Anti-KRAS Antibody [A8E6] - BSA and Azide free

HA610008

Product Type:	Recombinant Mouse monoclonal IgG1, primary antibodies
Species reactivity:	Human, Mouse, Rat
Applications:	WB, IHC-P
Molecular Wt:	Predicted band size: 22 kDa
Clone number:	A8E6

Description: The K-Ras protein is a GTPase, a class of enzymes which convert the nucleotide guanosine triphosphate (GTP) into guanosine diphosphate (GDP). In this way the K-Ras protein acts like a switch that is turned on and off by the GTP and GDP molecules. To transmit signals, it must be turned on by attaching (binding) to a molecule of GTP. The K-Ras protein is turned off (inactivated) when it converts the GTP to GDP. When the protein is bound to GDP, it does not relay signals to the cell's nucleus. Several germline KRAS mutations have been found to be associated with Noonan syndrome and cardio-facio-cutaneous syndrome. Somatic KRAS mutations are found at high rates in leukemias, colorectal cancer, pancreatic cancer and lung cancer. KRAS mutations are more commonly observed in cecal cancers than colorectal cancers located in any other places from ascending colon to rectum. KRAS gene can also be amplified in colorectal cancer. Tumors or cell lines harboring this genetic lesion are not responsive to EGFR inhibitors. Although KRAS amplification is an infrequent event in colorectal cancer, it might be responsible for precluding response to anti-EGFR treatment in some patients. Amplification of wild-type Kras has also been observed in ovarian, gastric, uterine, and lung cancers. Driver mutations in KRAS underlie the pathogenesis of up to 20% of human cancers. Hence KRAS is an attractive drug target, however lack of obvious binding sites has hindered pharmaceutical development. One potential drug interaction site is where GTP/GDP binds. However, due to the extraordinarily high affinity of GTP/GDP for this site, it is unlikely that drug-like small molecule inhibitors could compete with GTP/GDP binding. Other than where GTP/GDP binds, there are no obvious high affinity binding sites for small molecules.

Immunogen:	Recombinant protein within human KRAS aa 2-186.
Positive control:	HEK-293 cell lysate, 22RV1 cell lysate, mouse brain tissue lysate, mouse kidney tissue lysate, rat brain tissue lysate, mouse colon tissue, mouse kidney tissue, rat colon tissue.
Subcellular location:	Cell membrane. Cytoplasm.
Database links:	SwissProt: P01116 Human | P32883 Mouse | P08644 Rat
Recommended Dilutions:	
WB	1:500-1:2,000
IHC-P	1:1,000
Storage Buffer:	PBS (pH7.4).
Storage Instruction:	Store at +4℃ after thawing. Aliquot store at -20℃. Avoid repeated freeze / thaw cycles.
Purity:	Protein A affinity purified.

Images

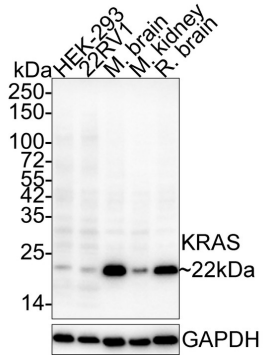

Fig1: Western blot analysis of KRAS on different lysates with Mouse anti-KRAS antibody (HA610008) at 1/2,000 dilution.

Lane 1: HEK-293 cell lysate
Lane 2: 22RV1 cell lysate
Lane 3: Mouse brain tissue lysate
Lane 4: Mouse kidney tissue lysate
Lane 5: Rat brain tissue lysate

Lysates/proteins at 30 µg/Lane.

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

Exposure time: 43 seconds;

4-20% SDS-PAGE gel.

Proteins were transferred to a PVDF membrane and blocked with 5% NFDM/TBST for 1 hour at room temperature. The primary antibody (HA610008) at 1/2,000 dilution was used in 5% NFDM/TBST at 4℃ overnight. Anti-Mouse IgG for IP Nano-secondary antibody (NBI02H) at 1/5,000 dilution was used for 1 hour at room temperature.

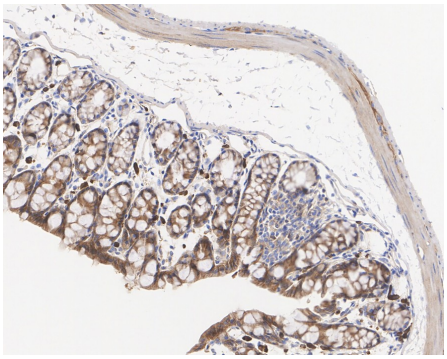

Fig2: Immunohistochemical analysis of paraffin-embedded mouse colon tissue with Mouse anti-KRAS antibody (HA610008) at 1/1,000 dilution.

The section was pre-treated using heat mediated antigen retrieval with Tris-EDTA buffer (pH 9.0) for 20 minutes. The tissues were blocked in 1% BSA for 20 minutes at room temperature, washed with ddH_2O and PBS, and then probed with the primary antibody (HA610008) at 1/1,000 dilution for 1 hour at room temperature. The detection was performed using an HRP conjugated compact polymer system. DAB was used as the chromogen. Tissues were counterstained with hematoxylin and mounted with DPX.

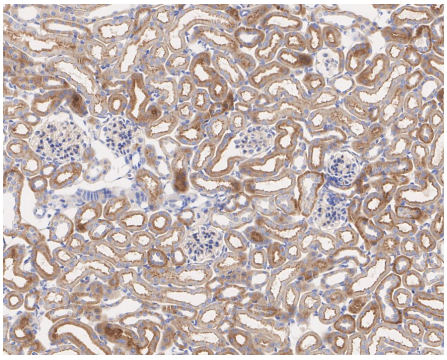

Fig3: Immunohistochemical analysis of paraffin-embedded mouse kidney tissue with Mouse anti-KRAS antibody (HA610008) at 1/1,000 dilution.

The section was pre-treated using heat mediated antigen retrieval with Tris-EDTA buffer (pH 9.0) for 20 minutes. The tissues were blocked in 1% BSA for 20 minutes at room temperature, washed with ddH_2O and PBS, and then probed with the primary antibody (HA610008) at 1/1,000 dilution for 1 hour at room temperature. The detection was performed using an HRP conjugated compact polymer system. DAB was used as the chromogen. Tissues were counterstained with hematoxylin and mounted with DPX.

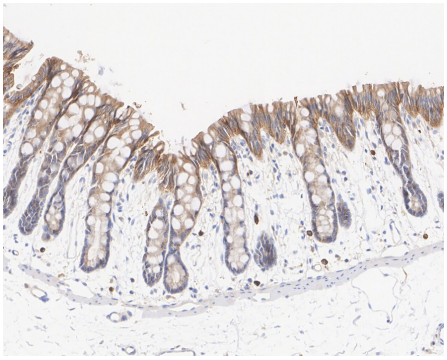

Fig4: Immunohistochemical analysis of paraffin-embedded rat colon tissue with Mouse anti-KRAS antibody (HA610008) at 1/1,000 dilution.

The section was pre-treated using heat mediated antigen retrieval with Tris-EDTA buffer (pH 9.0) for 20 minutes. The tissues were blocked in 1% BSA for 20 minutes at room temperature, washed with ddH_2O and PBS, and then probed with the primary antibody (HA610008) at 1/1,000 dilution for 1 hour at room temperature. The detection was performed using an HRP conjugated compact polymer system. DAB was used as the chromogen. Tissues were counterstained with hematoxylin and mounted with DPX.

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

Background References

1. Christophe Rosty et al. Colorectal carcinomas with KRAS mutation are associated with distinctive morphological and molecular features. Mod Pathol. 2013 Jun;26(6):825-34. doi: 10.1038/modpathol.2012.240. Epub 2013 Jan 25.
2. N Tsuchida, T Ryder, E Ohtsubo. Nucleotide sequence of the oncogene encoding the p21 transforming protein of Kirsten murine sarcoma virus. Science. 1982 Sep 3;217(4563):937-9. doi: 10.1126/science.6287573.