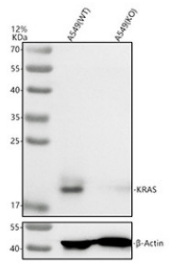
KRAS Antibody / Kirsten Ras Polyclonal Antibody (FY13678)

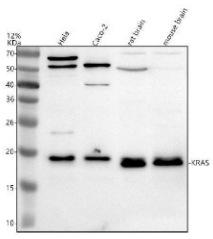
Catalog No.	Formulation	Size
FY13678	0.5mg/ml if reconstituted with 0.2ml sterile DI water	100 ug

Bulk quote request

Species Reactivity	Human, Mouse, Rat
Format	Antigen affinity purified
Host	Rabbit
Clonality	Polyclonal (rabbit origin)
Isotype	Rabbit Ig
Buffer	Each vial contains 4 mg Trehalose, 0.9 mg NaCl, 0.2 mg Na2HPO4.
UniProt	P01116
Applications	Western blot: 0.5-1ug/ml ELISA: 0.1-0.5 ug/ml
Limitations	This KRAS Antibody / Kirsten Ras Polyclonal Antibody is supplied exclusively for research use.



KRAS Antibody WB in A549 Wild-Type and KRAS Knockout Cells. Western blot analysis of human A549 wild-type (lane 1) and KRAS knockout (lane 2) whole-cell lysates using the Kirsten Ras Polyclonal Antibody. A prominent band at approximately 20 kDa is detected in the wild-type sample, with a substantially reduced but faint residual signal at the corresponding position in the knockout sample. The predicted molecular weight of KRAS is approximately 22 kDa. Samples were loaded at 30 ug per lane and separated by 12% SDS-PAGE under reducing conditions. The primary antibody was used at 0.5 ug/ml, with beta-actin serving as the loading control.



KRAS Antibody WB in Human HeLa, Caco-2, Rat Brain, and Mouse Brain. Western blot analysis of human HeLa (lane 1), human Caco-2 (lane 2), rat brain (lane 3), and mouse brain (lane 4) lysates using the Kirsten Ras Polyclonal Antibody. A prominent band is detected at approximately 18-20 kDa in all four samples, consistent with the expected molecular weight of KRAS (~22 kDa). Additional bands at approximately 50-60 kDa are visible in the human cell lysates, but their identities have not been established. Samples were loaded at 30 ug per lane and separated by 12% SDS-PAGE under reducing conditions. The primary antibody was used at 0.5 ug/ml.

Description

KRAS Antibody / Kirsten Ras Polyclonal Antibody targets KRAS, a small GTP-binding protein that functions as a molecular switch in signaling pathways controlling cell proliferation, differentiation, survival, and metabolism. KRAS belongs to the RAS family of small GTPases, which also includes HRAS and NRAS. These proteins transmit signals from activated cell surface receptors to intracellular effectors, particularly those involved in the RAF-MEK-ERK and PI3K-AKT pathways. KRAS is encoded by a proto-oncogene whose normal activity is essential for coordinating cellular responses to growth factors and other extracellular signals.

KRAS alternates between an inactive GDP-bound state and an active GTP-bound state. Guanine nucleotide exchange factors, including SOS proteins, promote the exchange of GDP for GTP following receptor activation. GTP-bound KRAS interacts with downstream effectors such as RAF kinases, initiating signaling cascades that regulate gene expression and cell cycle progression. GTPase-activating proteins accelerate GTP hydrolysis, returning KRAS to its inactive state. This regulatory cycle allows signaling intensity and duration to be controlled according to cellular requirements.

Alternative splicing of the KRAS gene produces two major protein isoforms, KRAS4A and KRAS4B, which differ in their C-terminal regions. These differences influence membrane association, intracellular trafficking, and interactions with regulatory proteins. Both isoforms undergo post-translational processing that contributes to their association with cellular membranes. Membrane localization is important for KRAS signaling because it facilitates interactions with activated receptors, exchange factors, and downstream effectors.

Activating KRAS mutations are among the most frequently identified oncogenic alterations in human cancer. Mutations affecting codons 12, 13, and 61 can impair normal GTPase regulation and promote persistent downstream signaling. KRAS mutations are particularly prevalent in pancreatic ductal adenocarcinoma, colorectal carcinoma, and lung adenocarcinoma. Their biological consequences depend on the specific mutation, tissue environment, and accompanying genetic alterations. The development of mutation-selective KRAS inhibitors has increased interest in understanding KRAS signaling and mechanisms of therapeutic resistance. This KRAS Antibody provides a reagent for investigating KRAS protein expression and its relationship to RAS-dependent signaling, oncogenic transformation, and cancer biology.

Explore related research targets in our Cell Biology Antibodies collection.

Application Notes

Users should optimize the working dilution of the KRAS Antibody / Kirsten Ras Polyclonal Antibody for their specific experimental setup.

Immunogen

The KRAS Antibody was generated using e. coli-derived human Kirsten Ras recombinant protein (amino acids N116-M189).

Storage

Once reconstituted, refrigerate the KRAS Antibody at 4oC for no longer than one month. For extended storage, freeze aliquots at -20oC for up to six months and avoid repeated freeze-thaw cycles.

Alternate Names

K-Ras antibody, Kirsten Ras antibody, Kirsten rat sarcoma viral oncogene homolog antibody, KRAS protein antibody, K-Ras GTPase antibody, KRAS proto-oncogene antibody, Kirsten Ras protein antibody, KRAS small GTPase antibody

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