Recombinant Human CSK/C-Src kinase Protein (GST Tag)

Catalog Number:PKSH030386

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

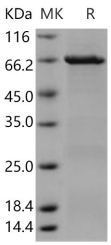
Description

Synonyms	MGC117393;CSK
Species	Human
Expression Host	Baculovirus-Insect Cells
Sequence	Met 1-Leu 450
Accession	NP_004374.1
Calculated Molecular Weight	77.0 kDa
Observed molecular weight	66 kDa
Tag	N-GST
Bioactivity	The specific activity was determined to be 127 nmol/min/mg using Poly(Glu, Tyr) 4:1 as substrate.

Properties

Purity	> 92 % as determined by reducing SDS-PAGE.
Endotoxin	< 1.0 EU per μg of the protein as determined by the LAL method.
Storage	Store at < -20°C, stable for 6 months. Please minimize freeze-thaw cycles.
Shipping	This product is provided as liquid. It is shipped at frozen temperature with blue ice/gel packs. Upon receipt, store it immediately at < - 20°C.
Formulation	Supplied as sterile solution of 20mM Tris, 500mM NaCl, 0.5mM PMSF, pH 7.4
Reconstitution	Not Applicable

Data



> 92 % as determined by reducing SDS-PAGE.

Background

The tyrosine kinase c-Src has been implicated as a modulator of cell proliferation, spreading, and migration. These functions are also regulated by Met. The structure of a large fragment of the c-Src kinase comprises the regulatory and kinase domains and the carboxy-terminal tail. c-Src kinase interactions among domains and is stabilized by binding of the phosphorylated tail to the SH2 domain. This molecule is locked in a conformation that simultaneously disrupts the kinase active site and sequesters the binding surfaces of the SH2 and SH3 domains. The structure shows how appropriate cellular signals, or transforming mutations in v-Src, could break these interactions to produce an open, active kinase. The protein-tyrosine kinase activity of c-Src kinase is inhibited by phosphorylation of tyr527, within the c-Src c-terminal tail. Genetic and biochemical data have suggested that this negative regulation requires an intact Src homology 2 (SH2) domain. Since SH2 domains recognize phosphotyrosine, it is possible that these two non-catalytic domains associate, and thereby repress c-Src kinase activity. Experiments have suggested that c-Src kinase plays a role in the biological behaviour of colonic

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carcinoma cells induced by migratory factors such as EGF, perhaps acting in conjunction with FAK to regulate focal adhesion turnover and tumour cell motility. Furthermore, although c-Src kinase has been implicated in colonic tumour progression, in the adenoma to carcinoma in vitro model c-Src is not the driving force for this progression but co-operates with other molecules in carcinoma development.

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