

CSTB Human

Description: CSTB Human Recombinant fused to His-Tag at N-Terminus produced in E.Coli is a single, non-glycosylated polypeptide chain containing 118 amino acids and having a molecular mass of 13 kDa.

Catalog #: PRPS-616

For research use only.

Synonyms: Cystatin-B, Stefin-B, Liver thiol proteinase inhibitor, CPI-B, CSTB, CST6, EPM1, PME, STFB.

Source: Escherichia Coli.

Physical Appearance: Sterile Filtered colorless solution.

Amino Acid Sequence: MGSSHHHHHH SSGLVPRGSH MMCGAPSATQ PATAETQHIA
DQVRSQLEEK ENKKFPVFKA VSFKSQVVAG TNYFIKVHVG DEDFVHLRVF QSLPHENKPL
TLSNYQTNKA KHDELTYF.

Purity: Greater than 95.0% as determined by SDS-PAGE.

Formulation:

The protein solution contains 20mM Tris-HCl pH-8 & 50mM NaCl.

Stability:

Store at 4°C if entire vial will be used within 2-4 weeks. Store, frozen at -20°C for longer periods of time. For long term storage it is recommended to add a carrier protein (0.1% HSA or BSA). Avoid multiple freeze-thaw cycles.

Usage:

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Introduction:

Type 1 cystatins are also called stefins which function as intracellular thiol protease inhibitors. Cystatin-B protein is able to form a dimer stabilized by noncovalent forces, inhibiting papain and cathepsins I, h and b. CSTB protein protects proteases leakage from lysosomes. Mutations in Stefin-B gene cause primary defects in patients with progressive myoclonic epilepsy (EPM1), a degenerative disease of the central nervous system. CSTB is overexpressed & elevated in the serum of HCC patients. Cystatin-B in vivo has a polymeric structure which is sensitive to the redox environment. Cystatin-B inhibits bone resorption by down-regulating intracellular cathepsin K activity despite increased osteoclast survival. Protein and mRNA levels of stefin B are significantly lower in atypical benign meningiomas. Stefins-A & Stefin-B which belong to the type-1 Cystatins, are up-regulated in lung tumours and thus able to counteract harmful tumour-associated proteolytic activity. Human stefin-A & Stefin-B form amyloid fibrils. Copper binding by stefin-B reduces amyloid fibril formation. A number of alternatively spliced CSTB isoforms were recognized in patients with progressive myoclonus epilepsy. Decreased CSTB activity in EPM1 pathogenesis is controlled by cathepsins through increased activity of cathepsin-S & cathepsin-L.

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