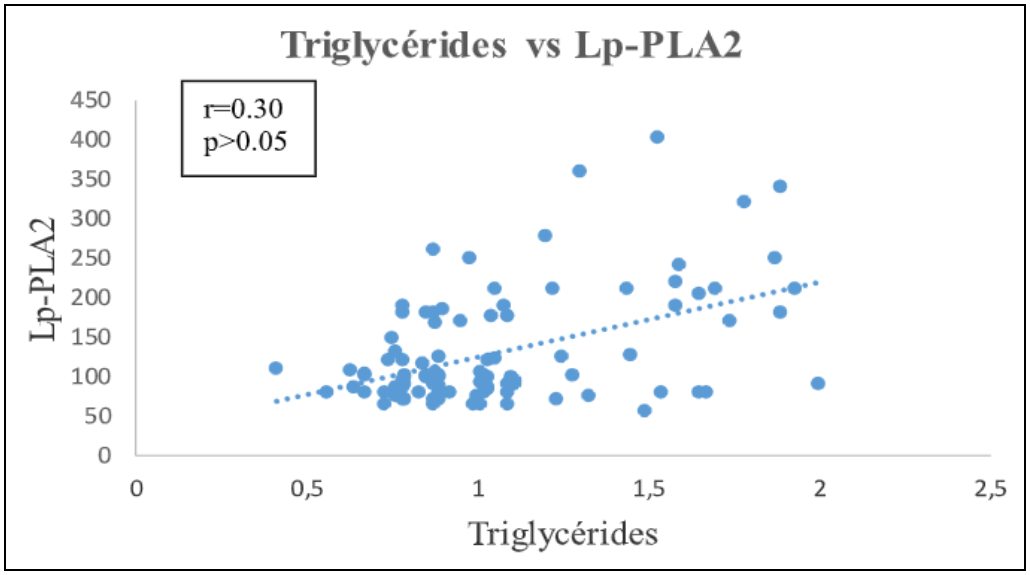

Fig 5 No Correlation between hs-CRP and Lp-PLA2

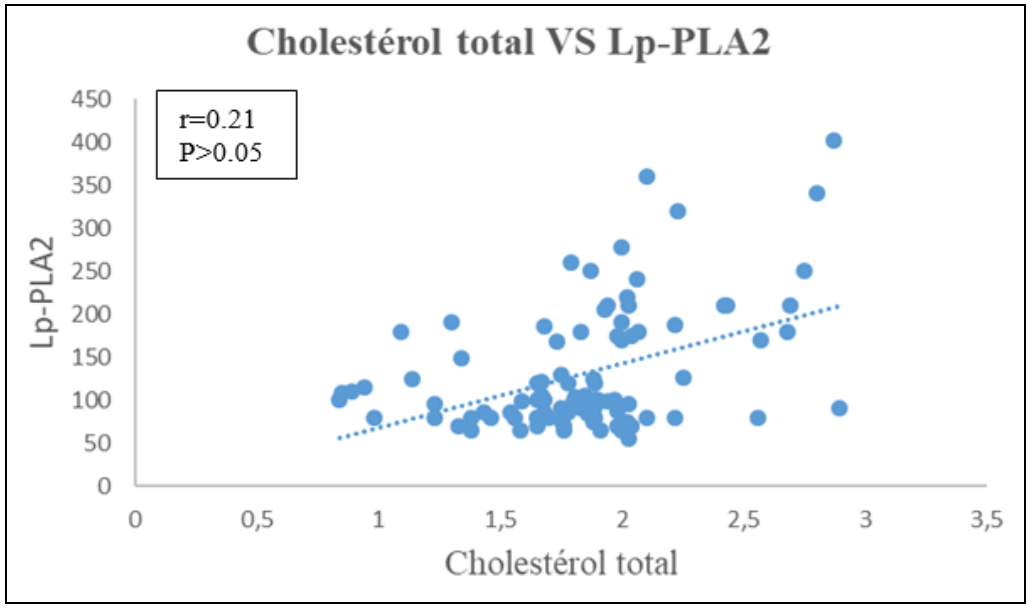

Fig 6 No Correlation between Total Cholesterol and Lp-PLA2

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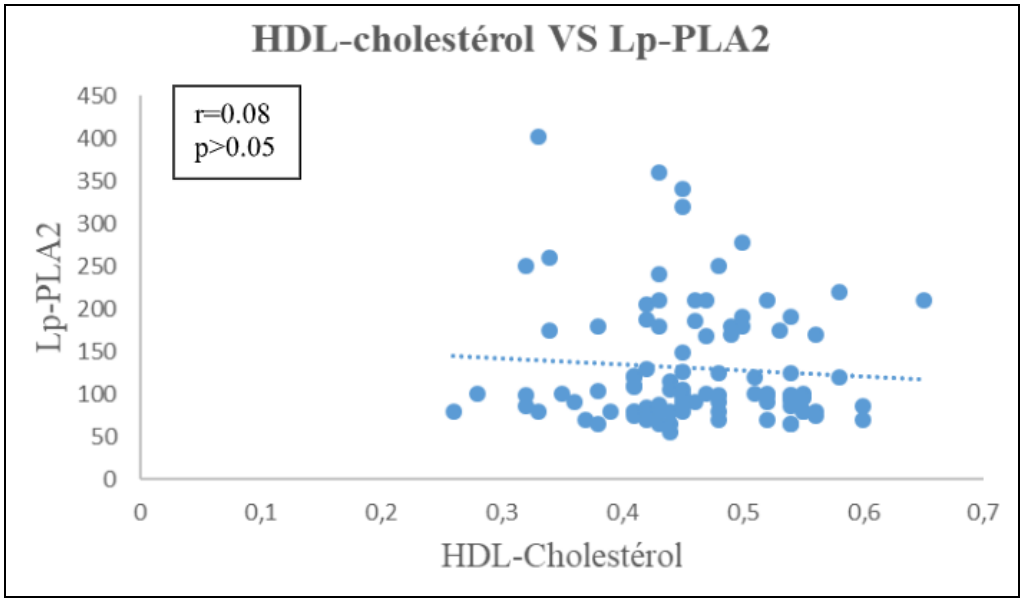

Fig 7 No Correlation between HDL Cholesterol and Lp-PLA2

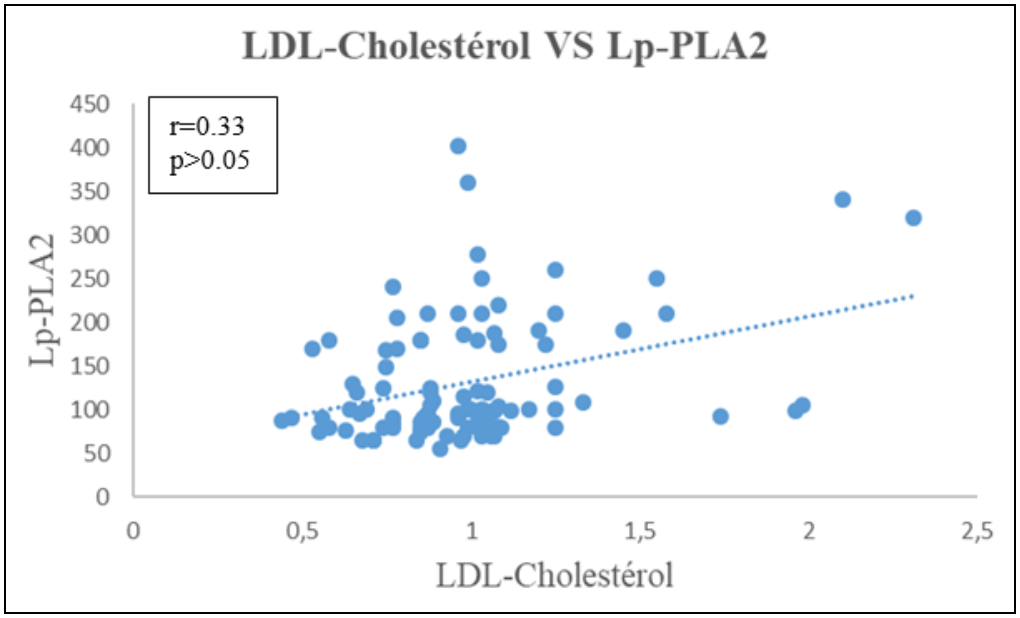

Fig 8 No Correlation between LDL Cholesterol and Lp-PLA2

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