Product datasheet

Anti-GTPase HRAS antibody [Y13-259] ab201054

1 References

Overview

Product name: Anti-GTPase HRAS antibody [Y13-259]
Description: Rat monoclonal [Y13-259] to GTPase HRAS
Host species: Rat
Specificity: ab201054 reacts with the phosphorylated and non-phosphorylated forms of Rat, Human and Mouse GTPase HRAS. It does not cross-react with the rap protein. The epitope recognized by ab201054 is comprised by amino acids 62-76 of Human GTPase HRAS.

Tested applications: Suitable for: WB, ICC/IF, IP, IHC-P, Neutralising, IHC-Fr
Species reactivity: Reacts with: Mouse, Rat, Human
Immunogen: Recombinant full length protein corresponding to Human GTPase HRAS aa 62-76. Database link: P01112

Properties

Form: Liquid
Storage buffer: pH: 7.4
Preservative: 0.1% Sodium azide
Constituent: 99% PBS
Purity: Affinity purified
Clonality: Monoclonal
Clone number: Y13-259
Isotype: IgG1
Light chain type: kappa

Applications

Our Abpromise guarantee covers the use of ab201054 in the following tested applications.
The application notes include recommended starting dilutions; optimal dilutions/concentrations should be determined by the end user.
**Function**
Ras proteins bind GDP/GTP and possess intrinsic GTPase activity.

**Involvement in disease**
Defects in HRAS are the cause of faciocutaneouskeletal syndrome (FCSS) [MIM:218040]. A rare condition characterized by prenatally increased growth, postnatal growth deficiency, mental retardation, distinctive facial appearance, cardiovascular abnormalities (typically pulmonary 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**

<table>
<thead>
<tr>
<th>Application</th>
<th>Abreviews</th>
<th>Notes</th>
</tr>
</thead>
<tbody>
<tr>
<td>WB</td>
<td></td>
<td>Use a concentration of 2.5 µg/ml.</td>
</tr>
<tr>
<td>ICC/IF</td>
<td></td>
<td>Use a concentration of 2.5 µg/ml.</td>
</tr>
<tr>
<td>IP</td>
<td></td>
<td>Use at an assay dependent concentration. Use 1 µg per sample.</td>
</tr>
<tr>
<td>IHC-P</td>
<td></td>
<td>Use a concentration of 1 - 2 µg/ml. Staining of formalin fixed, paraffin embedded tissue requires pepsin pretreatment. Use saponin to permeabilize.</td>
</tr>
<tr>
<td>Neutralising</td>
<td></td>
<td>Use at an assay dependent concentration. The antibody neutralizes the biological and biochemical activities of n of H-, K- and N-ras p21 by binding to residues E63, S65, A66, M67, Q70 and R73.</td>
</tr>
<tr>
<td>IHC-Fr</td>
<td></td>
<td>Use a concentration of 5 µg/ml.</td>
</tr>
</tbody>
</table>
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.

**Please note:** All products are “FOR RESEARCH USE ONLY. NOT FOR USE IN DIAGNOSTIC PROCEDURES”

---

**Our Abpromise to you: Quality guaranteed and expert technical support**

- Replacement or refund for products not performing as stated on the datasheet
- Valid for 12 months from date of delivery
- Response to your inquiry within 24 hours
- We provide support in Chinese, English, French, German, Japanese and Spanish
- Extensive multi-media technical resources to help you
- We investigate all quality concerns to ensure our products perform to the highest standards

If the product does not perform as described on this datasheet, we will offer a refund or replacement. For full details of the Abpromise, please visit [https://www.abcam.com/abpromise](https://www.abcam.com/abpromise) or contact our technical team.

---

**Terms and conditions**

- Guarantee only valid for products bought direct from Abcam or one of our authorized distributors