

Apolipoprotein A-I, Urine

Analyte: Apolipoprotein AI

Specimen Type: Urine

Optimum Volume: 0.5 mL

Stability:

2-8 Degrees C	-20 Degrees C	-70 Degrees C
6 days	1 month	2.8 years

Reporting Units: ng/mL or ng/mg Creatinine

Method: ELISA

Biological or Clinical Significance:

Human Apolipoprotein A-I (apo A-I) comprises about 70% of the high-density lipoproteins (HDL) protein mass, while apo A-II makes up another 15 – 20%. Apo A-I, a 243-amino acid molecule that contains a series of highly homologous amphipathic (polar and non-polar properties) alpha-helices, is a 28-kDa single polypeptide that lacks glycosylation or disulfide linkages. About 5 – 10% of human plasma apo A-I exists in a lipoprotein-unassociated state. Apo A-I appears to play a major role in the inhibition of atherosclerosis via its role in reverse cholesterol transport, and perhaps, because of its inherent antioxidant and anti-inflammatory properties or its central role in organizing HDL, which may possess additional antioxidant and anti-inflammatory properties. Oxidation of specific amino acid residues in apo A-I may contribute to atherogenesis by impairing cholesterol efflux from macrophages or by allowing the prolongation of free-radical oxidation, which leads to the spread of low-density lipoprotein (LDL) oxidation.

A majority of HDL functionality is derived from the capacity of apo A-I to sequester phospholipid and cholesterol and interact with plasma enzymes and cellular receptors. During reverse cholesterol transport, HDL interacts with lecithin:cholesteryl acyltransferase (LCAT) and cellular receptor class B type I in an ordered fashion that is reflected by HDL particle lipid composition. A high-affinity HDL receptor for apo A-I is in the beta-chain of ATP synthase on the surface of hepatocytes.

The plasma concentration of apo A-I is an excellent biomarkers of susceptibility to atherosclerosis and risk of cardiovascular disease.

In the kidney, apo A-I and some HDL particles may be filtered through the glomerulus and scavenged in the proximal tubule by binding to a receptor called cubulin. Once bound, the particles are internalized and catabolized. The HDL particle itself seems to have a higher affinity for cubulin than pure apo A-I, and HDL lipid composition influences HDL affinity. Some apo A-I and HDL must escape uptake in the tubule and can be measured in the urine (see reference 1).

Principle of Test Method:

The A-I assay is an Enzyme-Linked Immunosorbent Assay (ELISA) designed for detection of human apo A-I in urine and cell culture supernatant. This assay employs a quantitative sandwich enzyme immunoassay technique.



Biomarker Menu

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References:

1. A. P. Simonelli and D. S. Dresback. Principles of Formulation of Parenteral Dosage Forms (Stability Considerations), Kenneth A. Connors et al. Chemical stability of pharmaceuticals: a handbook for pharmacists, Canada. Wiley Interscience Publication, 1979, pg. 9-19.