

Recombinant Human Alkaline Phosphatase/ALPI Protein (Fc Tag)

Catalog Number: PKSH030691

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

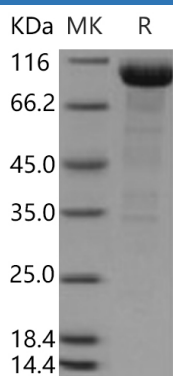
Description

Species	Human
Source	HEK293 Cells-derived Human Alkaline Phosphatase/ALPI protein Met 1-Asp 503, with an C-terminal hFc
Calculated MW	79.5 kDa
Observed MW	90-95 kDa
Accession	P09923
Bio-activity	Measured by its ability to cleave a fluorogenic substrate, 4-Methylumbelliferyl phosphate (4-MUP). The specific activity is > 10, 000 pmoles/min/μg.

Properties

Purity	> 83 % as determined by reducing SDS-PAGE.
Endotoxin	< 1.0 EU per μg 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 sterile PBS, pH 7.4 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



> 83 % as determined by reducing SDS-PAGE.

Background

For Research Use Only

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Rev. V3.5

Interferon-alpha/beta receptor alpha chain (IFNAR1) is a type I membrane protein that forms one of the two chains of a receptor for interferons alpha and beta. Binding and activation of the receptor stimulates Janus protein kinases; which in turn phosphorylate several proteins; including STAT1 and STAT2. The encoded protein also functions as an antiviral factor. Tyk2 slows down IFNAR1 degradation and that this is due; at least in part; to inhibition of IFNAR1 endocytosis. Mutant versions of IFNAR1; in which Tyr466 is changed to phenylalanine; can act in a dominant negative manner to inhibit phosphorylation of STAT2. These observations are consistent with a model in which IFNAR1 mediates the interaction between JAK kinases and the STAT transcription factors.