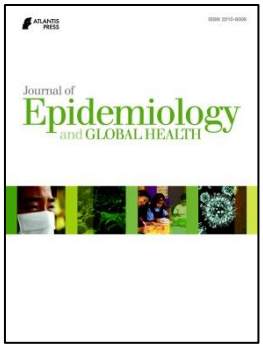

ATLANTIS PRESS

Journal of Epidemiology and Global Health

ISSN (Online): 2210-6014 **ISSN (Print): 2210-6006**
Journal Home Page: https://www.atlantis-press.com/journals/jegh

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To cite this article: Muzamil M. Abdel Hamid, Safa Ahmed, Awatif Salah, Etayeb M.A. Tyrab, Lemya M. Yahia, Elbagire A. Elbashir, Hassan H. Musa (2015) Association of lipoprotein lipase gene with coronary heart disease in Sudanese population, Journal of Epidemiology and Global Health 5:4, 405–407, DOI:

https://doi.org/10.1016/j.jegh.2015.04.007

To link to this article: https://doi.org/10.1016/j.jegh.2015.04.007

Published online: 23 April 2019

Journal of Epidemiology and Global Health (2015) 5, 405–407

http:// www.elsevier.com/locate/jegh

Association of lipoprotein lipase gene with coronary heart disease in Sudanese population

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Received 30 September 2014; received in revised form 14 April 2015; accepted 19 April 2015
Available online 27 May 2015

KEYWORDS
Risk factors;
Lipid profile;
LPL gene;
Coronary heart disease;
Sudan

Abstract Cardiovascular disease is stabilizing in high-income countries and has continued to rise in low-to-middle-income countries. Association of lipid profile with lipoprotein lipase gene was studied in case and control subject. The family history, hypertension, diabetes mellitus, smoking and alcohol consumption were the most risk factors for early-onset of coronary heart disease (CHD). Sudanese patients had significantly ($P < 0.05$) lower TC and LDL-C levels compared to controls. Allele frequency of LPL D9N, N291S and S447X carrier genotype was 4.2%, 30.7% and 7.1%, respectively. We conclude that lipoprotein lipase polymorphism was not associated with the incidence of CHD in Sudan.

1. Introduction

LPL gene, Asp9Asn, Asn291Ser, and S447X are the most important mutations described because of their greater frequency and influence on susceptibility to atherosclerosis [1]. The LPL D9N and LPL N291S variants have been associated with an adverse lipid profile, but the association with cardiovascular disease has been less consistent [2]. D9N and N291S have been associated in a meta-analysis with an increase in triglycerides of 20% and 31%, respectively [3], and S447X was

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http://dx.doi.org/10.1016/j.jegh.2015.04.007

Table 1 Associations of LPL genotypes with lipid profiles in Sudanese patient carriers of D9N, N291S and S447X genotypes compared with non-carrier controls.

Items	D9N			N291S			S447X		
Genotype frequency	Carrier *n* (%)	Non-carrier *n* (%)	*P* value	Carrier *n* (%)	Non-carrier *n* (%)	*P* value	Carrier *n* (%)	Non-carrier *n* (%)	*P* value
Case	3(0.05)	62(0.95)		22(0.30)	51(0.67)		8(0.15)	46(0.85)	
Control	3(0.04)	75(0.96)		17(0.32)	37(0.69)		8(0.14)	51(0.86)	
All	6(0.04)	137(0.96)		39(0.31)	88(0.69)		16(0.14)	97(0.86)	
Allele frequency	(%)	(%)		(%)	(%)		(%)	(%)	
Case	0.05	0.95		0.30	0.70		0.07	0.93	
Control	0.04	0.96		0.32	0.69		0.07	0.93	
All	0.04	0.96		0.31	0.69		0.07	0.93	
Lipid	Carrier (*n* = 2)	Non-carrier (*n* = 49)	*P* value	Carrier (*n* = 14)	Non-carrier (*n* = 26)	*P* value	Carrier (*n* = 5)	Non-carrier (*n* = 36)	*P* value
TC	133.50 ± 76.74	254.51 ± 15.50	0.129	173.43 ± 14.59	194.35 ± 10.70	0.255	173.80 ± 45.65	254.97 ± 17.01	0.104
TG	230.00 ± 115.19	300.47 ± 23.27	0.551	240.64 ± 36.34	229.19 ± 26.66	0.801	266.80 ± 71.65	283.19 ± 26.70	0.831
LDL	40.10 ± 69.89	146.50 ± 14.12	0.142	76.79 ± 12.77	105.05 ± 9.37	0.083	77.38 ± 39.53	152.19 ± 14.73	0.084
HDL	47.65 ± 26.13	48.83 ± 5.28	0.965	48.51 ± 4.13	41.65 ± 3.03	0.188	43.06 ± 18.55	47.39 ± 6.91	0.828
VLDL	46.00 ± 22.41	60.91 ± 4.53	0.517	48.13 ± 7.00	48.22 ± 5.14	0.992	53.36 ± 14.03	57.67 ± 5.23	0.775

The term carrier is used to refer to the sum of both homozygote and heterozygote for those polymorphisms. D9N G > A, N291S A > G, S447X C > G.
All data shown are means ± SE (mg/dl).

associated with reduced plasma triglyceride and increasing HDL-C [2]. The study aimed to determine risk factors and the association of lipid profiles with LPL gene in patients with coronary artery disease and healthy Sudanese population.

2. Materials and Methods

This case control study was designed to study risk factors, lipid profile in CHD patients and their association with lipoprotein lipase gene in Sudan. Informed consent was obtained from all participants. Detailed demographic and risk factors for CVD were collected using a structured questionnaire.

Lipids were analyzed by MINDRAY BS-200 analyzer (MINDRAY, Shenzhen, China). Genomic DNA was extracted from blood by kits and PCR-RFLP was applied to detect D9N, N291 and S447X lipoprotein lipase genotype, using TaqI, RsaI and Mnl1 restriction enzyme, respectively. Statistical analyses were performed using SPSS v.18.

3. Results and Discussion

Among the population 53.1% were male, 22% had family history of CHD, 42.6% hypertension, 41.6% diabetes, 18.2% smoking and 5.3% alcohol. The low smoking and alcohol consumption may be due to cultural denial of smoking and alcohol in our community especially among females [4]. Patients show lower TC and LDL-C levels compared to controls. African ancestry was significantly associated with decreased TC, LDL and triglycerides [4].

Allele frequency of LPL D9N, N291S and S447X carrier was 4.2%, 30.7% and 7.1%, respectively (Table 1). The carrier of frequency of N291S was ranging from 2% to 5% in different populations [5]. While for S447X was 18% in patients with CAD and 23% in the control [1]. In Tunisian population the frequency of p.Asp9Asn variation was 10.37% in CAD patients versus 3.66% in controls, and for p.Ser447X was 8.8% in CAD patients versus 13.7% in controls [2]. No significant ($P < 0.05$) association in lipid profiles was found between carriers (patient) and non-carriers (control) of D9N, N291S and S447X genotype (Table 1). D9N and N291S variants were associated with higher plasma TG [3]. The S447X variant was associated with lower TG and higher HDL, and lower risk of CHD [2]. In healthy Tunisian population, heterozygote carriers of the p.Asp9Asn substitution had a significant increase of total cholesterol and a decrease of HDL [2].

4. Conclusion

Heart diseases are prevalent in Sudan and have similar risk factors as elsewhere. The lipoprotein lipase polymorphism was not associated with the incidence of CHD in Sudan.

Conflict of interests

No conflict of interests.

Acknowledgement

This study was supported by grant from the Ministry of Higher Education, Sudan.

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