Data Sheet (Cat.No.TMPY-04444)

TargetMol

CSK Protein, Mouse, Recombinant (His & GST)

General Information

Synonyms:	AW212630;p50CSK;c-src tyrosine kinase
Protein Construction:	A DNA sequence encoding the mouse CSK (P41241?) (Met 1-Leu 450) was fused with the N-terminal polyhistidine-tagged GST tag at the N-terminus. Predicted N terminal: Met
Species:	Mouse
Expression Host:	Baculovirus Insect Cells
Accession:	P41241
Molecular Weight:	78.5 kDa (predicted); 65 kDa (reducing conditions)

QC Testing

Biological Activity:	The specific activity was determined to be 70 nmol/min/mg using Poly(Glu,Tyr) 4:1 as substrate.
Purity:	> 85 % as determined by SDS-PAGE
Endotoxin:	< 1.0 EU/μg of the protein as determined by the LAL method.
Formulation:	Supplied as sterile 20 mM Tris, 500 mM NaCl, pH 8.0, 10% glycerol.

Preparation and Storage

Reconstitution:

A Certificate of Analysis (CoA) containing reconstitution instructions is included with the products. Please refer to the CoA for detailed information.

Stability & Storage:

It is recommended to store the product under sterile conditions at -20°C to -80°C. Samples are stable for up to 12 months. Please avoid multiple freeze-thaw cycles and store products in aliquots.

Actual storage temperature shall be subject to the COA.

Shipping:

Proteins are shipped with blue ice.

Protein 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 tall. 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.References

Reference

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Tel:781-999-4286 E_mail:info@targetmol.com Address:34 Washington Street,Wellesley Hills,MA 02481