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Study of Lipid Parameters and Risk Factors for Cardiovascular Diseases in North Africa

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The objective of this study is to investigate the correlation between cardiovascular risk factors and levels of high-density lipoprotein (HDL), low-density lipoprotein (LDL), apolipoprotein A, and apolipoprotein B. This study included two groups: The first group consisted of 147 patients diagnosed with cardiovascular diseases, including heart failure (HF), myocardial infarction (MI), and ischemic stroke (IS). These patients were recruited from the Cardiology Department of the Ibn Rochd University Hospital Center in Casablanca, Morocco. The second group comprised 140 healthy individuals recruited from the Biomedical Center of the Pasteur Institute in Casablanca, Morocco. These individuals showed no signs of cardiovascular disease and were not undergoing any medical treatment. The mean ages of the patients and the control group were 62.15 ± 11.02 years and 54.74 ± 6.59 years, respectively. The levels of apolipoprotein A and B were 1.23 ± 0.31 g/L and 0.93 ± 0.21 g/L in patients and 1.45 ± 0.19 g/L and 0.92 ± 0.10 g/L in healthy individuals, respectively. The results showed significantly lower levels of apolipoprotein A in patients compared to the control group ($p < 0.001$). Although apolipoprotein B levels varied between the two groups, the difference was not statistically significant. Risk factors such as smoking, diabetes, and hypertension were found to play a major role in the development of cardiovascular diseases. This study confirms that LDL levels and apolipoprotein A, along with cardiovascular risk factors, contribute to the severity of cardiovascular diseases.

Keywords: Atherosclerosis; Apolipoprotein; Cardiovascular disease; Lipoprotein; North Africa; Risk factors.

Cardiovascular disorders (CVD), including heart failure, myocardial infarction, ischemia, and arterial blood vessel disease, are among the most dangerous medical conditions affecting the heart. CVD and coronary artery disease are major public health issues and leading causes of death worldwide.[1,2] CVD is a significant global health problem, with over three million deaths occurring before age 60 in 2008 and 17.3 million deaths from vascular diseases.[3]

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In developing countries, an epidemiological transition has been observed, characterized by an increase in vascular diseases and atherosclerosis development.t ·u

Atherosclerosis, a chronic inflammatory process caused by excess cholesterol, evolves gradually from childhood due to interactions between the arterial wall and genetic factors.v ,w Traditional CVD risk factors include hypertension, dyslipidemia, and smoking.x Recent studies, such as Ramezankhani et al. (2023), emphasize the importance of assessing lifetime risk (LTR) of CVD, particularly in populations with high metabolic risk factors, like those in North Africa and the Middle East.y Individuals with multiple risk factors (e.g., obesity, diabetes, hypertension) have a significantly higher lifetime risk of CVD, highlighting the need for early prevention strategies.

Hypercholesterolemia, particularly elevated LDL-C, is a primary CVD risk factor and a leading cause of mortality and morbidity globally, affecting approximately one in twenty individuals.[1]p Higher LDL levels increase atherosclerosis risk, while higher HDL levels reduce it.[11] Dyslipidemia is critical in the development of arterial diseases, such as CAD and CVD.[12] Regression of coronary plaque depends on low LDL-C, high HDL-C, and apolipoprotein B (ApoB) levels.[13]

Elevated cholesterol levels are statistically correlated with increased cardiovascular risk, as confirmed by the SCORE[1]t and Framingham studies.[1]u Oxidized LDL plays a significant role in atherosclerotic lesion progression,[1]v and dyslipidemia can lead to CAD, stroke, and peripheral vascular disease.[1]w Ramezankhani et al. (2023) further demonstrated that individuals with optimal traditional risk factors, such as normal cholesterol levels, lived significantly longer without CVD, underscoring the importance of maintaining healthy lipid profiles throughout life.y

MATERIALS AND METHODS

This retrospective epidemiological study involved two groups. The first group consisted of healthy subjects recruited from the Biomedical Center of the Pasteur Institute in Casablanca, Morocco. The second group consisted of patients suffering from cardiovascular diseases, including heart failure (HF), myocardial infarction (MI), and ischemic stroke (IS), who were recruited from the Cardiology Service of the University Hospital Center in Casablanca, Morocco. For each subject, a questionnaire was developed to collect information about age, height, weight, smoking habits, dietary habits, blood pressure, and other relevant factors. All participants in this study provided both oral and written informed consent.

Subjects

A total of 287 subjects were included in the study:

• 147 patients with cardiovascular disease (HF, MI, and IS): Diagnosis was confirmed using ECGs, angiography, and echocardiography. Pregnant women and patients with renal failure were excluded.

• 140 healthy subjects: These individuals exhibited no clinical signs of cardiovascular disease and were not receiving any medical treatment.

Blood sample collection and lipid profile measurement

Blood samples were collected in the morning after an 8-hour fast. Venous blood was drawn into specialized tubes to ensure compliance with international standards. The samples were centrifuged at 3000 × g for 10 minutes, and aliquots of plasma were immediately stored at “80 °C until analysis.

The following parameters were measured

• Glycemia (Gj)

• Lipid profile, including total cholesterol (TC), triglycerides (TG), and high-density lipoprotein (HDL), using standard enzymatic colorimetric methods.

• Low-density lipoprotein (LDL) cholesterol levels were calculated using the Friedewald formula.

Measurements were performed using a Vitros 250 automated analyzer, which employs both dry and liquid chemistry techniques. In this study, dry chemistry plates were used, with a processing capacity of 250 tests per hour.

Dosage of Glycated Hemoglobin (HbA1c)

The measurement of glycated hemoglobin (HbA1c) was performed using the Bio-Rad D-10™ Dual Program. This method is based on the separation of whole blood samples using high-performance liquid chromatography (HPLC) with ion exchange.

Measurement of Apolipoprotein AI and Apolipoprotein B

The concentrations of apolipoprotein AI (Apo AI) and apolipoprotein B (Apo B) were determined using an immunological assay on the BN ProSpec system. This method utilizes specific antibodies that bind to Apo AI and Apo B, initiating an antigen-antibody reaction. The resulting complexes are quantified based on their turbidity, which is proportional to the concentration of the apolipoproteins in the sample.

Research Strategy

A systematic literature search was conducted to identify relevant articles, including review studies, from databases such as Embase, PubMed, Scopus, and Web of Science.

Statistical analysis

Continuous variables were expressed as mean ± standard deviation (SD), and categorical variables as frequencies and percentages. Group comparisons were made using the Student's t-test for normally distributed data.

Logistic regression was used to assess associations between risk factors (e.g., smoking, diabetes, hypertension) and cardiovascular disease (CVD), with odds ratios (OR) and 97.5% confidence intervals (CI) reported. Variables with $p < 0.05$ in univariate analysis were included in multivariate models to adjust for confounders.

RESULTS

The study population was divided into two groups:

• Group 1 (Patients): 40.81% women and 59.18% men.

• Group 2 (Healthy Subjects): 54.28% women and 45.71% men.

Table 1. Demographic and clinical characteristics of patients and healthy subjects

Parameter	Patients (n = 147)	Healthy subjects (n = 140)	P values
Mean age (years)	62.15±11.02	54.74±6.59	<0.001***
Systolic blood pressure TAS (mm Hg)	129.04±24.13	122.80±17.19	0.0049*
Diastolic blood pressureTAD (mm Hg)	73.79±14.17	73.45±7.11	0.784
Glycemia (g/l)	1.56±0.73	0.96±0.17	<0.001***

Table 2. Apolipoproteins AI, B and other lipid parameters of the population studied

Parameter	Patients (n=147)	Healthy subjects (n = 140)	p values
Apo AI	1.23±0.31	1.45±0.19	<0.001***
Apo B	0.93±0.21	0.92±0.10	0.65
CT (g/L)	1.91±0.36	1.64±0.46	0.003**
HDL (g/L)	0.48±0.16	0.49±0.10	0.53
LDL (g/L)	1.53±0.52	1.17±0.27	<0.001***
TG (g/L)	1.91±0.65	1.64±0.38	0.003

*p<0.05 **p<0.01 ***p<0.001 odds ratios and 97.5% IC OR : Odds ratio

Table 3. Multivariate study by logistic regression of risk factors for cardiovascular diseases

Parameter	OR 2.5 % 97.5 %	p values
Gender	0.3627 [0.5807-0.9253]	0.0228 *
Mean age (years)	0.8826[0.9116-0.9389]	<0.001 ***
Systolic blood pressure TAS (mm Hg)	0.9677[0.9815-0.9944]	0.00721 **
Diastolic blood pressure TAD (mm Hg)	0.9751[0.9969-1.0191]	0.786

HDL-C (HDL-cholesterol), LDL-C (LDL-cholesterol), TC (total cholesterol), TG (triglycerides).

The mean age of patients was 62.15 ± 11.02 years, significantly higher than that of controls (54.74 ± 6.59 years; $p < 0.001$), as shown in Table 1. Males predominated in the patient group (59.18% vs. 40.81% females).

Key findings

• Apolipoprotein AI (Apo AI) levels were significantly lower in patients compared to controls (Table 2; $p < 0.001$). Apo AI is the primary protein component of high-density lipoprotein (HDL) and plays a critical role in reverse cholesterol transport, a process by which excess cholesterol is removed from peripheral tissues and transported to the liver for excretion. Lower Apo AI levels impair this process, leading to cholesterol accumulation in arterial walls and contributing to atherosclerosis.

• LDL and total cholesterol levels were significantly higher in patients ($p < 0.001$). Elevated LDL levels are a major driver of atherosclerosis, as LDL particles can infiltrate the arterial wall, become oxidized, and trigger an inflammatory response, leading to plaque formation.

• No significant differences were observed in HDL or apolipoprotein B (Apo B) levels. Apo B is the main structural protein of LDL and very-low-density lipoprotein (VLDL). It plays a key role in the delivery of cholesterol to peripheral tissues. While Apo B levels did not differ significantly, the higher LDL levels in patients suggest increased atherogenic potential.

These findings are consistent with a study conducted on the Tunisian population.[1]x

Hypertension and smoking prevalence

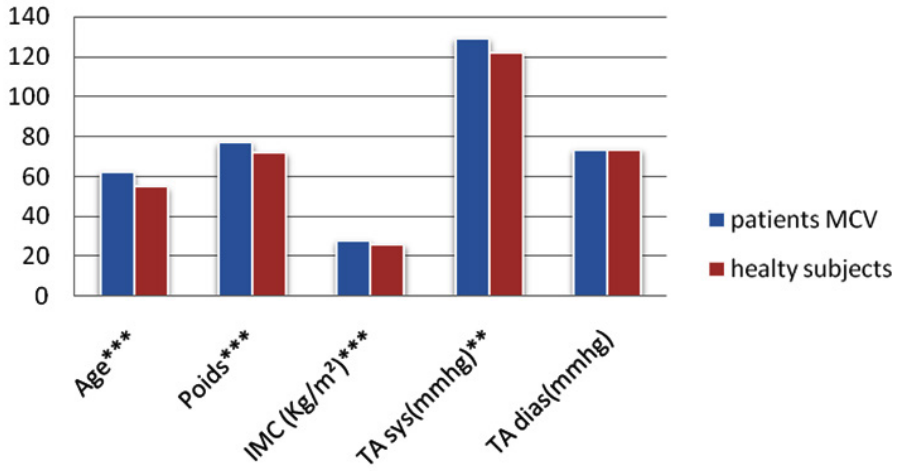
• Hypertension prevalence in patients (55.78%) is consistent with findings from the Tunisian population (31%)[1]y and other Tunisian studies

Table 4. Multivariate study by logistic regression of risk factors for cardiovascular diseases

Parameter	OR 2.5 % 97.5 %	pvalues
Smoking	6.8137[16.1413-47.6599]	<0.001 ***
Drinking	0.8017[1.0144-1.2838]	0.1
Activity	0.6011[0.9560-1.5197]	0.087
Obesity	0.4461[0.7263-1.1785]	0.29

Table 5. Multivariate study by logistic regression of risk factors for cardiovascular disease

Parameter	OR 2.5 % 97.5 %	p values
Glyc (g/l)	0.0011[0.0095-0.0237]	<0.001 ***
HbA1c %	0.1678[2.4647-3.4344]	<0.001 ***
Apo AI	9.7302[27.2278-84.6921]	<0.001 ***
Apo B	0.1852[0.7363-2.8745]	0.33



Graph 1. Comparison of anthropometric parameters between healthy subjects and patients

reporting hypertension prevalence rates of 29–37%.[2]p ,[21].

• Smoking prevalence (37.41%) was consistent with the rate reported in the Tunisian population (30%).[1]x

Table 6. Prevalence of major cardiovascular risk factors in patients

Characteristics	Patients (n)	Patients (%)
Gender (male) n(%)	87	(59.18) *
Gender (female)	60	-40.81
Smokers n(%)	55	(37.41) ***
No Smokers	92	-62.58
Obesity n(%):		
Overweight	52	-35.37
Obese	48	-32.65
Normal	47	(31,97)
Activity n(%)	72	-48.97
Without activity	75	-51.02
Diabetes n(%)	78	(53.06) ***
No Diabetes	69	-46.93
DT1 n(%)	2	-1.36
DT2	73	(49.65) ***
DT1-DT2	3	(2,04)
Hypertensive n(%)	82	(55.78) **
Non hypertensive	65	-44.21
Dyslipidemia n(%)	21	-14.28
No Dyslipidemia	126	-85.71
With Family history n(%)	39	-26.53
Without Family history	108	-73.46
Drinking (%) n(%)	9	-6.12
No Drinking (%)	138	-93.87

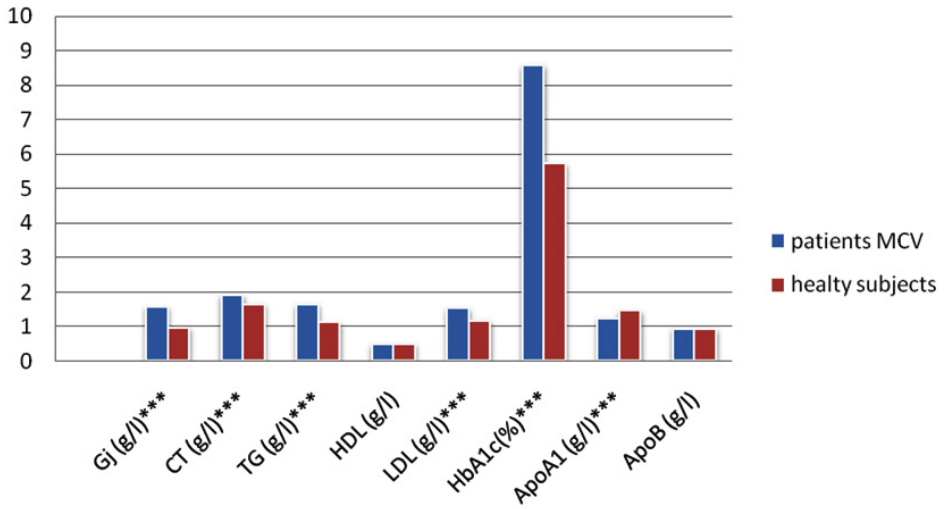
• Smoking was more prevalent among males, aligning with other studies.[22],[23]

Anthropometric and biochemical parameters

• Significant differences were observed in age, weight, BMI, and systolic pressure (Graph 1).

Table 7. Prevalence of cardiovascular risk factors in controls

Characteristics	Healthy subjects (n)	Healthy subjects (%)
Gender (male) n(%)	64	-45.71
Gender (female)	76	-54.28
Smokers n(%)	5	-3.57
No Smokers	135	-96.42
Obesity n(%):		
Overweight	71	-50.71
Obese	14	-10
Normal	55	-39.28
Activity n(%)	67	-47.85
No activity	73	-52.14
Diabetes n(%)	0	0
No Diabetes		
DT1 n(%)	0	0
DT2		
DT1-DT2		
Hypertendu n(%)	0	0
Non Hypertendu		
Dyslipidémia n(%)	0	0
No Dyslipidémia		
With Family history n(%)	0	0
Without Family history		
Drinking n(%)	0	0
No Drinking (%)		



Graph 2. Comparison of lipid parameters between healthy subjects and patients.

• No significant difference was found in diastolic pressure.
• Blood sugar and lipid parameters (Gj, CT, TG, LDL, HbA1c) were significantly higher in patients (Graph 2).
• Apolipoprotein A1 (Apo A1), a key marker of lipid dysregulation, was lower in patients. Apo A1 is essential for HDL formation and function. Reduced Apo A1 levels impair HDL's ability to mediate reverse cholesterol transport and exert anti-inflammatory and antioxidant effects, further exacerbating CVD risk.
• HDL levels showed no significant difference between the groups. However, the functional quality of HDL (e.g., its ability to promote cholesterol efflux) may be compromised in patients due to lower Apo A1 levels, even if HDL concentration remains unchanged.

Global context

The high prevalence of hypertension and smoking in the patient group aligns with studies conducted in both developed and developing countries.[24,25] These studies underscore the global burden of cardiovascular risk factors and their consistent association with CVD across diverse populations.

DISCUSSION

Lipid Profile and Cardiovascular Risk

• HDL levels were lower in patients than in controls, though the difference was not statistically significant (Table 2). In contrast, LDL cholesterol levels were significantly higher in patients ($p < 0.001$).[2]v ,[2]w Both elevated LDL and reduced HDL are recognized as independent risk factors for cardiovascular disease.[2]v ,[2]w
• HDL plays a critical role in reverse cholesterol transport, a process by which excess cholesterol is removed from peripheral tissues and transported to the liver for excretion.[2]x The protective role of HDL is diminished in patients, while elevated LDL levels may result from dysregulated lipid metabolism.
• Recent data from the Global Burden of Disease Study 2019 highlight that high LDL cholesterol remains a significant contributor to cardiovascular disease burden in North Africa and the Middle East, with a 26.5% decrease in age-standardized death rates (ASDR) from 1990 to 2019, despite a 5.5% increase in exposure.[2]y

Apolipoproteins and Cardiovascular Disease

• The study identified a correlation between apolipoprotein AI (Apo AI) levels and the development of cardiovascular diseases (Table 2). Apo AI levels were significantly lower in patients compared to healthy individuals, while Apo B showed less variation.
• Apo AI is a key component of HDL and is essential for its function in reverse cholesterol transport. Lower Apo AI levels impair HDL's ability to protect against atherosclerosis, while Apo B, the main structural protein of LDL, contributes to the delivery of cholesterol to peripheral tissues.
• These findings align with regional trends showing that dyslipidemia, characterized by low HDL cholesterol and elevated LDL cholesterol, is a critical factor in the progression of cardiovascular diseases.[3]p

Non-Modifiable and Modifiable Risk Factors

• Among non-modifiable risk factors, age and sex were associated with increased coronary risk, with a highly significant p-value for age ($p< 0.001$) and a significant p-value for sex (0.0228).
• Tobacco use was a highly significant risk factor ($p < 0.001$), with 37.41% of patients being smokers compared to only 3.57% of controls.
• Elevated blood sugar and HbA1C levels significantly contribute to cardiovascular disease development. Among patients, 50.34% had type 2 diabetes, 2.72% had type 1 diabetes, and only 46.93% were non-diabetic.
• Diabetes, particularly type 2 diabetes, is a major contributor to cardiovascular disease. Elevated HbA1C, along with increased LDL, decreased HDL, hypertension, age, male sex, and smoking, were identified as independent risk factors for coronary artery disease.[31]

Obesity and Dyslipidemia

• Obesity is a major risk factor for cardiovascular disease, and its atherogenic effects are partly mediated by dyslipidemia, characterized by low HDL cholesterol (HDL-C) and apolipoprotein AI levels.[32]
• Cardiovascular risk and disease progression remain poorly understood in diabetic patients, particularly those with type 2 diabetes.[33]

CONCLUSION

The findings of this study indicate that dyslipidemia is significantly associated with an increased risk of cardiovascular disease (CVD) in the studied patient population. Specifically, lower levels of apolipoprotein AI (Apo AI) and elevated LDL cholesterol levels were identified as critical factors contributing to the development of atheromatous plaques and the progression of atherosclerosis. These results are consistent with established mechanisms linking dyslipidemia to CVD pathogenesis.

The study population exhibited several modifiable and non-modifiable risk factors associated with CVD severity, including:

- Advanced age,
- Smoking,
- Diabetes, and
- Elevated systolic blood pressure.

Notably, dyslipidemia was more pronounced in diabetic patients, characterized by elevated levels of atherogenic lipoproteins, such as LDL cholesterol.

Within the context of the Moroccan population, these findings suggest a high prevalence of cardiovascular risk factors, potentially influenced by ongoing epidemiological and lifestyle transitions, including changes in dietary habits. However, it is important to note that these observations are specific to the studied cohort and may not be generalizable to the entire Moroccan population without further research.

The study also highlights the role of metabolic syndrome (MetS) in CVD development, with contributing factors such as:

- Obesity,
- Hyperglycemia,
- Hypertriglyceridemia,
- Low HDL levels, and
- Hypertension.

These factors are influenced by a combination of genetic predisposition and environmental influences, underscoring the multifactorial nature of CVD risk.[34]

Recent evidence from a systematic review and meta-analysis conducted in Africa supports these findings, demonstrating that dyslipidemia is prevalent among individuals with:

- A body mass index (BMI) >25.0 kg/m²,
- A waist circumference (WC) >94 cm, and
- Diabetes mellitus (DM) or hypertension (HTN).[35]

These associations emphasize the need for targeted public health interventions to address dyslipidemia and other modifiable risk factors in high-risk populations.[36] However, further studies are required to confirm these findings in broader and more diverse populations.

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Conflict of Interest

The author(s) do not have any conflict of interest.

Data Availability Statement

This statement does not apply to this article.

Ethics Statement

This research did not involve human participants, animal subjects, or any material that requires ethical approval.

Informed Consent Statement

This study did not involve human participants, and therefore, informed consent was not required.

Clinical Trial Registration

This research does not involve any clinical trials

REFERENCES

1. Borzyszkowska J, Stanislawska-Sachadyn A, Wirtwein M, et al. Angiotensin converting

enzyme gene polymorphism is associated with severity of coronary artery disease in men with high total cholesterol levels. *J Appl Genet*. 2012;53(2):175-182.
2. El Boukhrissi F, Bamou Y, Ouleghzal H, Safi S, Balouch L. Prévalence des facteurs de risque des maladies cardiovasculaires et du syndrome métabolique chez les femmes de la région de Meknès, Maroc. *Médecine des Maladies Métaboliques*. 2017;11(2):188-194.
3. Mendis S, Puska P, Norrving B. *Global Atlas on Cardiovascular Disease Prevention and Control*. Geneva: World Health Organization; 2011.
4. Touze JE. Les maladies cardiovasculaires et la transition épidémiologique du monde tropical. *Med Trop*. 2007;67(6):541-542.
5. Verdier F, Fourcade L. Evolution des facteurs de risque cardio-vasculaire dans les pays en développement. *Med Trop*. 2007;67(6):552-558.
6. Thiombiano LP, Mbaye A, Sarr SA, et al. Prévalence de la dyslipidémie dans la population rurale de Gueoul Sénégal. *Ann Cardiol Angeiol (Paris)*. 2015;64(2):2-4.
7. Nuoffer JM, Berneis K, et al. Athérosclérose et hyperlipidémies primaires – un problème pédiatrique. *Paediatrica*. 2005;16(6):12-18.
8. Saito M, Ishimitsu T, Minami J, et al. Relation of plasma high-sensitivity C-reactive protein to traditional cardiovascular risk factors. *Atherosclerosis*. 2003;167(1):73-79.
9. Ramezankhani A, Azizi F, Hadaegh F. Lifetime risk of cardiovascular disease stratified by traditional risk factors: Findings from the cohort of Tehran lipid and glucose study. Hellenic J Cardiol . 2023;73(1):36-46.
10. Jelassi A, Jguirimi I, Najah M, et al. L'hypercholestérolémie familiale en Tunisie. *Pathol Biol (Paris)*. 2009;57(5):444-450.
11. Ndongog GPY. Les infarctus du myocarde - thrombolyses: A propos de 36 cas au service de Réanimation de l'hôpital Principal de Dakar. Université de Mali; 2004.
12. Meyer E, Julien F, Vanina B, et al. High-density lipoprotein subclass profile and mortality in patients with coronary artery disease: Results from the GENES study. *Arch Cardiovasc Dis*. 2016;109(11):607-617.
13. Masson W, Siniawski D, Lobo M, et al. Association between LDL-C, non HDL-C, and Apolipoprotein B levels with coronary plaque regression. *Arq Bras Cardiol*. 2015;105(1):11-19.
14. Conroy RM, Pyörälä K, Fitzgerald AP, et al. Estimation of ten-year risk of fatal cardiovascular disease in Europe: the SCORE project. *Eur Heart J*. 2003;24(11):987-1003.
15. Dawber TR, Kannel WB, Revotskie N. The epidemiology of coronary heart disease. *Proc R Soc Med*. 1962;55(4):265-271.
16. Glorian M, Limon L. L'athérosclérose, une maladie inflammatoire. *Rev Francoph Lab*. 2007;389:43-48.
17. Thissen JP, et al. Les normes du cholestérol. *Louvain Med*. 1999;118:139-144.
18. Hrira MY, Kerkeni M, Hamdad B, et al. Apolipoprotein A-I, apolipoprotein B, high-sensitivity C-reactive protein and severity of coronary artery disease in Tunisian population. *Cardiovasc Pathol*. 2012;21(5):455-460.
19. Elasmi M, Feki M, Sanhaji H, et al. Prévalence des facteurs de risque cardiovasculaires conventionnels dans la population du Grand Tunis. *Rev Epidemiol Sante Publique*. 2009;57(2):87-92.
20. Benromhdane H, Bougatef S, Skihri H, et al. Le registre des maladies coronaires en Tunisie: organisation et premiers résultats. *Rev Epidemiol Sante Publique*. 2004;52(6):558-564.
21. Ghannem H, Hadj Fredj A. Epidemiology of hypertension and other cardiovascular disease risk factors in the urban population of Soussa, Tunisia. *East Mediterr Health J*. 1997;3(3):472-479.
22. Meaney E, Lara-Esqueda A, Ceballos-Reyes GM, et al. Cardiovascular risk factors in the urban Mexican population: the FRIMEX study. *Public Health*. 2007;121(5):378-384.
23. Gu D, Gupta A, Muntner P, et al. Prevalence of cardiovascular disease risk factor clustering among the adult population of China. *Circulation*. 2005;112(5):658-665.
24. Fédération Française de Cardiologie. Les facteurs de risque et comportements de prévention dans la population des trois registres de population; MONICA-France. Enquête Fédération Française de Cardiologie; 1997.
25. Gu D, Reynolds K, Wu X, et al. Prevalence, awareness, treatment and control of hypertension in China. *Hypertension*. 2002;40(6):920-927.
26. Gordon T, Castelli WP, Hjortland MC, et al. High density lipoprotein as a protective factor against coronary heart disease: the Framingham Study. *Am J Med*. 1977;62(5):707-714.
27. Briel M, Ferreira-Gonzalez I, You JJ, et al. Association between change in high density lipoprotein cholesterol and cardiovascular disease morbidity and mortality. *BMJ*. 2009;338:b92 .
28. Khera AV, Cuchel M, de la Llera-Moya M, et al. Cholesterol efflux capacity, high-density lipoprotein function, and atherosclerosis. *N Engl J Med*. 2011;364(2):127-135.
29. Malekpour M-R, Abbasi-Kangevari M, Ghamari S-H, et al. The burden of metabolic risk factors

in North Africa and the Middle East, 1990–2019: findings from the Global Burden of Disease Study. Lancet. 2023;60:1-13.

30. Stratton IM, Adler AI, Neil HA, et al. Association of glycaemia with macrovascular and microvascular complications of type 2 diabetes (UKPDS 35). *BMJ*. 2000;321(7258):405-412.
31. Rashid S, Genest J. Effect of obesity on high-density lipoprotein metabolism. *Obesity (Silver Spring)*. 2007;15(12):2875-2888.
32. Vergès B. Physiopathologie de la dyslipidémie du diabète de type 2: nouvelles perspectives. *Médecine des Maladies Métaboliques*. 2019;13(2):140-146.
33. Wolfram RM, Brewer HB, Xue Z, et al. Impact of low high-density lipoproteins on in-hospital events and one-year clinical outcomes in patients with non-ST-elevation myocardial infarction. *Am J Cardiol*. 2006;98(6):711-717.
34. Schlienger JL. Modifications thérapeutiques du mode de vie et prévention cardiovasculaire chez les sujets diabétiques de type 2. *Médecine des Maladies Métaboliques*. 2019;13(1):27-35.
35. Obsa MS, Ataro G, Awoke N, et al. Determinants of Dyslipidemia in Africa: A Systematic Review and Meta-Analysis. Front Cardiovasc Med. 2022;8:778891.
36. Farhane H, Motrane M, Anaibar FE, et al. Genetic polymorphisms associated with metabolic syndrome in North Africa: Systematic review and meta-analysis. *Biomed Pharmacol J*. 2023;16(4):1897-1913.