abcam

Product datasheet

Recombinant Human H-Ras (G12V) protein (Tagged) ab90627

1 References

Description

Product name	Recombinant Human H-Ras (G12V) protein (Tagged)
Purity	> 95 % SDS-PAGE.
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 CMSCKCVLS
Predicted molecular weight	21 kDa
Amino acids	1 to 189
Modifications	mutated G12V
Tags	GST tag N-Terminus
Additional sequence information	(NP_005334)

Specifications

Our **Abpromise guarantee** covers the use of **ab90627** in the following tested applications.

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

Applications	SDS-PAGE Functional Studies
Form	Liquid
Additional notes	The mutation G12V leads to elimination of the intrinsic GTPase activity.

ab90627 is effective in activation of PI3K and PKB.

Preparation and Storage

Stability and Storage

Shipped on dry ice. Upon delivery aliquot and store at -80ºC. Avoid freeze / thaw cycles.

pH: 7.50
Constituents: 0.87% Sodium chloride, 0.395% Tris HCl, 0.039% Beta mercaptoethanol, 0.0475% Magnesium chloride

General Info

Function

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

Tissue specificity

Widely expressed.

Involvement in disease

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

Belongs to the small GTPase superfamily. Ras family.

Post-translational modifications

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.
The covalent modification of cysteine by 15-deoxy-Delta12,14-prostaglandin-J2 is autocatalytic and reversible. It may occur as an alternative to other cysteine modifications, such as S-nitrosylation and S-palmitoylation.

Cellular localization

Cell membrane. Cell membrane. Golgi apparatus. 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 and Nucleus. Cytoplasm. Cytoplasm > perinuclear region. Colocalizes with GNB2L1 to the perinuclear region.

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