

## ORIGINAL ARTICLE

# Distribution of polymorphism rs693 of ApoB gene in a sample of Colombian Caribbeans

## Distribución del polimorfismo rs693 en el gen de la apolipoproteína B en una muestra caribeños colombianos

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### Abstract

#### Introduction:

Several studies have reported that the single nucleotide polymorphism rs693 of Apo lipoprotein B gene is associated with high levels of plasma lipids and high body mass index, which are risk factors for cardiovascular diseases. The distribution of this single nucleotide polymorphism and its association with the phenotype depend on the genetic background of each population.

#### Objective:

To evaluate the distribution of single nucleotide polymorphism rs693 and its association with lipid profile and body mass index in a sample of Colombian Caribbeans.

#### Methods:

108 non-related adult subjects of both gender were included in this study. Body mass index and lipid profile that included total cholesterol, triglycerides, Low Density Lipoprotein and High Density Lipoprotein were determined. The single nucleotide polymorphism rs693 was determined by Polymerase Chain Reaction/Restriction Fragment Length Polymorphism from genomic DNA followed by digestion with the restriction enzyme XbaI. The chi-square test was used to analyze the genotype distribution of rs693 and the genotype-phenotype association was evaluated through different inheritance model.

#### Results:

The genotype frequencies for single nucleotide polymorphism rs693 were CC (45.0%), TT (16.5%) and CT (38.5%). The allele frequencies were C (64.0%) and T (36.0%). The single nucleotide polymorphism was in Hardy-Weinberg equilibrium in the studied sample. No association of the single nucleotide polymorphism rs693 with lipid profile nor the body mass index was found ( $p > 0.05$ ).

#### Conclusion:

There is no significant association between single nucleotide polymorphism rs693 and body mass index nor lipid profile, in a sample of Colombian Caribbeans.



#### OPEN ACCESS

**Citation:** Mendoza-Torres E, Pereira SNS, Villarreal CJL, Mendoza SX, De La Espriella PC, Varela PLL, Villanueva TDA. Distribution of polymorphism rs693 of ApoB gene in a sample of Colombian Caribbeans Colomb Med (Cali). 2019; 50(3): 153-62. <http://dx.org/1025100/cm.v50i3.4048>

**Received:** 30 Oct 2018

**Revised:** 15 Mar 2019

**Accepted:** 13 Jul 2019

#### Keywords:

Apolipoprotein B, lipid profile, body mass index, polymorphism, genotyping

#### Palabras clave:

Apolipoproteína B, perfil lipídico, índice de masa corporal, polimorfismo, genotipificación.

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### Conflicts of interest:

The authors declare that there is not a conflict of interest

### Acknowledgements:

The authors extend their thanks to Laboratorio Clínico Continental from Barranquilla, Colombia, for the determination of tests lipid profile. Roche Colombia for their support with the reagents and equipment to determine the parameters of the lipid profile.

### Funding:

The study was funded by Universidad Libre Barranquilla, Colombia (CONV-INT2011-P001). N.S.P.S. held a Young researcher grant from COLCIENCIAS, Colombia with contract number PS-2012-000353. E.M.T held a PhD scholarship from COLCIENCIAS 55248166 PDBCEX

## Resumen

### Introducción:

Varios estudios han informado que el polimorfismo de un solo nucleótido rs693 del gen de la apolipoproteína B se asocia con altos niveles de lípidos plasmáticos e índice de masa corporal, los cuales son factores de riesgo para enfermedades cardiovasculares. La distribución de este polimorfismo y su asociación con el fenotipo dependen del antecedente genético de cada población. La población caribeña colombiana es producto de la mezcla de tres grupos étnicos principales: africano, amerindio y caucásico.

### Objetivo:

Evaluar la distribución del polimorfismo rs693 y su asociación con el perfil lipídico y el índice de masa corporal en una muestra de sujetos caribeños colombianos.

### Métodos:

Fueron incluidos en este estudio 108 sujetos adultos de ambos sexos y no relacionados. Se determinaron el índice de masa corporal y el perfil lipídico; de éste se incluyó colesterol total, triglicéridos, lipoproteínas de baja densidad y lipoproteína de alta densidad. El polimorfismo rs693 se determinó mediante Reacción en Cadena de la Polimerasa del ADN genómico seguida por digestión con la enzima de restricción XbaI. Se utilizó la prueba de ji cuadrado para analizar la distribución del genotipo de rs693 y se evaluó la asociación genotipo-fenotipo a través de diferentes modelos de herencia.

### Resultados:

Las frecuencias genotípicas para rs693 fueron CC (45.0%), TT (16.5%) y TC (38.5%). Las frecuencias alélicas fueron C (64.0%) y T (36.0%). El polimorfismo rs693 estaba en equilibrio de Hardy-Weinberg en la muestra estudiada y no presentó asociación con el perfil lipídico ni con el índice de masa corporal ( $p > 0.05$ ).

### Conclusión:

No existe asociación significativa del polimorfismo rs693 con el índice de masa corporal ni con el perfil lipídico en una muestra de caribeños colombianos.

## Remark

### 1) Why was this study conducted?

The present study was done to explore the association of SNP C/T7673 with dyslipidemia and overweight/obesity in a sample of the Colombian Caribbean population. The development of this study contributed to the field of potential biomarkers of risk of cardiovascular disease (CVD) in the Colombian population.

### 2) What were the most relevant results of the study?

The distribution of SNP C/T7673 is in Hardy-Weinberg equilibrium in the studied population and the genotypic frequencies are similar to those reported by studies conducted in other Colombian populations and Latin American countries. Finally, it is suggested that the SNP C/T7673 does not influence the lipid profile and BMI in the studied population.

### 3) What do these results contribute?

In Colombia, there are few studies on the possible association of polymorphism C/T7673 with dyslipidemias and overweight/obesity. These results contribute to new knowledge of biomarkers to identify the risk of CVD.

## Introduction

Cardiovascular diseases (CVD) are recognized as the leading cause of death worldwide; with a percentage of 30%, equal to that reported for Colombia<sup>1,2</sup>. Obesity and dyslipidemia are some of the main CVD risk factors<sup>3-5</sup>, the respective indicators are the Body Mass Index (BMI) and lipid profile. The determination of these indicators is important for the prevention, diagnosis and monitoring of cardiovascular disease in an individual<sup>6-8</sup>.

Lipid profile comprises determining triglycerides (TG), total cholesterol (TC), cholesterol linked to low density lipoproteins (LDL-C, Low Density Lipoprotein-Cholesterol) and cholesterol linked to High Density Lipoprotein (HDL-C, High Density Lipoprotein-Cholesterol)<sup>7,8</sup>. The protein component (apoprotein) plays a pivotal role in the structure and function of the lipoproteins; therefore, variations in the gene encoding the apoprotein can cause structural or functional effects<sup>9-10</sup>. Apolipoprotein B (ApoB) is the main protein component of LDL, VLDL (Very Low Density Lipoprotein) and chylomicrons<sup>11</sup>. ApoB is essential for the assembly and secretion of VLDL in the liver and has the domain to bind LDL to its receptor; therefore, it is the key for the transport and metabolism of lipids<sup>12,13</sup>. ApoB is encoded by a gene of 43 kpb located on the short arm of chromosome 2 (2p24) and comprises 29 exons and 28 introns<sup>14,15</sup>. This gene is polymorphic<sup>16,17</sup> and some polymorphisms are related to dyslipidemia<sup>18-21</sup>.

One of the most studied polymorphisms has been the single nucleotide polymorphism (SNP) rs693 also called XbaI and C/T<sub>7673</sub><sup>22</sup>. It is located in exon 26 of the apoB gene at codon 2488 and results from the substitution of cytosine by thymine at the third position (ACC → ACT), creating a silent mutation, as both codons encoding threonine. The presence of thymine generates the restriction site and gives the name to the resulting T allele, also known as X+ allele. However, in the absence of T allele, it is identified as C or X- allele<sup>23</sup>.

Although the SNP rs693 does not imply change in the amino acid sequence of the protein, it is associated with dyslipidemia, obesity and cardio-cerebrovascular diseases in populations from Brazil<sup>23</sup>, China<sup>24</sup> and European countries<sup>25</sup>. The association would favor the possibility of using the SNP rs693 as a predictor of risk of these diseases in the indicated populations<sup>23</sup>. However, in other populations, very low association has been found, for example, in Northern India<sup>26</sup>.

The genotypic and allelic distributions of rs693 in the Colombian population are not completely known. Historically, this population has been regarded as the mixture of three main ethnicities: Africans, Amerindians, and Caucasians<sup>27,28</sup>. In Andean population from Armenia-Colombia, Loango *et al.*<sup>29</sup>, evaluated the association between this polymorphism with plasmatic lipids in children and their parents. However, the admixture between the three ethnicities has different extent in each region of Colombia. This is why, the study of rs693 in a sample of Colombian Caribbean population is justified<sup>28</sup>. Furthermore, because clinical implications of the polymorphism rs693 on the cardiovascular risk factors are not universal, it is important to explore this variant in different populations<sup>25</sup>. The present study aims to determine the association of apoB rs693 polymorphism with the lipid profile and BMI in a sample of the Colombian Caribbean population.

## Materials and Methods

### Study group

This analytical cross-sectional study was performed from an initial group of 331 unrelated adult subjects, both gender, different ages and schooling and born in the Colombian Caribbean like their ancestors to the third degree of consanguinity. After applying a questionnaire and performing a medical interview at Universidad Libre from Barranquilla, smokers, vegetarians, pregnant women, diabetics and all those who were or had been under pharmacological treatment for cardio-cerebrovascular disease, dyslipidemia, hypertension, cancer, liver disease, endocrine disorders or kidney disease were excluded. Thus, the initial group was reduced to 108 subjects (mean age 52 ± 11 years), 34 men (31.5%) and 74 women (68.5%).

The study was approved by the local ethics committee at Universidad Libre from Barranquilla and informed written consent was obtained from all study subjects before their participation.

### Anthropometric measurements and lipid profile

Anthropometric measurements included the recording of weight and height by standard methods. Body Mass Index (BMI) was determined according to the traditional formula of Quetelet: weight in kilograms divided by the square of the height ( $\text{Kg/m}^2$ ). The criteria of the clinical guidelines of the NIH-USA were taken into account to classify the subjects according to the BMI into two categories: normal weight (values range from 18.5 to 24.99  $\text{Kg/m}^2$ ) and overweight/obesity (values  $\geq 25 \text{ Kg/m}^2$ )<sup>8</sup>.

For biochemical studies, a blood sample was drawn by venipuncture from an antecubital vein, following an overnight fast, from each subject under study. Serum was collected from all samples after centrifugation at room temperature. Serum levels of total cholesterol (TC), triglycerides (TGC), HDL cholesterol (HDL-C) and LDL cholesterol (LDL-C) were determined by using COBAS C501 equipment (Roche®, Switzerland), following the manufacturer's instructions.

### Genotyping of the SNP rs693

The genomic DNAs of all the subjects were extracted from 600  $\mu\text{L}$  of whole blood with Wizard® Genomic Kit (Promega®, USA) following the manufacturer's instructions. The genotyping of the SNP rs693 was done by Polymerase Chain Reaction (PCR) followed by digestion with restriction enzyme XbaI. The DNA fragment containing the SNP rs693 was amplified using a sense primer (5' GGAGACTATTCAGAAGCTAA 3') and an antisense primer (5' GAAGAGCCTGAAGACTGACT 3'). The PCR reaction was performed in a PTC-100 (MJ Research®Canada) thermocycler, in a final volume of 25  $\mu\text{L}$  containing 25 mM of each primer, 5  $\mu\text{L}$  of DNA and master mix MangoMix (Bioline®, England) which provided  $\text{MgCl}_2$  2.5 mM and 200 mM dNTPs. The PCR conditions consisted of an initial denaturation step at 95° C for 10 min, followed by 30 cycles of denaturation at 95° C for 1 min, annealing at 49° C for 1 min, and extension at 72° C for 1 min, with a final elongation at 72° C for 8 min. The PCR product was a 710 bp band which was digested with the enzyme XbaI (New England Biolabs®USA) following the instructions of the enzyme manufacturer. The digestion products were then visualized by gel electrophoresis with polyacrylamide 8% and staining with ethidium bromide. When the T allele was present in the restriction site, two bands (433 bp and 277 bp) were obtained. If after the digestion a single band of 710 bp was obtained it was interpreted as CC genotype; if three bands (433 bp, 710 bp and 277bp) were obtained it was interpreted as CT genotype and if two bands (433 bp and 277 bp) were obtained it was interpreted as TT genotype.

### Quality control

The quality of the biochemical tests was verified using commercial serum, normal and abnormal controls (Roche, Switzerland). Reagent controls were used in each amplification to monitor contamination in the molecular analysis. Genotyping was confirmed by repeating at random 40% of the analyzes performed.

### Statistical analysis

The Statistical Package for Social Studies SPSS version 20.0 was used to conduct all data analyses. The total population was grouped according to the values of the lipid profile (subjects with normal and abnormal levels) and the BMI (normal weight and overweight/obese). The data obtained are presented as mean  $\pm$  SD or percentages. Student's t-test was used to compare means and Chi-square test was applied to compare proportions.

**Table 1.** Anthropometric characteristics and lipid profile of the subjects under study.

Variable	Gender		(n=108) Mean $\pm$ SD
	Female (n=74) Mean $\pm$ SD	Male (n=34) Mean $\pm$ SD	
Age (year)	53 $\pm$ 10	50 $\pm$ 11	52 $\pm$ 11
Height (m)	1.61 $\pm$ 0.07	1.71 $\pm$ 0.07	1.64 $\pm$ 0.08
Weight (kg)	66.71 $\pm$ 9.51	79.97 $\pm$ 13.54	70.89 $\pm$ 12.51
Total cholesterol (mg/dL)	190.35 $\pm$ 40.74	178.00 $\pm$ 25.86	186.46 $\pm$ 37.04
High Density Lipoprotein-Cholesterol (mg/dL)	47.16 $\pm$ 12.02	42.85 $\pm$ 9.66	45.81 $\pm$ 11.47
Low Density Lipoprotein-Cholesterol (mg/dL)	119.09 $\pm$ 34.98	117.88 $\pm$ 23.12	118.71 $\pm$ 31.62
Triglycerides (mg/dL)	158.91 $\pm$ 128.72	131.74 $\pm$ 58.76	150.35 $\pm$ 111.94
Body mass index ( $\text{Kg/m}^2$ )	25.74 $\pm$ 3.50	27.25 $\pm$ 4.00	26.21 $\pm$ 3.72

Total cholesterol in men)  $\geq 170$ ; in women)  $\geq 180$

Triglycerides  $\geq 150$ ;

High Density Lipoprotein-Cholesterol in men  $< 40$ ; in women  $< 50$

Low Density Lipoprotein-Cholesterol  $\geq 100$  were considered abnormal values

**Table 2.** Frequency of abnormal body mass index and lipid profile with respect to the gender.

Variables	Gender	
	Female n=74 (%)	Male n=34 (%)
Body mass index	40 (54.1)	23 (67.6)
Triglycerides	26 (35.1)	8 (23.5)
Total Cholesterol	44 (59.5)	23 (67.6)
High Density Lipoprotein-Cholesterol	38 (51.4)	18 (52.9)
Low Density Lipoprotein-Cholesterol	53 (71.6)	27 (79.4)

**Table 3.** Comparison of the genotypic and allelic frequencies in the present study with other Latin American populations.

Genotype	Colombian Caribbeans (present study) n= 108	Brazilian population <sup>23</sup> n= 192	Mexican population <sup>31</sup> n=246	Colombian Andeans from Armenia <sup>29</sup> n= 148
CC 7673	45.0%	33%	47%	28%*
CT 7673	38.5%	50%	38%	65%*
TT 7673	16.5%	17%	15%	7%
<b>Alleles</b>				
C 7673	65.0%	58%	66%	61%
T 7673	35.0%	42%	34%	39%

\*p <0.05 compared to Caribbeans-Colombia (present study).

The genotype frequencies and the Hardy-Weinberg were determined with Arlequin version 3.11 software 30. The data were further grouped according to the three genotypes of the SNP rs693. The chi-square test was used to analyze the distribution of the rs693 genotype and the genotype-phenotype association was evaluated through different inheritance models: co-dominant, dominant, recessive and additive. A value of  $p < 0.05$  was considered statistically significant.

### Ethics approval and consent to participate

The ethical approval for this study was obtained from local ethics committee at Universidad Libre from Barranquilla. Written informed consent was obtained from all study subjects before their participation.

## Results

### Anthropometric characteristics and lipid profile

The anthropometric characteristics and the lipid profile of the study subjects classified by gender are presented in Table 1. When comparing means with Student's t-test, there was statistically significant difference between the average weight ( $p = 0.007$ ) and height ( $p = 0.008$ ) between men and women; but not difference was found between the mean age ( $p = 0.06$ ). The average body mass index (n= 108) was  $26.21 \pm 3.72$  Kg/m<sup>2</sup> and no statistically significant difference was found between the average BMI of women ( $25.74 \pm 3.50$  Kg/m<sup>2</sup>) and men ( $27.25 \pm 4.00$  Kg/m<sup>2</sup>), ( $p = 0.3435$ ).

With regard to the lipid profile, the averages of the parameters evaluated in all individuals are presented in Table 1. The subjects were grouped according to National Lipid Association recommendations<sup>7</sup>. After using Chi-square test, significant differences were not found between men and women when comparing the corresponding frequencies for each parameter of the lipid profile ( $p > 0.05$ ) (Table 2).

### Genotype and allele frequencies of SNP rs693

Of the 108 genotyped subjects, 49 (45.0%) presented CC genotype; 41 (38.5%) presented CT genotype and the remaining 18 (16.5%) presented TT genotype. These data show that the C allele was majority; in fact, the allele frequencies were 139 (64.0%) for C allele and 77 (36.0%) for the T allele. The distribution of the genotype frequencies was in Hardy-Weinberg equilibrium ( $p > 0.05$ ). Genotype and allele frequencies were compared with other Latin American populations (Table 3).

### Genotype frequencies regarding lipid profile and BMI

The genotype frequencies with respect to lipid profile and BMI are shown in table 4. None of the inheritance models showed a significant increase in the odds of alterations in BMI or lipid profile parameters (Table 4). There were also no significant differences in these outcomes when gender and age variables were included in these models.



**Table 4.** Analysis of the association between the genotype and alterations in the lipid profile and BMI, depending on the inheritance model\*.

Variable (N=108)	Model	Genotype	n	%	OR	CI (95%)
Body Mass Index (n=63)	co-dominant	CC7673	26	41.3	1	
		CT7673	26	41.3	0.712	0.321-1.579
		TT7673	11	17.5	0.871	0.309-2.453
	Dominant	CC7673	26	41.3	1	
		CT7673/ TT7673	37	58.7	0.672	0.311-1.452
	Recessive	CC7673/ CT7673/ TT7673	52	82.5	1	
			11	17.5	0.871	0.309-2.453
	additive				0.719	0.239-2.164
Triglycerides (n=34)	co-dominant	CC7673	13	38.2	1	
		CT7673	16	47.1	0.574	0.251-1.314
		TT7673	5	14.7	1.236	0.402-3.797
	dominant	CC7673	13	38.2	1	
		CT7673/ TT7673	21	61.8	0.653	0.285-1.496
	recessive	CC7673/ CT7673	29	85.3	1	
		TT7673	5	14.7	1.236	0.402-3.797
	additive				0.939	0.280-3.151
Total Cholesterol (n=67)	co-dominant	CC7673	30	44.8	1	
		CT7673	29	43.3	0.542	0.237-1.241
		TT7673	8	11.9	2.379	0.852-6.639
	dominant	CC7673	30	44.8	1	
		CT7673/ TT7673	37	55.2	0.939	0.430-2.048
	recessive	CC7673/ CT7673	59	88.1	1	
		TT7673	8	11.9	2.379	0.852-6.639
	additive				1.974	0.662-5.888
High Density Lipoprotein-Cholesterol (n=56)	co-dominant	CC7673	25	44.6	1	
		CT7673	24	42.9	0.648	0.295-1.419
		TT7673	7	12.5	1.878	0.667-5.284
	dominant	CC7673	25	44.6	1	
		CT7673/ TT7673	31	55.4	0.941	0.441-2.008
	recessive	CC7673/ CT7673	49	87.5	1	
		TT7673	7	12.5	1.878	0.667-5.284
	additive				1.637	0.544-4.921
Low Density Lipoprotein-Cholesterol (n=80)	co-dominant	CC7673	33	41.3	1	
		CT7673	34	42.5	0.451	0.172-1.182
		TT7673	13	16.3	1.12	0.360-3.486
	dominant	CC7673	33	41.3	1	
		CT7673/ TT7673	47	58.8	0.527	0.220-1.258
	recessive	CC7673/ CT7673	67	83.8	1	
		TT7673	13	16.3	1.12	0.360-3.486
	additive				0.793	0.241-2.612

\* Data are presented as frequencies and percentages of subjects with abnormal values. No association of the single nucleotide polymorphism rs693 with lipid profile nor the body mass index was found ( $p > 0.05$ ).

## Discussion

Polymorphisms in genes associated with dyslipidemia or obesity may be genetic predictors of CVD in populations where the association is obvious<sup>21-24</sup>. In this study, the genotypic and allelic frequencies were determined for the SNP rs693 such as its relationship with lipid profile and BMI in a sample of the Colombian Caribbean population, a product of the ancestral mixture of African black, Amerindian and European white<sup>28</sup>.

Genotypic analysis of polymorphism revealed: first, that in the sample studied the distribution of genotypes was not significantly different from the expected distribution for a population in Hardy-Weinberg equilibrium; second, that all possible genotypes for rs693 (TT, TC and CC) were present; and third, that the TT genotype was minority. Furthermore, the allelic analysis revealed that C was majority (64%) versus T (36%).

The T allele frequency found in this study is in agreement with those reported in a sample of mestizo Colombian Andean population from Armenia-Colombia<sup>29</sup>, in a sample of Brazilian people<sup>23</sup>, and in a sample of Mexican people<sup>31</sup>. However, there are notable differences with respect to the allelic frequencies found in European, Asian and African populations. In European populations, the frequency of T allele is higher (~53%) (32-35), except Norwegians (~27%)<sup>32</sup>, while it is lower in Asians (~12%)<sup>36-39</sup> and Africans (~21%)<sup>40-42</sup>.

Distribution of genotypes in Colombian Caribbeans differed from that of Colombian Andeans from Armenia<sup>29</sup>. The frequencies of CT and CC were higher in Colombian Andeans and a significant difference was found when the genotype distribution was compared between these two populations. However, no significant differences in genotype frequencies were observed when comparing Colombian Caribbeans to other Latin American populations (Brazilians and Mexicans)<sup>23,31</sup> (Table 3). This phenomenon is typical in Colombian population and is the product of historical events that gave rise to it<sup>43</sup>. The admixture between European, African, and Amerindian populations has a different extent in each region of Colombia. In central and Eastern Colombia, including Armenia, European and Amerindian ancestry predominate; in coastal regions, including Caribbean population in the present study, European, Amerindian and African ancestors all significantly contributed to this population<sup>44</sup>.

On the other hand, in the sample studied, the SNP rs693 does not appear to influence plasma lipid levels. Indeed, there was no significant association with the TT and CT genotypes or the mutant allele T<sub>7673</sub>;  $p$ -value was always  $>0.05$ . This finding agrees with that reported in other Colombian study<sup>29</sup>, when they did not find statistically significant differences (TC  $p=0.47$ , HDL-C  $p=0.23$ ; LDL-C  $p=0.40$ ; TG  $p=0.10$ ). It is also in agreement with the findings in a sample of the Brazilian population<sup>26</sup>, and in Indians<sup>45</sup>; in these cases, in all comparisons  $p>0.05$ .

By contrast, our results differ from the findings in Egyptians<sup>46</sup>, who reported association of the T allele with increased cholesterol levels ( $p=0.041$ ) and LDL-C ( $p=0.021$ ); in a population of Mongolia<sup>38</sup>, when found significant association with triglyceride levels since these were significantly higher in men with genotype CT<sub>7673</sub> than in men with CC genotype ( $p=0.047$ ); in China<sup>37</sup> that CT genotype carriers tend to have high LDL levels, but low levels of HDL-C ( $p<0.05$ ) and, finally, the results also in China<sup>47</sup>, when reported that subjects with T allele exhibited significantly higher levels of LDL-C, TC and TG, compared to subjects with C allele ( $p<0.05$ ).

For BMI, our results showed no significant association with genotypes nor with the mutant T allele ( $p>0.05$ ). This result agrees with that reported in Colombian mestizos<sup>29</sup>, found no significant differences ( $p=0.08$ ) among subjects with T allele and subjects with C allele. Our study agrees with Egyptian subjects<sup>46</sup>, who found no significant differences in BMI of with TT, CT and CC genotypes and the lack of association ( $p>0.05$ )<sup>26</sup>. However, our result is discordant with the findings in China, who found significantly higher values ( $p<0.05$ ) in subjects with T allele than in subjects with C allele<sup>47</sup>.

In this study the genotype-phenotype association with respect to wild-type C allele (X-) was also studied. Unlike the findings about positive association of this allele with coronary artery disease and myocardial infarction<sup>23,48</sup>, in this study no association ( $p>0.05$ ) was found.

The similarity of our results with those of another study conducted in Colombia, in a far and different geographical area leads us to infer that the characteristic ancestral mixture of the two samples studied was the key factor to reproduce the result, despite environmental factors and lifestyle.

Environmental factors, however, can contribute to differences that are mainly due to ethnic variability. Indeed, biochemical phenotypes such as levels of plasma lipids, anthropometric parameters such as weight, demographic variables such as gender and age, environmental conditions such as food or cigarette smoking, and specific factors of the study as the sample size or the criteria for inclusion and exclusion, can influence the differences between the results of this study and others already referenced.

In this study, the wide age range of subjects, the differences in lifestyle and the sample size constituted limitations. However, the findings of this exploratory work should be validated in the future, with a study of a representative sample of the population of the Colombian Caribbean, under the case-control design.

## Conclusion

This study suggests that there is no influence of the polymorphism on lipid profile parameters or on BMI.

## Author's contributions:

E.M.T and N.S.P.S designed and carried out all the genetic experiments. E.M.T., X.M.S. and D.A.V.T prepared the tables and drafted the manuscript. C.DeLaE.P carried out the determination of the lipid profile. E.M.T. and X.M.S performed all the statistical analysis. J.L.V.C. and L.L.V.P reviewed the manuscript. All authors read and approved the final manuscript.

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