bs-1033R

- ## Rabbit Anti-KRAS Polyclonal Antibody

Primary Antibodies

Background:
This gene, a Kirsten ras oncogene homolog from the mammalian ras gene family, encodes a protein that is a member of the small GTPase superfamily. A single amino acid substitution is responsible for an activating mutation. The transforming protein that results is implicated in various malignancies, including lung adenocarcinoma, mucinous adenoma, ductal carcinoma of the pancreas and colorectal carcinoma. Alternative splicing leads to variants encoding two isoforms that differ in the C-terminal region. [provided by RefSeq]

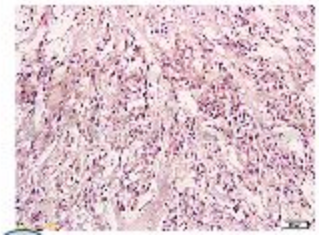
Ras, a proto-oncogene, is a small G-protein that has 3 primary isoforms (H-Ras, N-Ras, and K-Ras) that differ in there approximately 20 C-terminal amino acids. H-Ras was first discovered as a transforming product the retrovirus Harvey murine virus and K-Ras of Kirten sarcoma virus. Ras is a heavily studied target of both academic and pharmaceutical research because of its implications in various pathways and diseases as well as being mutated in a large number of human cancers. Ras is most notably the activator of the Erk/MAPK kinase pathway as activator of Raf, as well as an activator of PI3 Kinase (PI3K). In its oncogenic, mutated state, Ras is unable to hydrolyze GTP to GDP, thus staying in an active state and activating numerous pathways including the MAPK pathway through its activation of Raf, but also others as well that include PI3 Kinase and RalGDS. One path that the pharmaceutical industry has taken to control Ras and its activity is by finding what some consider its Achilles' heel. For its activation, Ras must localize to the plasma membrane, but interestingly, it lacks a transmembrane domain. To achieve this, Ras must first undergo a post-translational modification (PTM) known as prenylation or geranylation at its C-terminal CAAX motif. For this to take place, a controlled three step process must occur. The first step in the process is the prenylation or geranylation of the C in the CAAX motif that is initiated by the covalent attachment of farnesyl groups to the cysteine that is catalyzed by the . After this modification, the and heterodimer enzymes farnesyl transferases –aaX of the motif is proteolytically removed via Rce1 (Ras Converting Enzyme 1), a membrane associated endoprotease, by a mechanism that is still not fully understood. Finally, the C-terminal prenylcysteine is now methlylated by ICMT (Isoprenylcysteine Carboxymethyl Transferase). These drugs have yet to pass clinical trials though and there is doubt that they will ever be successful in treating tumors associated with Ras activation.

Source/Purification:
KLH conjugated synthetic peptide derived from human K-ras N-terminus. Was purified by Protein A and peptide affinity chromatography.

Storage: Prepared as lyophilized powder or liquid and shipped on ice. Store at -20℃ for one year.

Reconstitution:
If the antibody is in liquid form, no reconstitution needed.

Reconstitution is only required for the lyophilized antibody. Please refer to the reconstitution instruction card in the package.



Size: 100ul or 100ug lyophilized

Concentration: 1ug/uL

Host: Rabbit

Reactivities: Human,Mouse,Rat,

Application:

- WB(1:100-500)
- ELISA(1:500-1000)
- IP(1:20-100)
- IHC-P(1:100-500)
- IHC-F(1:100-500)
- IF(1:100-500)
- Not yet tested in other applications. Optimal working dilutions must be determined by the end user.

Antibody Type: Polyclonal

Isotype: IgG

Molecular Weight: 21kDa

Preservatives:
10ug/uL BSA and 0.1% NaN3.