

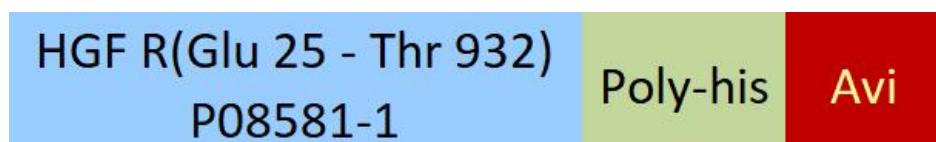
**Synonym**

MET, AUTS9, HGFR, RCCP2, c-Met

Source

Biotinylated Human HGF R, Avitag,His Tag (MET-H82E1) is expressed from human 293 cells (HEK293). It contains AA Glu 25 - Thr 932 (Accession # [P08581-1](#)).

Predicted N-terminus: Glu 25 (α chain) & Ser 308 (β chain)

Molecular Characterization[Other Tags and Version](#) [Biotin & Other Labeled Version](#)

This protein carries a polyhistidine tag at the C-terminus, followed by an Avi tag (Avitag™).

The protein has a calculated MW of 32.5 kDa (α chain) & 72.7 kDa (β chain). The protein migrates as 40-50 kDa (α subunit) and 90-100 kDa (β subunit) when calibrated against [Star Ribbon Pre-stained Protein Marker](#) under reducing (R) condition (SDS-PAGE) due to glycosylation.

Labeling

Biotinylation of this product is performed using Avitag™ technology. Briefly, the single lysine residue in the Avitag is enzymatically labeled with biotin.

Protein Ratio

Passed as determined by the HABA assay / binding ELISA.

Endotoxin

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

Purity

>90% as determined by SDS-PAGE.

Formulation

Lyophilized from 0.22 μ m 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.

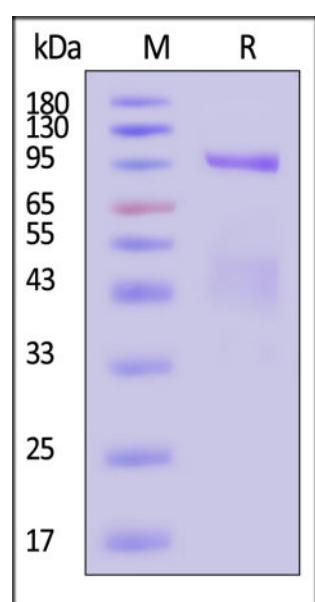
This product is stable after storage at:

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

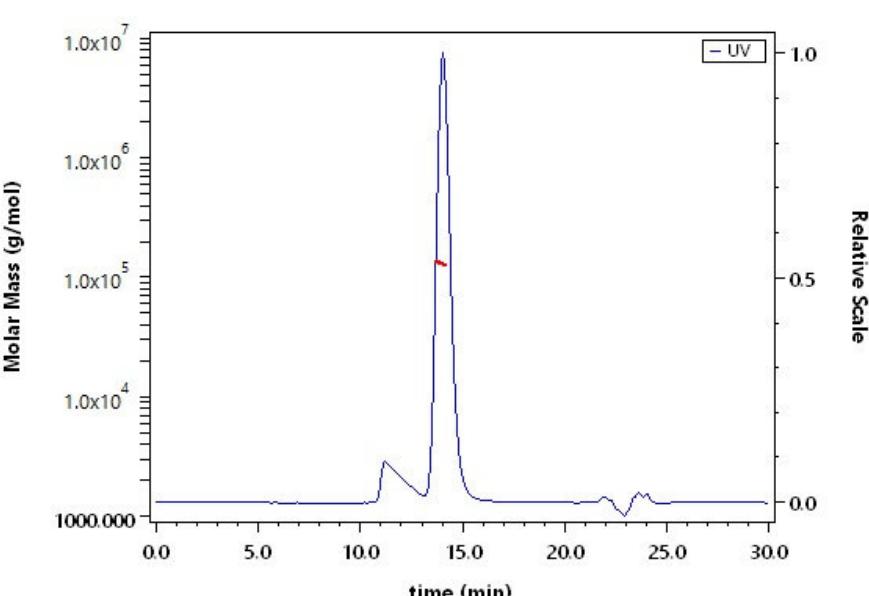
ACRO Quality Management System

- [QMS\(ISO, GMP\)](#)
- [Quality Advantages](#)
- [Quality Control Process](#)

SDS-PAGE**SEC-MALS**

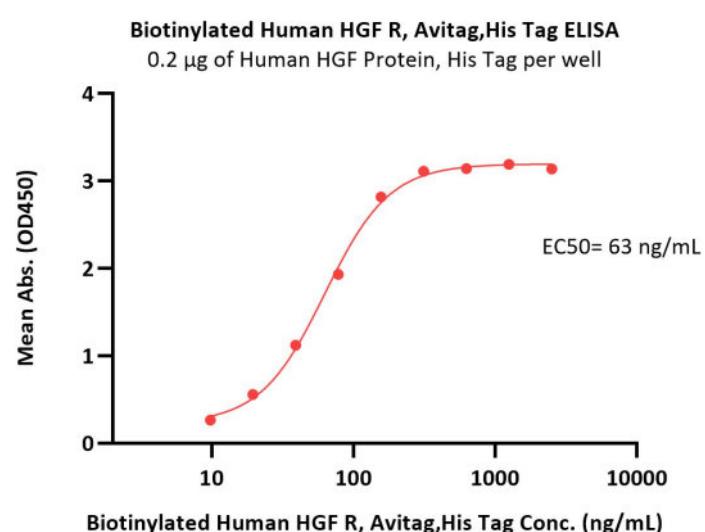


Biotinylated Human HGF R, Avitag, His Tag on SDS-PAGE under reducing (R) condition. The gel was stained with Coomassie Blue. The purity of the protein is greater than 90% (With [Star Ribbon Pre-stained Protein Marker](#)).



The purity of Biotinylated Human HGF R, Avitag, His Tag (Cat. No. MET-H82E1) is more than 85% and the molecular weight of this protein is around 110-140 kDa verified by SEC-MALS.

Bioactivity-ELISA



Immobilized Human HGF Protein, His Tag (Cat. No. HGF-H52H3) at 2 µg/mL (100 µL/well) can bind Biotinylated Human HGF R, Avitag, His Tag (Cat. No. MET-H82E1) with a linear range of 10-156 ng/mL (QC tested).

Background

Hepatocyte growth factor receptor (HGFR) is also known as mesenchymal-epithelial transition factor (MET), c-Met, and is a glycosylated receptor tyrosine kinase that plays a central role in epithelial morphogenesis and cancer development. HGFR protein possesses tyrosine-kinase activity. The primary single chain precursor protein is post-translationally cleaved to produce the alpha and beta subunits, which are disulfide linked to form the mature receptor. HGFR is normally expressed by cells of epithelial origin, while expression of HGF is restricted to cells of mesenchymal origin. Upon HGF stimulation, HGFR induces several biological responses that collectively give rise to a program known as invasive growth. Abnormal HGFR activation in cancer correlates with poor prognosis, where aberrantly active HGFR triggers tumor growth, formation of new blood vessels (angiogenesis) that supply the tumor with nutrients, and cancer spread to other organs (metastasis). HGFR is deregulated in many types of human malignancies, including cancers of kidney, liver, stomach, breast, and brain. Normally, only stem cells and progenitor cells express HGFR. However, cancer stem cells are thought to hijack the ability of normal stem cells to express HGFR, and thus become the cause of cancer persistence and spread to other sites in the body. Various mutations in the HGFR gene are associated with papillary renal carcinoma. HGFR mediates a complex program known as invasive growth. Activation of HGFR triggers mitogenesis, and morphogenesis.

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