APOLIPOPROTEIN A & B, (Apo A-I, Apo B, Apo A-I/ Apo B ratio), LIPOPROTEIN (a) [LP(a)], APOLIPOPROTEIN E (APO E)

NORMAL VALUES

Apo A-I
Males: 75-160 mg/dL; 0.75-1.6 g/L (SI)
Females: 80-175 mg/dL; 0.80-1.75 g/L (SI)

Apo B
Males: 50-125 mg/dL; 0.50-1.25 g/L (SI)
Females: 45-120 mg/dL; 0.45-1.20 g/L (SI)

Apo A-I/ Apo B ratio
Males: 0.8-2.24
Females: 0.76-3.23

Lipoprotein (a)
Caucasian (5^{th} to 95^{th} percentile)
Males: 2.2- 49.4 mg/dL
Females: 2.1- 57.3 mg/dL

African-American (5^{th} to 95^{th} percentile)
Males: 4.6-71.8 mg/dL
Females: 4.4-75 mg/dL

NUTRITIONAL SIGNIFICANCE

Apolipoproteins are surface proteins of lipoprotein particles. Apolipoprotein A (apo A) is the major polypeptide component of HDL. Apo A has two main forms, apo A-I and apo-A-II. Apo A-I constitutes about 75% of apo A in HDL. Apo A-II constitutes about 20% of the total HDL protein. As a general rule, as HDL levels increases, so do apo A levels. Physical exercise may increase levels. Eating a diet high in carbohydrates and polyunsaturated fats may decrease apo A-I levels. Also, smoking may decrease levels. Some experts have proposed that apo A is a better index of risk for atherosclerosis than HDL assay.

Apolipoprotein B is the major polypeptide component of LDL and VLDL. Approximately 80% of the protein in LDL is apo B and about 40% of the protein in VLDL is apo B. Apo B has two main forms apo B-48 and apo B-100.
Apo B-100 is synthesized in the liver and found in lipoproteins of endogenous origin. It is the principal transport mechanism for endogenous cholesterol. Apo B-100 has an affinity for the LDL receptor sites located on the cell surfaces of peripheral tissues. It is involved

in deposition of cholesterol in tissues. Eating a diet that is high in saturated fats and cholesterol may increase apo-B levels. Some experts propose that apo B-100 may be a better index for risk of atherosclerosis than LDL assay.

Apo B-48 is synthesized in the intestines and is mainly found in chylomicrons. It serves as a carrier for ingested lipids through the intestines into the blood stream. Some experts have proposed that apo B is a better indicator of risk for atherosclerosis than LDL.

Decreased levels of apo A and increased levels of apo B-100 are associated with increased risk of coronary heart disease. A low ratio of apo A to apo B may also be a risk factor.

Lp(a) or 'lipoprotein little a' is another lipoprotein particle found in LDL. It is similar in chemical structure to plasminogen. Some experts believe that Lp(a) is a mutation of plasminogen. Plasminogen is the precursor of the proteolytic enzyme plasmin. This enzyme is responsible for dissolving fibrin clots. It has been proposed that Lp(a) is an independent risk factor for atherosclerosis because of its relationship to plasminogen. Microthrombi containing fibrin on the vessel walls become incorporated into the arthrosclerotic plaque. Some researchers propose that following endothelial damage, Lp(a) may become incorporated into the arterial wall, inhibiting the cleavage of fibrin in microthrombi by competing with plasminogen for access to fibrin. Atherosclerotic damage of the arterial wall occurs and results in occlusive disease or an aneurysm. Familial hypercholesterolemia, some forms of renal failure, nephrotic syndrome and estrogen depletion in women over the age of 50 have been associated with increased levels of Lp(a). Individuals with Lp(a) appear to have a much higher risk for coronary heart disease.

Apolipoprotein E is involved in the transport of cholesterol. It has three alleles: E-2, E-3 and E-4. The apo E-4 gene has been proposed as a risk factor for Alzheimer's disease. While apo E-4 allele has a strong association with Alzheimer's disease, it is unclear how the apo E functions as a risk factor for modifying the age of onset of Alzheimer's disease. Apo E is present in neuritic amyloid plaques and may be involved in neurotic tangle formations since it binds with tau proteins.

RELATED TESTS: LDL-C, HDL-C

Apo A-I Increased with:	Apo A-I Decreased with:
■ Familial hyperalphalipoproteinemia	■ Familial hypoalphalipoproteinemia
■ Pregnancy	■ Ischemic coronary disease
■ Weight reduction	■ Myocardial infarction
	■ Coronary heart disease
	■ Uncontrolled diabetes mellitus
	■ Tangier's disease
	■ Nephrotic syndrome
	■ Chronic renal failure
	■ Cholestasis
	■ Hemodialysis
	■ Fish eye disease
	■ Hepatocellular disease
	■ Familial hypertriglyceridemia

Medications that may increase Apo-A-I:

Carbamazepine	Estrogens	Ethanol
Lovastatin	Niacin	Oral contraceptives
Phenobarbital	Pravastatin	Simvastatin

Apo B Increased with:	Apo B Decreased with:
■ Hyperlipoproteinemia (types IIa, IIb, IV, V) ■ Nephrotic syndrome ■ Pregnancy ■ Hemodialysis ■ Biliary obstruction ■ Coronary artery disease ■ Diabetes mellitus ■ Hypothyroidism ■ Anorexia nervosa ■ Hepatic disease and obstruction ■ Porphyria ■ Cushing's syndrome ■ Werner's syndrome	■ Hyperlipoproteinemia (type I) ■ Hyperthyroidism ■ Malnutrition ■ Inflammatory joint disease ■ Chronic pulmonary disease ■ Weight reduction ■ Chronic anemia ■ Reye's syndrome ■ Tangier's disease ■ Apo C-II deficiency

Medications that may increase Apo B:

Androgens	Beta-blockers	Diuretics
Ethanol abuse	Progestins	

Medications that may decrease Apo B:

Cholestyramine	Estrogen	Lovastatin
Simvastatin	Neomycin	Niacin
Thyroxine		

Lp(a) Increased with:	Lp(a) Decreased with:
■ Premature coronary artery disease	■ Alcoholism
■ Stenosis of cerebral arteries	■ Malnutrition

Lp(a) Increased with:	Lp(a) Decreased with:
■ Uncontrolled diabetes mellitus	■ Chronic liver disease
■ Severe hypothyroidism	
■ Familial hypercholesterolemia	
■ Chronic renal failure	
■ Estrogen depletion	

Medications that may decrease Lp(a):

Estrogens	Niacin	Neomycin
Stanozolol		