

Apolipoprotein A-I/APOA1 Protein, Human, Recombinant

General Information

Synonyms:	Apolipoprotein A-I;APOA1;Apo-AI;ApoA-I
Protein Construction:	Arg19-Gln267
Species:	Human
Expression Host:	E. coli
Accession:	P02647
Molecular Weight:	25-31 KDa (reducing condition)
AA Sequence:	Arg19-Gln267

QC Testing

Biological Activity:	Activity has not been tested. It is theoretically active, but we cannot guarantee it. If you require protein activity, we recommend choosing the eukaryotic expression version first.
Purity:	Greater than 95% as determined by reducing SDS-PAGE. (QC verified)
Endotoxin:	< 0.1 ng/μg (1 EU/μg) as determined by LAL test.
Formulation:	Lyophilized from a solution filtered through a 0.22 μm filter, containing 20 mM PB, 150 mM NaCl, pH 7.2.

Preparation and Storage

Reconstitution:

Reconstitute the lyophilized protein in distilled water. The product concentration should not be less than 100 μg/ml. Before opening, centrifuge the tube to collect powder at the bottom. After adding the reconstitution buffer, avoid vortexing or pipetting for mixing.

Stability & Storage:

Lyophilized powders can be stably stored for over 12 months, while liquid products can be stored for 6-12 months at -80°C. For reconstituted protein solutions, the solution can be stored at -20°C to -80°C for at least 3 months.

Please avoid multiple freeze-thaw cycles and store products in aliquots.

Actual storage temperature shall be subject to the COA.

Shipping:

In general, lyophilized powders are shipped with blue ice, while solutions are shipped with dry ice.

Protein Background

Apolipoprotein A1 (APOA1) is a secreted protein which belongs to the Apolipoprotein A1/A4/E family. APOA1 is the major protein component of high density lipoprotein (HDL) in plasma. APOA1 plays a critical role in various biological processes, such as Cholesterol metabolism, Lipid metabolism and transport, Steroid metabolism. APOA1 promotes cholesterol efflux from tissues to the liver and thus helps to clear cholesterol from arteries. Defects in this gene resulted in HDL deficiencies, including Tangier disease (TGD), systemic non-neuropathic

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amyloidosis, premature coronary artery disease, hepatosplenomegaly and progressive muscle wasting and weakness. In addition, ApoA-I is implicated in the anti-endotoxin function of HDL via interaction with lipopolysaccharide or endotoxin.

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Tel:781-999-4286 E_mail:info@targetmol.com Address:34 Washington Street,Wellesley Hills,MA 02481