Recombinant Human ADA Protein (His Tag)

Catalog Number: PDEH100812

Note: *Centrifuge before opening to ensure complete recovery of vial contents.*

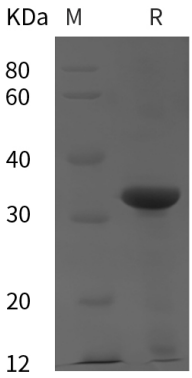
Description

Species	Human
Source	E.coli-derived Human ADA protein Lys11-Val280, with an N-terminal His
Calculated MW	29.6 kDa
Observed MW	32 kDa
Accession	P00813
Bio-activity	Not validated for activity

Properties

Purity	> 95% as determined by reducing SDS-PAGE.
Endotoxin	< 10 EU/mg of the protein as determined by the LAL method
Storage	Generally, lyophilized proteins are stable for up to 12 months when stored at -20 to -80°C. Reconstituted protein solution can be stored at 4-8°C for 2-7 days. Aliquots of reconstituted samples are stable at < -20°C for 3 months.
Shipping	This product is provided as lyophilized powder which is shipped with ice packs.
Formulation	Lyophilized from a 0.2 μm filtered solution in PBS with 5% Trehalose and 5% Mannitol.
Reconstitution	It is recommended that sterile water be added to the vial to prepare a stock solution of 0.5 mg/mL. Concentration is measured by UV-Vis.

Data



SDS-PAGE analysis of Human ADA proteins, 2 μg/lane of Recombinant Human ADA proteins was resolved with SDS-PAGE under reducing conditions, showing bands at 32 kDa.

Background

Adenosine Desaminase (ADA) deficiency, is a purine metabolic disorder that cause severe combined immunodeficiency (SCID) due to the accumulation of toxic metabolites that primarily affects development, differentiation and function of T and B lymphocytes. Adenosine deaminase is a polymorphic enzyme that has an important role in immune functions and in the regulation of intracellular and extracellular concentrations of adenosine and adenosine receptor activity. ADA activity might be considered as a useful diagnostic tool among the other markers in these diseases. Genetic variability of ADA activity may have, therefore, an important role in resistance to malaria. Adenosine Deaminase (ADA) deficiency is an autosomal recessive variant of severe combined immunodeficiency (SCID) caused by systemic accumulation of ADA substrates.

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