# abcam

### Product datasheet

## Recombinant Human Ras protein ab61239

#### 1 Image

**Description** 

Product name Recombinant Human Ras protein

**Purity** > 97 % Densitometry.

**Expression system** Escherichia coli

Protein length Full length protein

Animal free No

Nature Recombinant

**Species** Human

Tags GST tag N-Terminus

#### **Specifications**

Our <u>Abpromise guarantee</u> covers the use of ab61239 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

Form Liquid

#### **Preparation and Storage**

**Stability and Storage** Shipped on dry ice. Upon delivery aliquot and store at -80°C. Avoid freeze / thaw cycles.

pH: 7.50

Constituents: 0.0038% EGTA, 0.00174% PMSF, 0.00385% DTT, 0.79% Tris HCl, 0.00292%

EDTA, 25% Glycerol (glycerin, glycerine), 0.87% Sodium chloride

#### **General Info**

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

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

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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

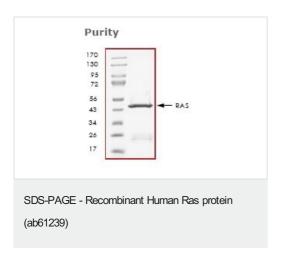
Palmitoylated by the ZDHHC9-GOLGA7 complex. A continuous cycle of de- and re-palmitoylation regulates rapid exchange between plasma membrane and Golgi.

S-nitrosylated; critical for redox regulation. Important for stimulating guanine nucleotide exchange. No structural perturbation on nitrosylation.

**Cellular localization** 

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**



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