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

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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.

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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
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          | Genotype frequency | D9N          | N291S         | S447X         |
|----------------|-------------------|--------------|--------------|--------------|
|                | 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)     | 0.129        | 22(0.30)     | 51(0.67)       | 0.255    | 8(0.15)           | 46(0.85)       | 0.104   |
| Control        | 3(0.04)           | 75(0.96)     | 0.074        | 17(0.32)     | 37(0.69)       | 0.801    | 8(0.14)           | 51(0.86)       | 0.084   |
| All            | 6(0.04)           | 137(0.96)    | 0.551        | 39(0.31)     | 88(0.69)       | 0.775    | 16(0.14)          | 97(0.86)       | 0.828   |
| Allele frequency (%) | (%)   | (%)          | (%)          | (%)          | (%)          | (%)      | (%)               | (%)            |
| Case           | 0.05              | 0.95         | 0.30         | 0.70         | 0.07         | 0.93     | 0.07              | 0.93          |
| Control        | 0.04              | 0.96         | 0.32         | 0.69         | 0.07         | 0.93     | 0.07              | 0.93          |
| All            | 0.04              | 0.96         | 0.31         | 0.69         | 0.07         | 0.93     | 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.As9Asn 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.As9Asn 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.

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References

[1] Katia AA, Strunz CMC, Maranhão RC, et al. The S447X polymorphism of lipoprotein lipase: effect on the incidence of premature coronary disease and on plasma lipids. Arq. Bras. Cardiol. 2007;88(3):267–73.
[2] Jelassi A, Jguirim I, Slimani A, et al. Association between variants of lipoprotein lipase and coronary heart disease in a Tunisian population. Pathol. Biol. (Paris) 2012;60(3):180–4.
[3] Wittrup HH, Tybjaerg-Hansen A, Nordestgaard BG. Lipoprotein lipase mutations, plasma lipids and lipoproteins, and risk of ischemic heart disease: a meta-analysis. Circulation 1999;99(22):2901–7.
[4] Musa HH, Tyrab EM, AbdelHamid MM, et al. Characterization of lipid profile in coronary heart disease patients in Sudan. Indian Heart J. 2013;65:232–3.
[5] Reymer PWA, Gagne SE, Groenemeyer BE, et al. A lipoprotein lipase mutation (Asn291Ser) is associated with reduced HDL levels in premature atherosclerosis. Nat. Genet. 1995;10:28–33.