abcam

Product datasheet

Recombinant Human GTPase HRAS protein ab93949

2 References 1 Image

Description

Product name Recombinant Human GTPase HRAS protein

Purity > 90 % SDS-PAGE.

ab93949 was purified using conventional chromatography techniques.

Expression system Escherichia coli

Accession P01112

Protein length Full length protein

Animal free No

Nature Recombinant

Species Human

Sequence MTEYKLVVVG AGGVGKSALT IQLIQNHFVD EYDPTIEDSY

RKQVVIDGET CLLDILDTAG QEEYSAMRDQ YMRTGEGFLC VFAINNTKSF EDIHQYREQI KRVKDSDDVP MVLVGNKCDL AARTVESRQA

QDLARSYGIP YIETSAKTRQ GVEDAFYTLV REIRQHKLRK

LNPPDESGPG CMSCKCLEHH HHHH

Predicted molecular weight 22 kDa

Amino acids 1 to 186

Tags His tag C-Terminus

Specifications

Our Abpromise guarantee covers the use of ab93949 in the following tested applications.

The application notes include recommended starting dilutions; optimal dilutions/concentrations should be determined by the end user.

Applications Mass Spectrometry

SDS-PAGE

Form Liquid

Preparation and Storage

Stability and Storage Shipped at 4°C. Store at +4°C short term (1-2 weeks). Upon delivery aliquot. Store at -20°C or -

80°C. Avoid freeze / thaw cycle.

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Constituents: 0.316% Tris HCl, 20% Glycerol (glycerin, glycerine), 0.58% Sodium chloride

General Info

Function

Involvement in disease

Ras proteins bind GDP/GTP and possess intrinsic GTPase activity.

Defects in HRAS are the cause of faciocutaneoskeletal syndrome (FCSS) [MIM:218040]. A rare condition characterized by prenatally increased growth, postnatal growth deficiency, mental retardation, distinctive facial appearance, cardiovascular abnormalities (typically pulmonic stenosis, hypertrophic cardiomyopathy and/or atrial tachycardia), tumor predisposition, skin and musculoskeletal abnormalities.

Defects in HRAS are the cause of congenital myopathy with excess of muscle spindles (CMEMS) [MIM:218040]. CMEMS is a variant of Costello syndrome.

Defects in HRAS may be a cause of susceptibility to Hurthle cell thyroid carcinoma (HCTC) [MIM:607464]. Hurthle cell thyroid carcinoma accounts for approximately 3% of all thyroid cancers. Although they are classified as variants of follicular neoplasms, they are more often multifocal and somewhat more aggressive and are less likely to take up iodine than are other follicular neoplasms.

Note=Mutations which change positions 12, 13 or 61 activate the potential of HRAS to transform cultured cells and are implicated in a variety of human tumors.

Defects in HRAS are a cause of susceptibility to bladder cancer (BLC) [MIM:109800]. 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.

Note=Defects in HRAS are the cause of oral squamous cell carcinoma (OSCC).

Sequence similarities

Post-translational modifications

Cellular localization

Belongs to the small GTPase superfamily. Ras family.

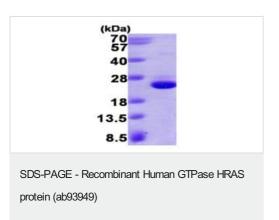
Palmitoylated by the ZDHHC9-GOLGA7 complex. A continuous cycle of de- and re-palmitoylation regulates rapid exchange between plasma membrane and Golgi.

S-nitrosylated; critical for redox regulation. Important for stimulating guanine nucleotide exchange.

No structural perturbation on nitrosylation.

Cell membrane. Golgi apparatus membrane. The active GTP-bound form is localized most strongly to membranes than the inactive GDP-bound form (By similarity). Shuttles between the plasma membrane and the Golgi apparatus.

Images



15% SDS-PAGE analysis of ab93949 (3µg)

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