Recombinant Human ADSL/Adenylosuccinate Lyase Protein (His Tag)

Catalog Number: PKSH031168

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

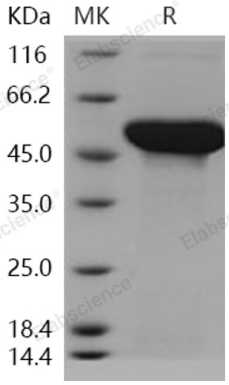
Description

Species	Human
Source	E.coli-derived Human ADSL/Adenylosuccinate Lyase protein Met 1-Leu 484, with an N-terminal His
Calculated MW	57.0 kDa
Observed MW	53 kDa
Accession	P30566-1
Bio-activity	Not validated for activity

Properties

Purity	> 95 % as determined by reducing SDS-PAGE.
Endotoxin	Please contact us for more information.
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 sterile PBS, 10% glycerol, pH 7.5 Normally 5% - 8% trehalose, mannitol and 0.01% Tween 80 are added as protectants before lyophilization. Please refer to the specific buffer information in the printed manual.
Reconstitution	Please refer to the printed manual for detailed information.

Data



> 95 % as determined by reducing SDS-PAGE.

Background

Adenylosuccinate lyase, also known as adenylosuccinase, ADSL or ASL, is an enzyme implicated in the reaction of adenylosuccinat converting to AMP and fumarate as part of the purine nucleotide cycle. The two substates of adenylosuccinate lyase (ADSL) are dephosphorylated derivatives of SAICA ribotide (SAICAR) and adenylosuccinate (S-AMP), which catalyzes an important reaction in the de novo pathway of purine biosynthesis. ADSL catalyzes two distinct reactions in the synthesis of purine nucleotides, both of which involve the _-elimination of fumarate to produce either aminoimidazole carboxamide ribotide from SAICAR or AMP from S-AMP. The Adenylosuccinate lyase deficiency is a rare autosomal recessive metabolic disorder characterized by the present of SAICA riboside and succinyladenosine (S-Ado). ADSL defect in different patients is often caused by different mutations to the enzyme.

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