

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

Catalog Number: 50893-M20B



Sino Biological
Biological Solution Specialist

General Information

Gene Name Synonym:

AW212630; p50CSK

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.

Source: Mouse

Expression Host: Baculovirus-Insect Cells

QC Testing

Purity: > 85 % as determined by SDS-PAGE

Bio Activity:

The specific activity was determined to be 70 nmol/min/mg using Poly(Glu,Tyr) 4:1 as substrate.

Endotoxin:

< 1.0 EU per µg of the protein as determined by the LAL method

Stability:

Samples are stable for up to twelve months from date of receipt at -70 °C

Predicted N terminal: Met

Molecular Mass:

The secreted recombinant mouse CSK/GST chimera consists of 687 amino acids and has a calculated molecular mass of 78.5 kDa. The recombinant protein migrates as an approximately 65 kDa band in SDS-PAGE under reducing conditions.

Formulation:

Supplied as sterile 20mM Tris, 500mM NaCl, pH 8.0, 10% glycerol

Normally 5 % - 8 % trehalose, mannitol and 0.01% Tween80 are added as protectants before lyophilization. Specific concentrations are included in the hardcopy of COA. Please contact us for any concerns or special requirements.

Usage Guide

Storage:

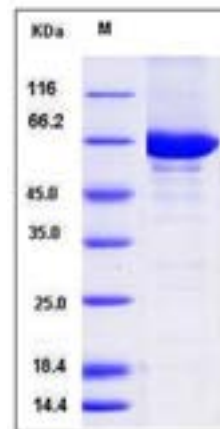
Store it under sterile conditions at -20°C to -80°C upon receiving. Recommend to aliquot the protein into smaller quantities for optimal storage.

Avoid repeated freeze-thaw cycles.

Reconstitution:

Detailed reconstitution instructions are sent along with the products.

SDS-PAGE:



Protein Description

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. References

References

1.Brauninger A. et al.,1992, Gene. 110: 205-11. 2.Sondhi D. et al., 1999, Biochemistry. 38 (34): 11147-55. 3.Ogawa A. et al., 2002, J Biol Chem. 277 (17): 14351-4.

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