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BioActive Human ALK (L1198F) Recombinant Protein,Fc Tag

Catalog Number:SGRP00646

DESCRIPTION	
Product Name	BioActive Human ALK (L1198F) Recombinant Protein,Fc Tag
Gene Name	ALK
Source	Full length Human ALK (F856S,A348D), expressed in HEK293 cells.
Alternative names	
SPECIFICATIONS	
Biological Activity	Fully biologically active
Purity	> 95% by SDS-PAGE & HPLC
Endotoxin Level	< 1.0 EU per μg protein as determined by the LAL method
Expression System	HEK293 Cells
Format	Recombinant
Species	Human
Predicted MW	
Actual MW	
Applications	Sandwich ELISA Functional Studies Mass Spectrometry SDS-PAGE HPLC
Form	Lyophilized from sterile PBS, pH 7.29
Concentration	N/A
Stability and Storage	Samples are stable for up to twelve months from date of receipt at -20℃ to -80℃. Store it under sterile conditions at -20℃ to -80℃. It is recommended that the protein be aliquoted for optimal storage. Avoid repeated freeze-thaw cycles.
Reconstitution	Reconstitute with Phosphate Buffered Saline.
BACKGROUND	
Gene Accession	Q9UM73
Gene Alias	Protein names Recommended name ALK tyrosine kinase receptor Curated EC number EC:2.7.10.1 2 Publications (UniProtKB | ENZYME | Rhea) Alternative names Anaplastic lymphoma kinase 1 Publication CD Antigen Name CD246 Gene names Name ALK

	identification of His1124 and Tyr1278 as critical amino acids involved in ATP binding and phosphorylation. The L1198F substitution confers resistance to lorlatinib through steric interference with drug binding. However, L1198F paradoxically enhances binding to crizotinib, negating the effect of C1156Y and resensitizing resistant cancers to crizotinib.
Background	ALK L1198F lies within the protein kinase domain of the Alk protein (UniProt.org). L1198F confers a gain of function to the Alk protein as demonstrated by increased Alk kinase activity and downstream Pi3k and Mapk pathway activation (PMID: 21596819). L1198F mutation of ALK resulted in the conformational change at the inhibitor site and altered the binding affinity of ALK to crizotinib and lorlatinib. L1198F mutation also affected the autoactivation of ALK as supported by the