Human CD117 / c-kit Protein, Fc Tag (MALS verified)

Catalog # CD7-H525b

Synonym

CD117,SCFR,c-Kit,KIT

Source

Human CD117 Protein, Fc Tag(CD7-H525b) is expressed from human 293 cells (HEK293). It contains AA Val 308 - Thr 516 (Accession # P10721-2).

Molecular Characterization

CD117(Val 308 - Thr 516) P10721-2	Fc(Pro 100 - Lys 330) P01857

This protein carries a human IgG1 Fc tag at the C-terminus.

The protein has a calculated MW of 50.2 kDa. The protein migrates as 65-90 kDa when calibrated against Star Ribbon Pre-stained Protein Marker under reducing (R) condition (SDS-PAGE) due to glycosylation.

Endotoxin

Less than 1.0 EU per μg by the LAL method / rFC method.

Purity

>95% as determined by SDS-PAGE.

>90% as determined by SEC-MALS.

Formulation

Lyophilized from 0.22 μm filtered solution in PBS, pH7.4 with trehalose as protectant.

Contact us for customized product form or formulation.

Reconstitution

Please see Certificate of Analysis for specific instructions.

For best performance, we strongly recommend you to follow the reconstitution protocol provided in the CoA.

Storage

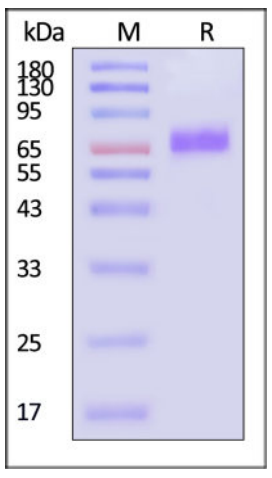
For long term storage, the product should be stored at lyophilized state at -20°C or lower.

Please avoid repeated freeze-thaw cycles.

This product is stable after storage at:

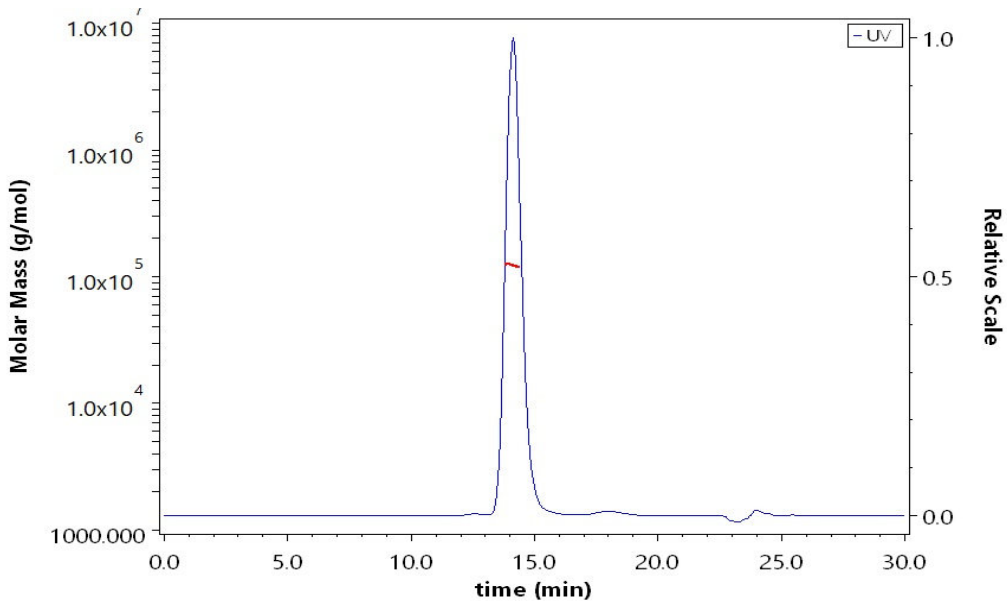
- -20°C to -70°C for 12 months in lyophilized state;
- -70°C for 3 months under sterile conditions after reconstitution.

SDS-PAGE



Human CD117 Protein, Fc Tag on SDS-PAGE under reducing (R) condition. The gel was stained with Coomassie Blue. The purity of the protein is greater than 95% (With Star Ribbon Pre-stained Protein Marker).

SEC-MALS



The purity of Human CD117 Protein, Fc Tag (Cat. No. CD7-H525b) is more than 90% and the molecular weight of this protein is around 115-140 kDa verified by SEC-MALS.
Report

Background

c-KIT activation has been shown to have oncogenic activity in gastrointestinal stromal tumors (GISTs), melanomas, lung cancer, and other tumor types. The targeted therapeutics nilotinib and sunitinib have shown efficacy in treating KIT overactive patients, and are in late-stage trials in melanoma and GIST. KIT overactivity can be the result of many genomic events from genomic amplification to overexpression to missense mutations. Missense mutations have been shown to be key players in mediating clinical response and acquired resistance in patients being treated with these targeted therapeutics.