Human HRAS / GTPase Hras Protein (His Tag)

Catalog Number: 12059-H08B

General Information

Gene Name Synonym:

C-BAS/HAS; C-H-RAS; C-HA-RAS1; CTLO; H-RASIDX; HAMSV; HRAS1; p21ras; RASH1

Protein Construction:

A DNA sequence encoding the human HRAS (P01112) (Met 1-Cys 186) was fused with a polyhistidine tag at the C-terminus.

Source: Human

Expression Host: Baculovirus-Insect Cells

QC Testing

Purity: > 94 % as determined by SDS-PAGE

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 ℃

Predicted N terminal: Met 1

Molecular Mass:

The recombinant human HRAS consists of 197 amino acids and predicts a molecular mass of 22.45 kDa. It migrates as an approximately 23 kDa band in SDS-PAGE in SDS-PAGE under reducing conditions.

Formulation:

Lyophilized from sterile 50mM Tris, 100mM NaCl, pH 8.0, 10% gly

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:

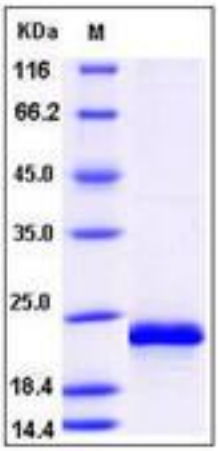
Store it under sterile conditions at -20℃ to -80℃ upon receiving. Recommend to aliquot the protein into smaller quantities for optimal storage.

Avoid repeated freeze-thaw cycles.

Reconstitution:

Detailed reconstitution instructions are sent along with the products.

SDS-PAGE:



Protein Description

HRas, also known as HRAS, belongs to the small GTPase superfamily, Ras family and is widely expressed. It functions in signal transduction pathways. HRas can bind GTP and GDP, and they have intrinsic GTPase activity. It undergoes a continuous cycle of de- and re-palmitoylation, which regulates its rapid exchange between the plasma membrane and the Golgi apparatus. Defects in HRAS are the cause of faciocutaneoskeletal syndrome (FCSS). FCSS is arare condition characterized by prenatally increased growth, postnatal growth deficiency, mental retardation, distinctive facial appearance, cardiovascular abnormalities, tumor predisposition, skin and musculoskeletal abnormalities. Defects in HRAS also can cause congenital myopathy with excess of muscle spindles. HRAS deficiency may be a cause of susceptibility to Hurthle cell thyroid carcinoma. It has been shown that defects in HRAS can cause susceptibility to bladder cancer which is a malignancy originating in tissues of the urinary bladder. It often presents with multiple tumors appearing at different times and at different sites in the bladder. Most bladder cancers are transitional cell carcinomas. They begin in cells that normally make up the inner lining of the bladder. Other types of bladder cancer include squamous cell carcinoma (cancer that begins in thin, flat cells) and adenocarcinoma (cancer that begins in cells that make and release mucus and other fluids). Bladder cancer is a complex disorder with both genetic and environmental influences. Defects in HRAS are the cause of oral squamous cell carcinoma.

References

1.Schulten HJ, *et al.* (2011) Mutational screening of RET, HRAS, KRAS, NRAS, BRAF, AKT1, and CTNNB1 in medullary thyroid carcinoma. Anticancer Res. 31(12):4179-83. 2.Gripp KW, *et al.* (2011) Molecular confirmation of HRAS p.G12S in siblings with Costello syndrome. Am J Med Genet A. 155A(9):2263-8. 3.Na KY, *et al.* (2012) Allelic loss of susceptibility loci and the occurrence of BRAF and RAS mutations in patients with familial non-medullary thyroid cancer. J Surg Oncol. 105(1):10-4.