Recombinant Mouse CSK/C-Src kinase Protein (His &GST Tag)

Catalog Number: PKSM040291

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

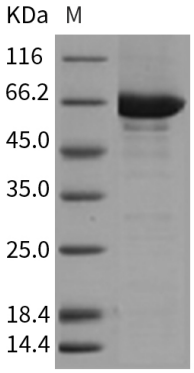
Description

Species	Mouse
Source	Baculovirus-Insect Cells-derived Mouse CSK/C-Src kinase protein Met 1-Leu 450, with an N-terminal His & GST
Mol_Mass	78.5 kDa
Accession	P41241
Bio-activity	The specific activity was determined to be 70 nmol/min/mg using Poly(Glu, Tyr) 4:1 as substrate.

Properties

Purity	> 85 % 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, pH 8.0, 10% glycerol
Reconstitution	Not Applicable

Data



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Background

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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 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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