abcam

Product datasheet

Anti-Ras antibody [EPR3255] (HRP) ab199557

Recombinant RabMAb

1 References 2 Images

Overview

Product name	Anti-Ras antibody [EPR3255] (HRP)
Description	Rabbit monoclonal [EPR3255] to Ras (HRP)
Host species	Rabbit
Conjugation	HRP
Tested applications	**Suitable for:** WB
Species reactivity	**Reacts with:** Human **Predicted to work with:** Mouse, Rat
Immunogen	Synthetic peptide within Human Ras aa 1-100 (N terminal). The exact sequence is proprietary.
Positive control	WB: HEK293 and SHSY-5Y whole cell lysates.
General notes	This product is a recombinant monoclonal antibody, which offers several advantages including: - High batch-to-batch consistency and reproducibility - Improved sensitivity and specificity - Long-term security of supply - Animal-free production For more information see here. Our RabMAb® technology is a patented hybridoma-based technology for making rabbit monoclonal antibodies. For details on our patents, please refer to RabMAb® patents.

Properties

Form	Liquid
Storage instructions	Shipped at 4°C. Store at +4°C short term (1-2 weeks). Upon delivery aliquot. Store at -20°C. Stable for 12 months at -20°C. Store In the Dark.
Storage buffer	pH: 7.4 Preservative: 0.1% Proclin Constituents: 30% Glycerol, PBS, 1% BSA
Purity	Protein A purified
Clonality	Monoclonal
Clone number	EPR3255

Isotype	IgG

Applications

Our Abpromise guarantee covers the use of **ab199557** in the following tested applications.

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

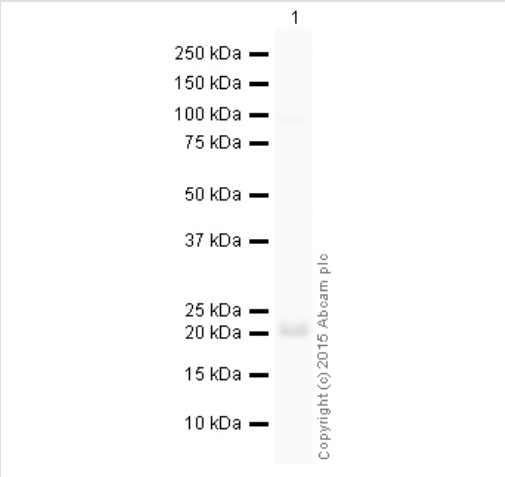
Application	Abreviews	Notes
WB		1/5000. Detects a band of approximately 22 kDa (predicted molecular weight: 22 kDa).

Target

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 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

Images



Western blot - Anti-Ras antibody [EPR3255] (HRP) (ab199557)

Anti-Ras antibody [EPR3255] (HRP) (ab199557) at 1/5000 dilution + HEK293 (Human embryonic kidney cell line) Whole Cell Lysate at 10 µg

Developed using the ECL technique.

Performed under reducing conditions.

Predicted band size: 22 kDa
Observed band size: 22 kDa

Exposure time: 90 seconds

This blot was produced using a 4-12% Bis-tris gel under the MES buffer system. The gel was run at 200V for 35 minutes before being transferred onto a Nitrocellulose membrane at 30V for 70 minutes. The membrane was then blocked for an hour using 3% milk before being incubated with ab199557 overnight at 4°C. Antibody binding was visualised using ECL development solution ab133406.

Western blot - Anti-Ras antibody [EPR3255] (HRP) (ab199557)

Anti-Ras antibody [EPR3255] (HRP) (ab199557) at 1/5000 dilution + SHSY-5Y (Human neuroblastoma cell line) Whole Cell Lysate at 10 µg

Developed using the ECL technique.

Performed under reducing conditions.

Predicted band size: 22 kDa
Observed band size: 22 kDa

Exposure time: 4 minutes

This blot was produced using a 4-12% Bis-tris gel under the MES buffer system. The gel was run at 200V for 35 minutes before being transferred onto a Nitrocellulose membrane at 30V for 70 minutes. The membrane was then blocked for an hour using 3% milk before being incubated with ab199557 overnight at 4°C. Antibody binding was visualised using ECL development solution ab133406.

Please note: All products are "FOR RESEARCH USE ONLY. NOT FOR USE IN DIAGNOSTIC PROCEDURES"

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