ISSN:0976-3031

Available Online at http://www.recentscientific.com

CODEN: IJRSFP (USA)

International Journal of Recent Scientific Research
Vol. 9, Issue, 9(C), pp. 28854-28858 September, 2018

International Journal of Recent Scientific Research

DOI: 10.24327/IJRSR

Research Article

XBA 1 AND ECOR 1 POLYMORPHISMS OF APOLIPOPROTEIN B100 GENE AND LIPIDS ABNORMALITIES IN HIV INFECTED PATIENTS

Koné F*[1,3]., Edjème-Aké A[1]., Toni T[3]., Anné B.JC[2]., Hauhouot-Attoungbré ML[1]., Ahiboh H[1,3]., Djaman A.J[2] and Monnet D[1]

[1]Department of Biochemistry and Molecular Biology, Faculty of Pharmaceutical and Biological Sciences, FHBU -Abidjan, Côte d'Ivoire
[2]Laboratory of Pharmacodynamics and Biochemistry, Faculty of Biosciences (FHB University-Abidjan, Côte d'Ivoire)
[3]Centre de diagnsotic et de Recherche sur le SIDA et les autres Maladies infectieuses- Abidjan, Côte d'Ivoire

DOI: http://dx.doi.org/10.24327/ijrsr.2018.0909.2746

ARTICLE INFO

Article History:
Received 4th June, 2018
Received in revised form 25th July, 2018
Accepted 23rd August, 2018
Published online 28th September, 2018

Key Words:
Lipoproteins, mutations, PCR-RFLP, HIVART-naive, Côte d'Ivoire

ABSTRACT

Human Immuno-deficiency Virus (HIV) infection is associated with lipoprotein abnormalities leading to cardiovascular diseases. Few reports are available about plasma apoproteins concentration abnormalities and apolipoproteins genes mutations in HIV infected persons. This study aimed to investigate the association between lipoprotein abnormalities and the Apolipoprotein B100 gene Xba1 and EcoR1 polymorphisms in HIV infected patients. A total of 87 controls and 70 HIV-infected antiretroviral-naive patients were included. Their serum lipids and apolipoproteins profiles were determined. In subjects with dyslipidemia, the molecular characterization of the apolipoprotein B100 gene was carried out using polymerase chain reaction-restriction fragment length polymorphism (PCR-RFLP). Total cholesterolemia (1.82±0.46 g/L or 4.70±1.2 mMol/L *vs* 1.97±0.44 g/L or 5.08±1.14 mMol/L) and apolipoproteinemia A1 (1200±0250 mg/L or 1.20±0.25 g/L *vs* 1430±240 mg/L or 1.43±0, 24g/L) were lower in HIV-positive (*p=0.038* and *0.0001* respectively) but the atherogenic index ApoB100/ApoA1 (0.79±0.32 *vs* 0.69±0.22) was higher in HIV-positive (*p=0.041*). Apo B100 gene mutations related to both polymorphisms studied were found in the 2 groups. Hyperapolipoproteinemia B100 and normal LDL Cholesterolemia were predominant regardless of the polymorphism (Xba1 or Eco R1) and allele status (mutant or wild).HIV-positive patients were more at risk of cardiovascular diseases. The mutations of the ApoB100 gene have been found but their established relationship with dyslipoproteinemias is to be confirmed.

INTRODUCTION

Among West African countries, Côte d'Ivoire has the highest rate of HIV infection with a prevalence rate of 3.2% in the adult population[1, 2]. The natural evolution of this infection is accompanied by disorders of the serum lipid levels. However, lipid profiles described are sparse, especially for total cholesterol (CT)[3, 4, 5]. Essentially they consisted of decreased high density cholesterol (HDL-C) and low density cholesterol (LDL-C) and an increase in triglycerides (TG)[4, 6]. During antiretroviral therapy (ART), these disorders consisted of decreased HDL-C[3] and increased LDL-C which together constitute a highly atherogenic lipid profile[7,8,9]. Often increased plasma LDL-C is associated with increased apolipoproteinemia B (ApoB) [7]. Therefore, HIV positive patients receiving ART or not, have lipid metabolism abnormalities or dyslipidemia that exposes them to cardiovascular disease, such as atherosclerosis[10].

In the literature, several data describing the lipid metabolic disorders in HIV-positive patients have been reported, but those concerning apoprotein abnormalities are rare. However, apolipoproteins are the major proteins transporting and guiding metabolism of blood lipids in high density lipoprotein (HDL) and low density lipoproteins (LDL). To our knowledge, very few reports and data concerning the molecular abnormalities affecting the genes of these apolipoproteins in HIV-positive patients are available. Knowing that all proteins are encoded by

Corresponding author:* **Koné F
Department of Biochemistry and Molecular Biology, Faculty of Pharmaceutical and Biological Sciences, FHBU -Abidjan

a specific gene, we hypothesized forward that there are abnormalities in the gene of apoproteins which transport plasma lipids or abnormalities in their genetic expressions of this gene.

The aim of this study was to investigate the relationship between lipoprotein abnormalities and the polymorphism of the ApoB100 gene during HIV infection.

EXPERIMENTAL SECTION

MATERIAL

Period of study and study population

From february 2013 to march 2015, in Abidjan (Côte d'Ivoire), the study enrolled 87 healthy voluntary blood donors from the National Blood Transfusion Center (NBTC) and 70 HIV-infected antiretroviral-naive patients from the Blood donors monitoring center (BDMC). The study population consisted of a group of assumed healthy subjects apt to donate blood according to the NBTC criteria on blood donation [11]. The second group consisted of HIV-positive ART-naive patients. All HIV ART-naïve patients received for all reasons were included until we obtained 70 participants. Each group had a properly filled medical card. The biochemical measurements were done for every participants, however only subjects with a disturbed lipid balance were considered for the molecular biology analyzes. For a preliminary study, we randomly selected 16 participants (5 healthy and 11 HIV-positives) among those with lipid abnormalities.

Concerning ethical considerations, we conducted the study with the agreement of the national ethics committee of Côte d'Ivoire and the reference number is: N° 3766/MSHP/30/Juillet 2009. The study was validated by the leaders of the NBTC and the BDMC. The identification of each subject was done under anonymity by assigning them a code.

Primers and endonucleases used [14, 15]

The primers were synthesized by SIGMA-ALDRICH (France). Their sequences were as followed: Bc1(forward): batch HA06611949 (5'-GGAGACTATTCAGAAGCTAA-3') / Bc1R (reverse): batch HA06611950 (5'-CTGAGAGAAGTGTCTTCGAAG-3') for the study of Xba1 polymorphism andBc2 (forward): batch HA06611951 (5'-GAAGAG CCTGAAGACTGACT-3') / Bc2R (reverse): batch HA06611952 (5'-CTCGAAAGGAAGTGTAATCAC-3') for the study of EcoR1 polymorphism. Enzymatic digestion was performed using the 2 restrictive enzymes: Xba1 (recognized sequence: 5'T / CTAGA3') and EcoR1 (recognized sequence: 5'G / AATTC3') [12].

METHODS

Type of study

A prospective cross-sectional study was carried out, involving first the measurement of substrates in serum (TG, total cholesterol and its fractions, Apolipoprotein A1 and Apolipoprotein B100). Then the concordance between the disorders in the lipid balance (TG, TC and its fractions) and those of the apoproteins (A1 and B100) were analyzed in order to highlight the one that would allow a better detection of cardiovascular risk. In the second step, we carried out the molecular characterization of the apolipoprotein B100 gene. This second step was performed only in subjects with lipoprotein abnormalities in order to enhance the possibilities to observe molecular abnormalities.

Sample Collection and storage

Blood samples were collected through venipuncture at the elbow after fasting for at least 12 hours (h). The blood was collected in 2 tubes: one tube without anticoagulant for the determination of lipids and apolipoproteins and the second tube containing Ethylene Diamine Tetra Acetic Acetic (EDTA) for molecular biology analyzes. After centrifugation of these tubes, serum, plasma and cells were aliquoted and frozen at -80°C when the analyses were to be done later.

Methods of biological analysis

Determination of lipoprotein contents

Lipid parameters in serum, particularly TG, TC, HDL-C and LDL-C were determined using the COBAS INTEGRAS 400 PLUS (Roche, France) by enzymatic colorimetric test according to Trinder method. ApoA1 and ApoB100 were measured by immunoturbidimetry method.

Determination of the ApoB100 Xba1 and EcoR1 gene polymorphism

Total genomic DNA was isolated from peripheral blood leukocytes using ion exchange resin column through the Qiagen® kit method[13]. A final volume of 100 µl pure DNA was obtained. Concentration (µg/ml) and purity of DNA extracted was checked using ultra-violet visible spectrophotometry at absorbance 260 nm and 280 nm. When the DNA concentration of the extract solution was less than 30 ng/µl or the purety less than 1.5, the extraction of the sample concerned was resumed. Each validated extract was then divided into 2 aliquots in cryotubes; one of them was stored at 4°C until amplification and the second at -80°C for further analyzes.

PCR of each sample was carried out in a total volume of 50µl comprising 1.5 µl of each primer (10 Mm), 5 µl of a mixed solution of the 4 dNTP (2 mM), 1.5 µl of $MgCl_2$ (50 mM), 5 µl of 10X buffer solution, 0.5 µl Taq polymerase (5 U/µl), 5 µl genomic DNA and 30 µl of molecular grade water. We did an end point PCR which was performed on an ABI 2720 Thermal Cycler (Life Technology). The PCR programs consisted of initial denaturation for 5 min at 95°C, followed by 33 cycles of denaturation at 95°C for 45 s, annealing at 58°C for 45 s and extension at 72°C for 45 s followed by a final extension at 72°C for 10 min and finally 4°C continually [14, 15]. After PCR, the amplification products were submitted to digestion with the restriction enzymes separately: Xba1 (T / CTAGA) and EcoR1 (G / AATTC) [12,15, 16] after mix and incubation at 37°C overnight. After the digestion step, the DNA fragments were separated by electrophoresis on 2% agarose gel containing ethidium bromide (ETB) for DNA staining. The revelation of the migrated fragments was done under ultraviolet light (UV) and the gel images were stored as photographic and electronic file. In order to check the size of the DNA fragments, a DNA ladder was migrated in the first well of the gels (Figure. 1).

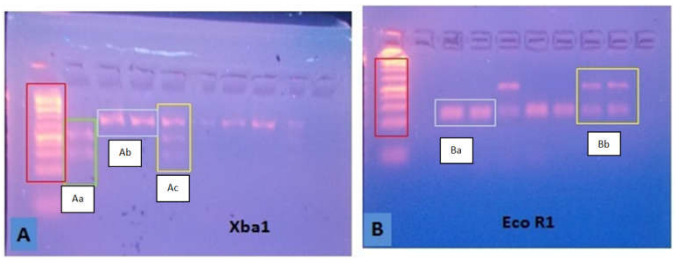

Figure 1 Electrophoretic profiles of ApoB100 gene amplicons after enzymatic digestion with Xba1 endonuclease (profiles A) and EcoR1 endonuclease (profiles B)

A: genotypes of Xba1 Polymorphism; **Profile (Aa)**: Homogeneous population of digested DNA fragments (**mutant DNA= X+/+**), **Profile** (**Ab**): homogeneous population of undigested DNA fragments (**wild DNA= X-/-**), **Profile (Ac)**: heterogeneous population of undigested DNA fragments and digested DNA fragments (mixture of **wild and mutantDNA= X+/-)**

B: genotypes of Eco R1 polymorphism; **Profile (Ba)**: homogeneous population of digested DNA fragments (**wild DNA= E+/+**), **Profile (Bb)**:heterogeneous population of digested and undigested DNA fragments (mixture of **wild and mutant DNA=E+/-)**

Statistical analysis

The data collected was analyzed using the SPSS 16.0 software. The presence of DNA digestion was noted (+) and its absence (-). Quantitative variables were expressed as mean ± SD, minimum and maximum. The qualitative ones were expressed as percentages (%) and numbers (n).The comparisons were made using statistical tests:Student t test for the quantitative values and Chi 2 for the qualitative values. The tests were considered significant at risk $\alpha<0.05$.

RESULTS AND DISCUSSION

RESULTS

Sociodemographic characteristics

In this study, we recruited 157 subjects of whom 55.4% (n = 87) were presumed to be healthy and 44.6% (n = 70) were HIV-positive. The average age of the study population was 37.62 ± 9.73 years and there was a male predominance (sex ratio: 2.27). The mean body mass index (BMI) of HIV-positive was higher than that of the presumed healthy subjects (25.03 ± 4.81Kg / m2 vs 23.49 ± 3.53 Kg / m^2; p = *0.025*).

Biochemical characteristics

The prevalence of apoprotein abnormalities and that of classic lipid abnormalities was respectively 77.07% and 49.68%. TheHIV-positivegroup had lower mean of total cholesterolemia and apoproteinemia A1 than the healthy group (*p = 0.038* and *p <0.0001* respectively) but they had higher mean value of atherogenic index ApoB100/ApoA1 (p=0.041) (Table I).

Table I: Comparison of Mean Concentrations of Biochemical Parameters

Parameter	Healthy subject (n=87)	HIV-positive (n=70)	P-Value
	mean±SD	mean±SD	
TG	1.06±0.04 g/L 1,21±0,04 mMol/L	1.19±0 .64 g/L 1,36±0,73 mMol/L	0.138
T C	1 .97±0 .44 g/L 5.08±1.14 mMol/L	1.82±0.46 g/L 4.70±1.2 mMol/L	0.038
HDL-C	0.55±0.16 g/L 1.37±0.40 mMol/L	0.50±0.23 g/L 1.25±0.57 mMol/L	0.083
LDL-C	1.08±0.37 g/L 2.76±0.95 mMol/L	1.17±0.43 g/L 2.99±1.10 mMol/L	0.14
APO A1	1430±240 mg/L 1.43±0.24 g/L	1200±0250 mg/L 1.20±0.25 g/L	0.000
APO B	970±300 mg/L 0.97±0.30 g/L	900±270 mg/L 0.90±0.27 g/L	0.121
TC/HDL-C	3.88±1.50	4.55±4.43	0.134
APO B /APO A1	0.69±0.22	0.79±0.32	0.041

The analysis of the concordance between lipid parameters measurement and apolipoproteins ones showed that apoprotein abnormalities were more sensitive in the search for atherogenic risk than those of classic lipids abnormalities (X^2: *p=0.013;* Kappa: k=0,17).

Molecular characterization

The two polymorphisms of the apoB100 gene studied were found in the 2 groups of the study population. Of the 3 possible expected genotypes in each case, we found 3 genotypes of Xba1 polymorphisms: X -/-, X-/+ and X +/+ and two of Eco R1 polymorphism: E+/+ and E +/- (Figure 1 and Table II) were found. Mutant alleles were more frequent in HIV-positive (Table II).

Table II: Frequency of Genotypes and Alleles

Genotypes and alleles	Global Frequency (N=16)	Healthy subjects (N=5)	HIV-positive (N=11)
Xba 1 Polymorphism			
Wild genotype (X-/-)	10 (62.50%)	3 (30.00%)	7 (70.00%)
Mutant / wild genotype (X-/+)	5 (31.20%)	2 (40.00%)	3 (60.00%)
Mutant genotype (X+ /+)	1 (6.20%)	0 (0.00%)	1 (100.00%)
Wild Allele (X-)	25 (78.13%)	8 (32.00%)	17 (68.00%)
Mutant Allele (X+)	7 (21.87%)	2 (28.57%)	5 (71.43%)
EcoR 1 Polymorphism			
Wild genotype (E+/+)	12 (75.00%)	3 (25.00%)	9 (75.00%)
Mutant /wild genotype(E+/-)	4 (25.0%)	2 (50.00%)	2 (50.00%)
Mutant genotype (E-/-)	0 (0.00%)	0 (0.00%)	0 (0.00%)
Wild allele E+	28 (87.50%)	8 (28.57%)	20 (71.43%)
Mutant allele E-	4 (12.50%)	2 (50.00%)	2 (50.00%)

The link between the Apo B100 Xba1 and Ecor1 polymorphisms profiles and alleles and the levels of serum lipids showed that normal LDL cholesterolomia and hyperapoproteinemia B100 were predominant (Table III).

Table III: Analysis of the Presence of Alleles and Variations in Apolipoproteinemia B100 and LDL cholesterolemia

Alleles	Apoproteinemia B100		
	Low (< 0.50 g/L)	Normal (0.50 to 0.82 g/L)	High (> 0.82 g/L)
Xba 1 Polymorphism			
Wild allele (X-)	1 (10.00%)	2 (20.00%)	7 (70.00%)
Mutant allele (X+)	0 (0.00%)	1 (16.70%)	5 (83.30%)
EcoR 1 Polymorphism			
Wild allele (E+)	0 (0.00%)	3 (25.00%)	**9 (75.00%)**
Mutant allele (E-)	1 (25.00%)	0 (0.00%)	**3 (75.00%)**
	LDL-C		
	Normal (< 1.60 g/L)		**High (≥ 1.60 g/L)**
Xba 1 Polymorphism			
Wild allele (X-)	9 (90.00%)		1 (10.00%)
Mutant allele (X+)	4 (66.70%)		2 (33.33%)
EcoR 1 Polymorphism			
Wild allele (E+)	9 (75.00%)		3 (25.00%)
Mutant allele (E-)	4 (100%)		0 (0.00%)

DISCUSSION

This cross-sectional study was conducted for the detection of protein and genetic disorders of apoproteins B100 during dyslipidemia in HIV-infected patients. It highlighted genetic mutations previously described as in favor of dyslipidemia.

The study population was predominantly male and young. This double characteristic is one of NBTC voluntary blood donors. Indeed, it has already been demonstrated that most of the voluntary donors in the NBTC were young and male gender[11]. HIV-positive had higher BMI than the controls. This weight gain in HIV-positive was due to a healthy lifestyle. This could be explained by the quality of the medical care given to the patients of this center thanks to its multidisciplinary medical team. Similar results were reported in previous studies [8, 17].

The prevalence of apoprotein abnormalities was higher than that of classic lipid abnormalities. Comparing the two groups, we observed statistically significant decreased apoproteinemia and increased APO B 100/APO A1 ratio in HIV-positive population which are cardiovascular risk factors. This trend is common in the literature. Some studies carried out in ART-naive patients reported high incidence of dyslipidemia with low HDL concentration and hypertriglyceridemia [18]; more currently studies reported high prevalence of dyslipidemia in HIV-positive[8,9,17,19]. The concordance between lipid parameters measurement and apolipoproteins ones showed that apoprotein abnormalities were more sensitive in the search for atherogenic risk than those of classic lipids abnormalities. This result suggested that in lipoprotein abnormalities, apolipoproteins are the first components to be disrupted during HIV infection. In fact, apolipoproteins A1 and B100 are the major proteins transporting blood lipids in high density lipoprotein (HDL) and low density lipoproteins (LDL, V-LDL). So normally, lipids abnormalities should appear after apoproteins ones.

The mutations related to the 2 polymorphisms (Xba1 and Eco R1) were found in both healthy group and HIV-positive group. In our study, there was also mutants for the 2 alleles of the Xba1 polymorphism (X +/+). The study of Jaime SC and al.[20] in Mexico and that of Kodogo *et al*. in Zimbabwé [19] reported the presence of the 3 possible genotypes (wild: X-/-, wild / mutant: X-/+ and mutant : X+/+). However, a study carried out in China with a large population, found no mutants for these two alleles (X+/+) [15]. This difference could be explained by the genetic variability between their study populations and ours. The differences can also be explained by the study population's size. The study done by Scartezini M et al. [21] which investigated the Xba1 and Eco R1 polymorphisms found all possible genotypes (wild, mutant /wild, mutant) while we did not find the mutant genotype for the E alleles (E-/-). This could be explained due to the fact that our population was small, but could also due to genetic variability.

Hyperapoproteinemia B was predominant regardless of the polymorphism (Xba1 or Eco R1) and the allele (mutant or wild), but LDL cholesterolemia was normal in most cases. Concerning the Xba1 polymorphism, the study of Liu *et al*.[15] reported results which were different from ours. In their study, Apoproteinemia B was normal in all cases while LDL cholesterol was significantly higher in mutants / wild (X -/+). It has also been shown that there is a relationship between increased total cholesterolemia, LDL cholesterolemia, serum ApoB100 and portability of the mutant allele (X+) of the Xba1 polymorphism[15,14]. Kallel *et al*.[22] reported that the mutant allele of Xba1 increases serum lipid concentrations. In fact, the meta analysis study of W. Gu *et al*. [23] showed that the 2 genetic variants of apoB (Xba1 and Ecor1) are sometimes associated with serum lipids abnormalities in Chinese and sometimes not. Also, Q-L Gu *et al*. reported that the Apo B gene polymorphism's effect may predispose specifics populations to hyperlipidemia in combination with other genetic or nutritional factors [24].

The limitation of this study is that the molecular characterization was performed on a small subsample. Nevertheless future studies will correct this aspect.

CONCLUSION

This cross-sectional study comparing the two groups of subjects allowed to find that atherogenic risk was more common among HIV-positive. In addition, protein disorders were more frequent than lipid disorders in the search for dyslipidemias. The polymorphisms of the ApoB100 gene (Xba1 and Eco R1) were present in both healthy and HIV-positive subjects. However, their link with dyslipidemia is to be confirmed on a larger workforce.

Acknowledgements

NBTC and DBMC for the participants's recruitment

Declarations

- Funding: This study was conducted at our expense. We did not get any funding.
- Authors's contributions

This work was carried out in collaboration between all authors. Author KF was responsible for the literature searches, wrote the first manuscript of the protocol, performed the data collection, the molecular biology analyses, the data analyses and their interpretation. Then she wrote the first manuscript of the article. Author EAA take care of the literature searches, gave the final approval of the study design of the protocol on the biochemistry side. Author TT provided technical supervision of molecular biology analyses: PCR, electrophoresis and RFLP experiments. Authors ABC performed the RFLP reactions in collaboration with author KF. Authors HAML and AHFT proof read the article. Author DAJ gave the final approval of the study design and protocol on the molecular biology side. He gave the final approval of the article version to be published in collaboration with author MD. Author MD coordinated data collection, biochemical and molecular biological levels of the study. All authors have read and approved the final manuscript.

References

1. Anonymous 1: Onusida-Donnes Pays. Estimation VIH et SIDA. 2015. p1
2. Anonymous 2 : Le prix de l'oubli: des millions de personnes en Afrique occidentale et centrale restent en marge de la lutte mondiale contre le VIH. Médecin sans frontières. 2016. p84.
3. Elaine RDA, Edna MVR, Ana PK, Tamires F and Maria AEW. The Roles of Genetic Polymorphisms and Human

Immunodeficiency Virus Infection in Lipid Metabolism. Bio Med Research International. 2013. 15 pages. http://dx.doi.org/10.1155/2013/836790

4. Myerson M, Malvestutto C and Aberg JA. Management of Lipid Disorders in Patients Living With HIV. The Journal of Clinical Pharmacology. 2015; 55(9):957–974. DOI: 10.1002/jcph.473
5. Zhou DT, Oektedalen O, Shawarira-Bote S and Stray-Pedersen B. Changes in coronary heart disease risk profiles of HIV patients in Zimbabwe over 9 months: a follow-up study. HIV/AIDS - Research and Palliative Care. 2016; 8:165–174. DOI: http://dx.doi.org/ 10.2147%2FHIV.S113206
6. Lo J. Dyslipidemia and lipid management in HIV-infected patients. Curr Opin Endocrinol Diabetes Obes. 2011;18(2):144-147. ttp://www.ncbi.nlm.nih.gov/pmc/articles/PMC3154840/
7. Aberg JA. Lipid Management in Patients Who Have HIV and Are Receiving HIV Therapy. Endocrinol Metab Clin North Am. 2009; 38 (1): 207–222. http://dx.doi.org/10.1016%2Fj.ecl.2008.11.009
8. Tadewos A, Addis Z, Ambachew H and Banerjee S. Prevalence of dyslipidemia among hiv-infected patients using first-line highly active antiretroviral therapy in southern ethiopia: a cross-sectional comparative group study. AIDS res and ther. 2012; 9:31. https://aidsrestherapy.biomedcentral.com/articles/10.1186/1742-6405-9-31
9. Dave JA, Levitt NS, Ross IL et al.Anti-Retroviral Therapy Increases the Prevalence of Dyslipidemia in South African HIV-Infected Patients. PLoSONE. 2016; 11(3) : e0151911.doi:10.1371/journal.pone.0151911
10. Sawadogo M, Sakandé J, Kabré E, and Sougué M. Plasma lipids profile of non-treated HIV infected adults in Ouagadougou (Burkina Faso). Ann Biol Clin (Paris). 2005; 63(5):507-12.
11. Yayo SE, Hauhouot-Attoungbre ML, Yapi HF et al. Apport du protidogramme sérique dans la sélection de donneurs de sang à Abidjan. Immuno-analyse & Biologie Spécialisée. 2009; 24(1), 32-36
12. Biswas S, Ghoshal PK, Halder B et al. Apolipoproteins AI/B/E gene polymorphism and their plasma levels in patients with coronary artery disease in a tertiary care-center of Easter. Ind. hea. J. 2013; 6 5: 658-665
13. Qiagen QIAmp DNA Mini Kit and QIAmp DNA Blood Mini Kit. 2010; 04/2010, 3e EDITION
14. Dixit M, Choudhuri GS and Mittal B. Association of apolipoprotein A1-C3 gene cluster polymorphisms with gallstone disease. Can J Gastroenterol. 2007; 21: 137-144
15. Liu FL, Lu WB, andNiu WX. XbaI polymorphisms of apolipoprotein B gene: Another risk factor of gallstone formation after radical gastrectomy. World J Gastroenterol. 2010; 16 (20): 2549-2553
16. Bogari N, Abdel-Fadel AM, Hassan M and Fawzy A. Apolipoprotein B (XbaI) allele frequencies in an Egyptian Population: impact on blood lipids. Int J Biol Med Res. 2014; 5(2):3981-3987. https://www.researchgate.net/profile/Neda_Bogari/publication/284183779.
17. Nsagha DS, Weledji EP, Assob NJC et al. Highly active antiretroviral therapy and dyslipidemia in people living with HIV/AIDS in Fako Division, South West Region of Cameroon. Cardiovascular Disorders. 2015; 15:95-102
18. Kanjanavanit S, Puthanakit T, Vibol U et al.Prevalence of lipid abnormalities among Antiretroviral naïve HIV-infected Asian children with mild to moderate Immunosuppression. Antivir Ther.2011; 16 (8):1351–1355. http://dx.doi.org/10.3851%2FIMP1897
19. Kodogo V, Zhou DT, Oektedalen O et al. Apolipoprotein B Gene Polymorphisms and Dyslipidemia in HIV Infected Adult Zimbabweans. The Open AIDS Journal. 2016; 10, 190-198. http://dx.doi.org/10.2174%2F1874613601610010190
20. Jaime SC, Maribel AM, Eliakym AM et al. ApoB-100, ApoE and CYP7A1 gene polymorphisms in Mexican patients with cholesterol gallstone disease. World J Gastroenterol. 2010; 16 (3): 4685-4690. http://dx.doi.org/10.3748%2Fwjg.v16.i37.4685
21. Scartezini M., Zago MA, Chautard-Freire-Maia EA et al. The X-X-/E+E+ genotype of the XbaI/EcoRI polymorphisms of the apolipoprotein B gene as a marker of coronary artery disease in a Brazilian sample. Braz Med Biol Res. 2003; 36 (3):369-375. http://www.scielo.br/scielo.php?pid=S0100-879X2003000300012&script=sci_arttext&tlng=pt
22. Kallel A, Jemaa R, Feki M et al. Polymorphisme XbaI du gène de l'apolipoprotéine B dans une population tunisienne: fréquences alléliques et relation avec les paramètres lipidiques plasmatiques. Ann Biol Clin. 2007;65:265-70. http://europepmc.org/abstract/med/17502298
23. Gu W, Mingduo Z and Shaojun W. Lipids in Health and Disease (2015) 14:123 DOI 10.1186/s12944-015-0125-z
24. Gu QL, Han Y, Lan YM et al. Association between polymorphisms in the APOB gene and hyperlipidemia in the Chinese Yugur population. Brazilian Journal of Medical and Biological Research 2017; 50(11): e6613, http://dx.doi.org/10.1590/1414-431X20176613 ISSN 1414-431X

How to cite this article:

Koné F *et al*.2018, Xba 1 and Ecor 1 Polymorphisms of Apolipoprotein B100 Gene and Lipids Abnormalities in Hiv Infected Patients. *Int J Recent Sci Res.* 9(9), pp. 28854-28858. DOI: http://dx.doi.org/10.24327/ijrsr.2018.0909.2746

****