Human KRAS / K-Ras (12 Asp) Protein (His Tag)

Catalog Number: 12259-H07E1

Sino Biological
Biological Solution Specialist

General Information

Gene Name Synonym:

C-K-RAS; CFC2; K-RAS; K-RAS2A; K-RAS2B; K-RAS4A; K-RAS4B; KI-RAS; KRAS1; KRAS2; NS; NS3; RALD; RASK2

Protein Construction:

A DNA sequence encoding the human KRAS (AAH13572.1) (Thr2-Cys185, natural variant Gly 12 Asp) was expressed with a polyhistide tag at the N-terminus.

Source: Human

Expression Host: E. coli

QC Testing

Purity: > 85 % as determined by SDS-PAGE

Endotoxin:

Please contact us for more information.

Stability:

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

Predicted N terminal: Met

Molecular Mass:

The recombinant human KRAS consists of 202 amino acids and has a calculated molecular mass of 23.3 kDa.

Formulation:

Lyophilized from sterile PBS, pH 7.4.

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:

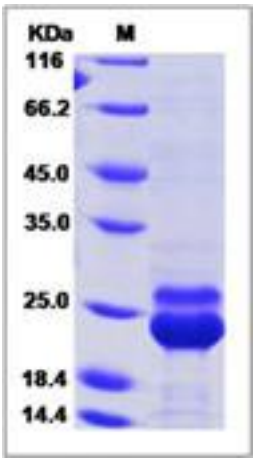
Store it under sterile conditions at -20℃ to -80℃ upon receiving. Recommend to aliquot the protein into smaller quantities for optimal storage.

Avoid repeated freeze-thaw cycles.

Reconstitution:

Detailed reconstitution instructions are sent along with the products.

SDS-PAGE:



Protein Description

K-Ras belongs to the small GTPase superfamily, Ras family. As other members of the Ras family, K-Ras is a GTPase and is an early player in many signal transduction pathways. It is usually tethered to cell membranes because of the presence of an isoprenyl group on its C-terminus. K-Ras functions as a molecular on/off switch. Once it is turned on it recruits and activates proteins necessary for the propagation of growth factor and other receptors' signal, such as c-Raf and PI 3-kinase. It binds to GTP in the active state and possesses an intrinsic enzymatic activity which cleaves the terminal phosphate of the nucleotide converting it to GDP. Upon conversion of GTP to GDP, K-Ras is turned off. The rate of conversion is usually slow but can be sped up dramatically by an accessory protein of the GTPase activating protein class, for example RasGAP. In turn K-Ras can bind to proteins of the Guanine Nucleotide Exchange Factor class, for example SOS1, which forces the release of bound nucleotide. Subsequently, K-Ras binds GTP present in the cytosol and the GEF is released from ras-GTP. Besides essential function in normal tissue signaling, the mutation of a K-Ras gene is an essential step in the development of many cancers. Several germline K-Ras mutations have been found to be associated with Noonan syndrome[4] and cardio-facio-cutaneous syndrome. Somatic K-Ras mutations are found at high rates in Leukemias, colon cancer, pancreatic cancer and lung cancer.

References

1.Ling J, *et al.* (2012) KrasG12D-induced IKK2//NF-B activation by IL-1 alpha and p62 feedforward loops is required for development of pancreatic ductal adenocarcinoma. Cancer Cell. 21(1):105-20. 2.Matallanas D, *et al.* (2011) Mutant K-Ras activation of the proapoptotic MST2 pathway is antagonized by wild-type K-Ras. Mol Cell. 44(6):893-906. 3.Regala RP, *et al.* (2011) Matrix metalloproteinase-10 promotes Kras-mediated bronchio-alveolar stem cell expansion and lung cancer formation. PLoS One. 6(10):e26439.