e-ISSN: 0976-822X, p-ISSN:2961-6042

Available online on http://www.ijcpr.com/

International Journal of Current Pharmaceutical Review and Research 2026; 18(1); 1086-1091

Original Research Article

Study of Lipoprotein (A) in Type 2 Diabetes Mellitus as a Marker of Atherosclerotic Cardiovascular Disease: A Case-Control Analysis

Pradeep Kumar Sharma[1], Kumari Suruchi[2], Ravindra Kumar Das[3]

[1]Senior Resident, Department of Medicine, Darbhanga Medical College & Hospital, Laheriasarai, Bihar.
[2]Senior Resident, Department of Medicine, Darbhanga Medical College & Hospital, Laheriasarai, Bihar.
[3]Associate Professor, Department of Medicine, Darbhanga Medical College & Hospital, Laheriasarai, Bihar.

Received: 01-10-2025 / Revised: 15-11-2025 / Accepted: 21-12-2025
Corresponding author: Dr. Kumari Suruchi
Conflict of interest: Nil

Abstract

Background: Atherosclerotic cardiovascular diseases, or ASCVD, are a significant consequence of Type 2 diabetes mellitus (T2DM), affecting a significant portion of the population and posing a healthcare burden. The purpose of this study is to show that lipoprotein (a) [Lp (a)] is a marker of ASCVD and to establish a likely association between it and type 2 diabetes.

Methods: There were 200 participants in this case-control study: 50 healthy volunteers who were matched for age and gender and 150 diabetic patients. A comprehensive history of type 2 diabetes was taken, and vascular diabetic complications were evaluated. Every participant had their fasting plasma glucose, HbA1c, lipid profile, s. creatinine, and Lp (a) level measured.

Results: Lp (a) levels were significantly low in diabetic patients (19.8 ± 13.4 mg/dl) compared to control group (32.6 ± 20.8 mg/dl) (p <0.001). Lp (a) level was significantly higher in diabetics with macro-vascular complications (22.7 ± 14.4 mg/dl) than diabetics with micro-vascular complications (11.7 ± 6.5 mg/dl). Lp (a) level among diabetics with macro-vascular complications was insignificant higher than diabetics without vascular complications (p= 0.08).

Conclusion: Given the substantial correlation between Lp (a) and type 2 diabetes and accompanying vascular consequences, more research is necessary, particularly in the area of genetics.

Keywords: Lipoprotein (a), Diabetes Mellitus, HbA1c, ASCVD, case control study.

DOI: 10.25258/ijcpr.18.1.182

Introduction

According to estimates from the International Diabetes Federation (IDF), there will be 463 million people with diabetes in 2019. Over half of the world's population may be impacted by 2045, with 578 million cases expected by 2030.[1] Diabetics may have a twofold increased risk of cardiovascular disease (CVD). Although type 2 diabetes mellitus (T2DM) patients typically have dyslipidemia and hypertension, two established risk factors for the development of CVD, these conditions are not clearly linked to the population-specific increased risk of CVD.[2,3]

Numerous studies have been conducted to identify and associate nontraditional risk variables with macrovascular outcomes in people with diabetes. The lipoprotein (a) [Lp (a)] has been the subject of extensive research and is regarded as one of the several possible risk factors. Because apoB-100 and apolipoprotein (a) are covalently bound by a disulfide link, Lp (a) resembles low-density lipoprotein (LDL). Plasma Lp (a) concentration is mostly determined by the LPA gene, which is located on chromosome 6q26-27. Because the Lp (a) gene has an unpredictable number of kringle IV repeats, apo (a) proteins vary in size. There are many forms of the protein apo(a), which are known as isoform (a).[4,5]

Because the endoplasmic reticulum of hepatocytes sequesters larger molecules, lowering plasma levels, Lp (a) levels are inversely proportional to the size of the apo(a)isoform.[6]

The much slower rate of generation of the larger isoforms caps the amount of Lp (a) in the plasma because the precursor protein for Lp (a) does not exit the cell until the last steps of protein synthesis are finished.[7]

The purpose of our study was to show that Lp (a) is a marker of ASCVD and to establish a likely association between it and T2DM.

Material and Methods

This cross-sectional case control analysis with 50 healthy controls who were matched in age and gender and 150 individuals who had type 2 diabetes. Between September 2020 and February 2021, participants were chosen from the Department of Medicine, Darbhanga Medical College and Hospital, Laheriasarai, Bihar. The patients in the study had normal lipid profiles and ranged in age from 40 to 70.

Patients with one or more of the following characteristics were excluded; Patients with Type 1 DM, gestational diabetes, patients receiving estrogens or progesterone, patients receiving drugs affecting metabolism of Lp (a) like long time using of steroids as well as niacin and other diseases that might affects Lp (a) level as liver cirrhosis, heart failure, thyroid disease, acute illness, severe infection, or malignancy.

Subjects were grouped as; the control group (group A), constituting 50 apparently healthy volunteers, and the case group (group B) with 150 T2DM patients. The diabetic group were further subdivided into the following 3 subgroups; 1) Subgroup B: it involved 50 patients of T2DM without complications, 2) Subgroup C: it involved 50 patients of T2DM who had macro-vascular complications (one or more of cerebrovascular disease, peripheral vascular disease as well as coronary artery disease), and 3) Subgroup D: it involved 50 patients of T2DM who had micro-vascular complications (one or more of retinopathy, neuropathy and nephropathy). Demographic and anthropometric data were collected, additionally, treatment and vascular consequences from diabetes.

At the Clinical Pathology Department, we tested fasting plasma glucose (FPG), lipid profile, serum creatinine, as well as glycated hemoglobin (HbA1c) at DMCH, Laheriasarai, Bihar, according to the laboratory's standard procedures. For Lp (a) measurement; the laboratory received whole blood collected in tubes containing separating gel for analysis then centrifuged, the serum was collected and kept at -80^{0}C. (ELISA) was used for the analysis. When Lp (a) was below 14 mg/dL, it was considered desirable; and when between 14 and 30 mg/dL, it was considered at risk; and when between 31 and 50 mg/dL, it was considered high risk; and beyond 50 mg/dL, it was considered extremely high risk [8].

Investigation report forms were used to document the collected information. The analysis was performed in SPSS (Statistical Package for the Social Sciences) version 26. Means and standard deviations (SD) were used for summarization of quantitative data, and frequencies and related percentages were used to present qualitative data. For the statistical significance of difference, means of two sets of numerical data had been compared by the Student's t-test. The Mann-Whitney U-test was employed for comparing the two groups, as it is appropriate for non-parametric continuous data. We used the chi-square test to compare means across groups in our categorical data analysis (X^2). When the p value of a statistical test was equals or less than 0.05, it was considered to be significant.

Results

Our study included 150 T2DM patients (group B), which was further divided into three subgroups: subgroup B (diabetic patients without vascular complications), subgroup C (diabetes with macro-vascular complications), and subgroup D (diabetic patients with micro-vascular complications). Group A consisted of 50 healthy control individuals (28 males and 22 females).

Table (1) demonstrated that systolic blood pressure (SBP), diastolic blood pressure (DBP), HbA1c, FPG, S.creatinine, triglyceride (TG) level, Low-density lipoprotein cholesterol (LDL-C) and total cholesterol (TC) were significantly higher in all diabetic patients than the control group ($p<0.001$). However, body mass index (BMI) was insignificantly higher in diabetic patients (32.23 ± 5.52 kg\m2) than control group (30.60 ± 5.51kg\m2) ($p= 0.07$). The mean duration of DM was 10.25 years (SD 2.12).

Table 1: Subjects' sociodemographic and laboratory data

Variable (Quantitative Data)	Cases (n=150)	Control (n=50)	p-value
	Mean±SD	Mean±SD	
Age (years)	54.88±8.50	53.1±8.8	0.2
BMI(Kg\m^2)	32.23±5.52	30.60±5.51	0.07
SBP(mmHg)	139.92±15.72	121.24±10.53	<0.001
DBP (mmHg)	84.23±9.08	76.94±5.68	<0.001
HbA1c%	8.38±1.83	5.20±0.24	<0.001
FPG(mg\dl)	130.26±4.76	82.84±6.77	<0.001
S. Creatinine(mg \dl)	1.38±0.14	0.83±0.14	<0.001
TG level(mg\dl)	137.17±15.56	118.66±18.27	<0.001

LDL-C(mg\dl)		89.67±9.36		85.38±7.11		0.003
HDL-C(mg \dl)		42.31±7.46		44.16±5.18		0.1
Total-Cholesterol(mg\dl)		178.15±17.41		158.06±17.79		<0.001
Duration of DM(years)		10.25±2.12		------		---
Variable(Qualitative Data)		**Number**	**%**	**Number**	**%**	
Sex	Female	86	57.3%	28	56.0%	0.9
	Male	64	42.7%	22	44.0%	
Smoking	No	98	65.3%	32	64.0%	0.9
	Yes	52	34.7%	18	36.0%	
DM Treatment	Insulin	48	32%	---	---	---
	OAD	102	68%	---	---	---

SD= standard deviation, BMI= Body mass index, SBP=systolic blood pressure DBP=diastolic blood pressure, HbA1c= hemoglobin A1C, FPG=fasting plasma glucose, TG =triglycerides, LDL, C=low density lipoprotein cholesterol, HDL,C=high density lipoprotein cholesterol ,DM =diabetes mellitus ,OAD=oral anti diabetic drugs.

Table (2) shows that SBP, DBP, HbA1c, FPG, TG level, HDL–C and TC were significantly higher in the diabetic patients without complications compared to the control group. Lp (a) level was significantly higher in the control than the diabetic groups without complications (p <0.001).

Table 2: Clinical and laboratory data of group (A) and subgroup (B)

Variable	**Group (A) (n=50)**	**Subgroup (B) (n=50)**	**T-test**	**p-value**
	Mean±SD	**Mean±SD**		
SBP(mmHg)	121.24±10.53	133.36±14.42	4.8	<0.001*
DBP (mmHg)	76.94±5.68	80.96±8.14	2.8	0.003*
HbA1C%	5.20±0.24	7.70±0.79	21.4	<0.001*
FPG(mg/dl)	82.84±6.77	125.20±31.70	9.2	<0.001*
S. Creatinine(mg/dl)	0.83±0.14	0.86±0.15	0.9	0.2
TG level (mg/dl)	118.66±18.27	128.64±20.46	2.6	0.02*
LDL-C(mg/dl)	85.38±7.11	85.52±8.65	0.1	0.5
HDL-C(mg/dl)	44.16±5.18	40.68±8.16	2.5	0.006*
T-Cholesterol	158.06±17.79	168.28±17.10	2.9	0.002*
Lp(a) (mg/dl)	32.6±7.38	19.8±13.4	3.31	<0.001*

Table (3) demonstrated that SBP, DBP, HbA1c, TG, TC were significantly higher in macro-vascular diabetic complications in comparison to subgroup (B). Mean Lp (a) levels were insignificant higher in macrovascular diabetic complications (22.7 ± 4.64 mg/dl) than subgroup (B) (19.8 ± 3.84 mg/dl) (p value 0.08).

Table 3: Clinical and laboratory data of subgroups (B) and (C)

	Subgroup B (n=50)	**Subgroup C (n=50)**		
Variable	**Mean±SD**	**Mean±SD**	**T-test**	**p-value**
SBP (mmHg)	133.36±14.42	145.4±15.3	4.03	<0.001*
DBP (mmHg)	80.96±8.14	85.5±8.9	2.6	0.005*
HbA1c%	7.70±0.79	9.3±2.5	4.3	<0.001*
FPG (mg/dl)	125.20±31.70	134.3±30.4	1.5	0.07
S. Creatinine (mg\dl)	0.86±0.15	0.8±0.16	1.8	0.04*
TG level (mg\dl)	128.64±20.46	139.1±10.3	3.2	<0.001*
LDL-C (mg\dl)	85.52±8.65	87.2±8.4	1.01	0.2
HDL-C (mg\dl)	40.68±8.16	40.6±5.8	0.1	0.5
T-Cholesterol (mg\dl)	168.28±17.10	176.1±14.9	2.4	0.008*
Lp(a) (mg/dl)	19.8±3.84	22.7±4.64	1.7	0.08

Table (4), lipid profile was significantly higher in subgroup (D) than subgroup (C). The levels of Lp (a) were significantly higher in subgroup (C) than subgroup (D).

Table 4: Clinical and laboratory data of subgroups (C) and (D)

Variable	Subgroup (C) (n=50)	Subgroup (D) (n=50)	T-test	p-value
	Mean±SD	Mean±SD		
SBP (mmHg)	145.4±15.3	141.1±15.3	1.4	0.1
DBP (mmHg)	85.5±8.9	86.2±9.4	0.4	0.3
HbA1C%	9.3±2.5	8.1±1.3	3.1	0.003*
FPG (mg/dl)	134.3±30.4	131.3±4.2	0.4	0.3
S. Creatinine	0.8±0.16	2.5±0.2	9.9	<0.001*
TG level (mg\dl)	139.1±10.3	143.8±9.5	2.3	0.01*
LDL-C (mg\dl)	87.2±8.4	96.2±7.4	5.7	<0.001*
HDL-C (mg\dl)	40.6±5.8	45.7±7.2	3.9	<0.001*
T-Cholesterol (mg\dl)	176.1±14.9	190.1±12.6	5.04	<0.001*
Lp(a) (mg/dl)	22.7±4.84	11.7±1.75	4.9	<0.001*

Discussion

More knowledge of the underlying causes of CVD and T2DM is necessary for improved clinical diagnosis and treatment of high-risk individuals. Our results demonstrated that diabetes individuals had significantly lower Lp (a) than the control group. These results supported research of a European population that discovered a negative correlation between Lp (a) levels and the onset of type 2 diabetes. In [9]

Our results are supported by the results of prospective research that looked at the connection between plasma Lp (a) levels and the onset of T2DM in 26,746 women from the Women's Health Services (WHS) and the Copenhagen City Heart Study (CCHS). Results from the WHS and CCHS studies showed that patients with diabetes had lower Lp (a) levels than those without diabetes.[9] The San Antonio Heart Study found no significant difference in Lp (a) levels between the diabetes and non-diabetic populations, while we found the opposite to be true.[10] Some reasons for decreased Lp (a) in diabetic people could include; The LPA gene encodes Lp (a), and studies have demonstrated that the kringle IV type 2 (KIV-2) variant is critical to the size of the Lp (a) isoform. Low Lp (a) levels are associated with an increased risk of developing T2DM, which is in turn linked to large isoform size (indicated by a high number of KIV-2 repetitions in the gene). Hence, it is not just Lp (a) concentrations that mediate the link between Lp (a) and T2DM; rather, the high isoform size of Lp (a) molecules plays a role as well. Genetic study revealed a correlation between increasing isoform size and an increased chance of acquiring T2DM.[11]

Low Lp (a) concentrations may be a sign of insulin resistance, as indicated in one study, which found an inverse relationship between Lp (a) and insulin and 2-hour postprandial glucose levels.[12] Another study showed that Insulin blocks the function of apolipoprotein(a) in hepatocytes, which may explain why Lp (a) concentrations are lower in type 2 DM and higher in type 1 DM.[13]

Our findings suggest a weak association between Lp (a) and the onset of ASCVD in diabetics, with Lp (a) levels being insignificantly higher in the group of patients with macrovascular problems compared to those without such complications. However, Lp (a) levels were considerably higher in the group of diabetics who experienced macro-vascular difficulties as opposed to those who experienced micro-vascular complications. Increased risk of CVD was found to correspond with greater plasma levels of Lp (a) in the Copenhagen Cardiovascular Health Study (CCHS), the Copenhagen Ischemic Heart Disease Study (CIHDS), and the Copenhagen General Population Study (CGPS).[14] In addition, Clarke et al. found a significant association between high Lp (a) levels and CHD risk in their case control genetic study.[15] Two Mendelian randomization studies have found an association between Lp (a) and the risk of atherosclerosis and CVD.[16]

Patients having coronary angiography (including those with diabetes) were studied, and researchers found that whereas Lp (a) was a robust and independent predictor of CVD events among those without diabetes, it was not among those with diabetes.[17]. According to a study conducted by the European Society of Cardiology, there was a linear correlation between Lp (a) and CVD occurrences. With the following clinical scenarios, it is recommended that Lp (a) be measured: (i) premature CVD, (ii) Familial hypercholesterolemia (FH), (iii) increased Lp (a) levels or a family history of CVD, and (iv) CVD recurrence despite excellent statin therapy; 10-year mortality risk of less than 5%.[18] However, one study revealed no association between plasma Lp (a) levels and CVD risk in patients with T2DM.[19] After 13 years of follow-up, patients with T2DM who had high or low Lp (a) levels did not differ significantly in their risk of CHD or stroke, according to a study conducted by Abu-Lebdeh et al.[20] Another study identified a modest connection between plasma Lp (a) levels and the incidence of CVD and death in age-adjusted diabetics, using data from the NHS

and Health Professional Follow up Study (HPFS) trials.[21]

A general population study with a 10-year follow-up found no significant difference in the association between Lp (a) and CHD between those with and without diabetes.[22] The association of Lp (a) with ASCVD could be explained as follow; Firstly, Cholesterol is deposited in the expanding atherosclerotic lesions because Lp (a) is more tightly confined than LDL by binding to the extracellular matrix via apo lipoprotein(a) and apo lipoprotein B component.[23] Wound healing is aided by Lp (a) because the apo(a) molecule has an affinity for fibrin, stimulates cell proliferation, and transports cholesterol. Lipid buildup in the arterial wall is caused by Lp (a), binding to fibrinogen, proteoglycans, and fibronectin among other components of the subendothelial matrix.[24] Secondly, Oxidative modification of Lp (a) creates a substrate for macrophage absorption and promotes foam cell formation, just as it does for low-density lipoprotein (LDL).[25]

As oxidized Lp (a) was discovered to have an inflammatory impact by increasing monocyte chemotaxis and inducing the formation of vascular adhesion molecules.[26] Thirdly, because of its similarities to plasminogen, Apo(a), which is present in Lp (a), may reduce the activity of plasminogen and raise the risk of thrombosis. To rephrase, Plasminogen activation may be inhibited by Lp (a) because of competition for fibrin binding.[27]

Evidence suggests that apo(a) interacts with tissue plasminogen activator (tPA) and plasminogen to alter the kinetics of plasmin generation within the fibrinolytic complex.[28] As Lp (a) promotes synthesis of plasminogen activator inhibitor-1, less tPA is available for plasminogen activation (PAI-1).[29]

Additional thrombogenic properties of Lp (a). Include the ability to enhance platelet aggregation and the inactivation of tissue factor pathway inhibitor.[30] Lastly, Lp (a) is a member of the same family as apoE and has been demonstrated to stimulate the formation of endothelium and smooth muscle cells in laboratory experiments (a).

Ichikawa et al. [31] supported proliferation of smooth muscle cells in regions of Lp (a) deposition in transgenic rabbits. Smooth muscle cell proliferation, regulated negatively by transforming growth factor-, was shown to be reduced by Lp (a).[32] The actin cytoskeleton of cultured endothelial cells has been demonstrated to rearrange itself in response to the apo(a) component of Lp (a). The development of atherosclerosis is preceded by the endothelium being damaged and more permeable due to a lack of cell-to-cell interaction.[33]

Conclusion

Since atherosclerosis is the most prevalent cause of morbidity and mortality among diabetics and causes a significant strain on Egypt's healthcare resources, Lp (a) screening may be advised for atherosclerotic patients with DM as a secondary preventive measure. For a more accurate evaluation of Lp (a) as a CVD indicator, more prospective research involving a large number of populations is required.

References

1. Saeedi P, Petersohn I, Salpea P et al. (2019): Global and regional diabetes prevalence estimates for 2019 and projections for 2030 and 2045: Results from the International Diabetes Federation Diabetes Atlas, 9th edition. Diabetes Res Clin Pract., 157:107843.
2. Pyorola K (1990): Diabetes and coronary artery disease: what a coincidence? J Cardiovasc Pharmacol., 16: 8-14.
3. Gries F, Koschinsky T (1991): Diabetes and arterial disease. Diabetic Med., 8: 82–87.
4. McLean J, Tomlinson J, Kuang W et al. (1987): cDNA sequence of human apolipoprotein (a) is homologous to plasminogen. Nature, 330(6144):132-7.
5. Utermann G, Menzel H, Kraft H et al. (1987): Lp(a) glycoprotein phenotypes. Inheritance and relation to Lp(a)-lipoprotein concentrations in plasma. J Clin Invest., 80(2):458-65.
6. Sandholzer C, Hallman D, Saha N et al. (1991): Effects of the apolipoprotein (a) size polymorphism on the lipoprotein(a) concentration in 7 ethnic groups. Hum Genet., 86(6):607-14.
7. Lobentanz E, Krasznai K, Gruber A et al. (1998): Intracellular metabolism of human apolipoprotein(a) in stably transfected Hep G2 cells. Biochemistry, 37(16):5417-25.
8. Farzam K, Senthilkumaran S (2021): Lipoprotein A. StatPearls.Treasure Island (FL): StatPearls Publishing. https://www.ncbi.nlm.nih.gov/books/NBK570621/
9. Mora S, Kamstrup P, Rifai N et al. (2010): Lipoprotein(a) and risk of type 2 diabetes. Clin Chem., 56(8):1252-60.
10. Mack S, Coassin S, Rueedi R et al. (2017): A genomewide association meta-analysis on lipoprotein(a) concentrations adjusted for apolipoprotein(a) isoforms. J Lipid Res., 58:1834-44.
11. Tolbus A, Mortensen M, Nielsen S et al. (2017): Kringle IV type 2, not low lipoprotein(a): as a cause of diabetes: a novel genetic approach using SNPs associated

selectively with lipoprotein(a) concentrations or with kringle iv type 2 repeats. Clin Chem., 63:1866-76.
12. Rainwater D, Haffner S (1998): Insulin and 2-hour glucose levels are inversely related to Lp(a) concentrations controlled for LPA genotype. Arterioscler Thromb Vasc Biol., 18(8):1335-1341.
13. Neele D, De Wit E, Princen H (1999): Insulin suppresses apolipoprotein(a) synthesis by primary cultures of cynomolgus monkey hepatocytes. Diabetologia, 42(1):41-44.
14. Kamstrup P, Tybjaerg-Hansen A, Steffensen R et al. (2009): Genetically elevated lipoprotein (a) and increased risk of myocardial infarction. JAMA., 301: 2331-2339.
15. Clarke R, Peden J, Hopewell J et al. (2009): Genetic variants associated with Lp(a) lipoprotein level and coronary disease. N Engl J Med., 361:2518-2528.
16. Erqou S, Kaptoge S, Perry P et al. (2009): Lipoprotein(a) concentration and the risk of coronary heart disease, stroke, and nonvascular mortality. JAMA., 302: 412-423.
17. Saely C, Koch L, Schmid F et al. (2006): Lipoprotein(a): Type 2 diabetes and vascular risk in coronary patients. Eur J Clin Invest., 36(2): 91-97.
18. Nordestgaard B, Chapman M, Ray K et al. (2010): Lipoprotein(a) as a cardiovascular risk factor: Current status. Eur Heart J., 31:2844-2853.
19. Haffner S, Moss S, Klein B et al. (1992): Lack of association between lipoprotein (a) concentrations and coronary heart disease mortality in diabetes: the Wisconsin epidemiologic study of diabetic retinopathy. Metabolism, 41(2):194-197.
20. Abu-Lebdeh H, Hodge D, Nguyen T (2002): Predictors of macrovascular disease in patients with Type 2 diabetes mellitus. Mayo Clin Proc., 76(7):707-712.
21. Qi Q, Workalemahu T, Zhang C et al. (2012): Genetic variants, plasma lipoprotein(a) levels, and risk of cardiovascular morbidity and mortality among two prospective cohorts of type 2 diabetes. Eur Heart J., 33: 325-334.
22. Kamstrup P, Benn M, Tybjaerg-Hansen A et al. (2008): Extreme lipoprotein(a) levels and risk of myocardial infarction in the general population. Circulation, 117(2):176-184.
23. Nielsen L (1999): Atherogenecity of lipoprotein(a) and oxidized low density lipoprotein: insight from in vivo studies of arterial wall influx, degradation and efflux. Atherosclerosis, 143:229-243.
24. Lundstam U, Hurt-Camejo E, Olsson G et al. (1999): Proteoglycans contribution to association of Lp(a) and LDL with smooth muscle cell extracellular matrix. Arterioscler Thromb Vasc Biol., 19: 1162-7.
25. Haberland M, Fless G, Scanu A et al. (1992): Malondialdehyde modification of lipoprotein(a) produces avid uptake by human monocyte-macrophages. J Biol Chem., 267:4143-51.
26. Allen S, Khan S, Tam S et al. (1998): Expression of adhesion molecules by lp(a): a potential novel mechanism for its atherogenicity. FASEB J., 12:1765-76.
27. Rouy D, Grailhe P, Nigon F et al. (1991): Lipoprotein(a) impairs generation of plasmin by fibrin-bound tissue-type plasminogen activator. In vitro studies in a plasma milieu. Arterioscler Thromb., 11:629-38.
28. Hancock M, Boffa M, Marcovina S et al. (2003): Inhibition of plasminogen activation by lipoprotein(a): critical domains in apolipoprotein(a) and mechanism of inhibition on fibrin and degraded fibrin surfaces. J Biol Chem., 278:23260-9.
29. Levin E, Miles L, Fless G et al. (1994): Lipoproteins inhibit the secretion of tissue plasminogen activator from human endothelial cells. Arterioscler Thromb., 14:438-42.
30. Caplice N, Panetta C, Peterson T et al. (2001): Lipoprotein (a) binds and inactivates tissue factor pathway inhibitor: a novel link between lipoproteins and thrombosis. Blood, 98:2980-7.
31. Ichikawa T, Unoki H, Sun H et al. (2002): Lipoprotein(a) promotes smooth muscle cell proliferation and dedifferentiation in atherosclerotic lesions of human apo(a) transgenic rabbits. Am J Pathol., 160:227-36.
32. Grainger D, Kirschenlohr H, Metcalfe J et al. (1993): Proliferation of human smooth muscle cells promoted by lipoprotein(a). Science, 260:1655-8.
33. Pellegrino M, Furmaniak-Kazmierczak E, LeBlanc J et al. (2004): The apolipoprotein(a) component of lipoprotein(a) stimulates actin stress fiber formation and loss of cell-cell contact in cultured endothelial cells. J Biol Chem., 279(8):6526-33.