

Recombinant Human Alkaline Phosphatase/ALPI Protein (Fc Tag)

Catalog No. PKSH030691

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

Description

Synonyms IAP
Species Human

Expression Host HEK293 Cells **Sequence** Met 1-Asp 503

Accession P09923
Calculated Molecular Weight 79.5 kDa
Observed molecular weight 90-95 kDa
Tag C-hFc

Bioactivity 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 $< 1.0 \text{ EU per } \mu \text{g of the protein as determined by the LAL method.}$

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

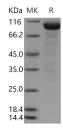
Normally 5 % - 8 % trehalose, mannitol and 0.01% Tween80 are added as

protectants before lyophilization.

Please refer to the specific buffer information in the printed manual.

Reconstitution Please refer to the printed manual for detailed information.

<u>Data</u>



> 83 % as determined by reducing SDS-PAGE.

Background

Interferon-alpha/beta receptor alpha chain (IFNAR1) is a type I membrane protein that forms one of the two chains of a

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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.

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