Anti-Ras Antibody [JE44-13]

HA721883



Product Type: Recombinant Rabbit monoclonal IgG, primary antibodies

Species reactivity: Human, Mouse, Rat
Applications: WB, IF-Cell, FC

Molecular Wt: Predicted band size: 21 kDa

Clone number: JE44-13

Description: Defects in HRAS are the cause of faciocutaneoskeletal syndrome (FCSS). 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). CMEMS is a variant of Costello syndromDefects in HRAS may be a cause of susceptibility to Hurthle cell thyroid carcinoma (HCTC). 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.

Immunogen: Synthetic peptide within Human Ras aa 1-50 / 189.

Positive control: HEK-293 cell lysate, Jurkat cell lysate, SH-SY5Y cell lysate, MCF7 cell lysate, A431 cell

lysate, A375 cell lysate, C6 cell lysate, NIH/3T3 cell lysate, rat spleen tissue lysate, mouse

spleen tissue lysate, MCF7.

Subcellular location: Cell membrane, Golgi apparatus membrane.

Database links: SwissProt: P01111 Human | P01112 Human | P01116 Human | P08556 Mouse | P32883

Mouse | Q61411 Mouse | P08644 Rat | P20171 Rat | Q04970 Rat

Recommended Dilutions:

WB 1:1,000-1:2,000

IF-Cell 1:100 **FC** 1:1,000

Storage Buffer: 1*TBS (pH7.4), 0.05% BSA, 40% Glycerol. Preservative: 0.05% Sodium Azide.

Storage Instruction: Shipped at 4° C. Store at $+4^{\circ}$ C short term (1-2 weeks). It is recommended to aliquot into

single-use upon delivery. Store at -20 °C long term.

Purity: Protein A affinity purified.

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Images

Fig1: Western blot analysis of Ras on different lysates with Rabbit anti-Ras antibody (HA721883) at 1/1,000 dilution.

Lane 2: Jurkat cell lysate (20 µg/Lane)
Lane 3: SH-SY5Y cell lysate (20 µg/Lane)
Lane 4: MCF7 cell lysate (20 µg/Lane)
Lane 5: A431 cell lysate (20 µg/Lane)
Lane 6: A375 cell lysate (20 µg/Lane)
Lane 7: C6 cell lysate (20 µg/Lane)
Lane 8: NIH/3T3 cell lysate (20 µg/Lane)
Lane 9: Rat spleen tissue lysate (40 µg/Lane)

Lane 10: Mouse spleen tissue lysate (40 µg/Lane)

Lane 1: HEK-293 cell lysate (20 µg/Lane)

Predicted band size: 21 kDa Observed band size: 21 kDa

Exposure time: 25 seconds;

4-20% SDS-PAGE gel.

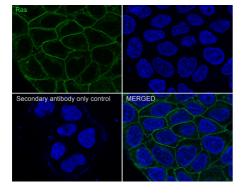


Fig2: Immunocytochemistry analysis of MCF7 cells labeling Ras with Rabbit anti-Ras antibody (HA721883) at 1/100 dilution.

Cells were fixed in 4% paraformaldehyde for 20 minutes at room temperature, permeabilized with 0.1% Triton X-100 in PBS for 5 minutes at room temperature, then blocked with 1% BSA in 10% negative goat serum for 1 hour at room temperature. Cells were then incubated with Rabbit anti-Ras antibody (HA721883) at 1/100 dilution in 1% BSA in PBST overnight at 4 ℃. Goat Anti-Rabbit IgG H&L (iFluor™ 488, HA1121) was used as the secondary antibody at 1/1,000 dilution. PBS instead of the primary antibody was used as the secondary antibody only control. Nuclear DNA was labelled in blue with DAPI.

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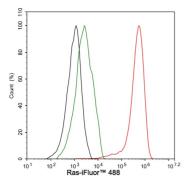


Fig3: Flow cytometric analysis of MCF7 cells labeling Ras.

Cells were fixed and permeabilized. Then stained with the primary antibody (HA721883, 1µg/mL) (red) compared with Rabbit IgG Isotype Control (green). After incubation of the primary antibody at +4 $^{\circ}$ for an hour, the cells were stained with a iFluor † 488 conjugate-Goat anti-Rabbit IgG Secondary antibody (HA1121) at 1/1,000 dilution for 30 minutes at +4 $^{\circ}$. Unlabelled sample was used as a control (cells without incubation with primary antibody; black).

Note: All products are "FOR RESEARCH USE ONLY AND ARE NOT INTENDED FOR DIAGNOSTIC OR THERAPEUTIC USE".

Background References

1. Yin C., Zhu B., Zhang T., Liu T., Chen S., Liu Y., Li X., Miao X., Li S., Cui R. Pharmacological targeting of STK19 inhibits oncogenic NRAS-driven melanomagenesis. Cell 176:1113-1127 (2019)