

Rabbit Anti-KRAS Polyclonal: RC0311, RC0311RTU7

Intended Use: For Research Use Only

Description: The mammalian Ras (also designated v-Ha-Ras, Harvey rat sarcoma viral oncogene homolog, HRAS1, KRAS, NRAS, RASH1 or c-bas/HAS) gene family consists of the Harvey and Kirsten Ras genes (c-H-Ras1 and c-K-Ras2), an inactive pseudogene of each (c-H-Ras2 and c-K-Ras1) and the NRAS gene. The three Ras oncogenes, HRAS, KRAS and NRAS, encode proteins with GTP/GDP binding and GTPase activity. Ras proteins alternate between an inactive form bound to GDP and an active form bound to GTP, activated by a guanine nucleotide-exchange factor (GEF) and inactivated by a GTPase-activating protein (GAP). Ras nomenclature originates from the characterization of human DNA sequences homologous to cloned DNA fragments containing oncogenic sequences of a type C mammalian retrovirus, the Harvey strain of murine sarcoma virus (HaMSV), derived from the rat. Under normal conditions, Ras family members influence cell growth and differentiation events in a subcellular membrane compartmentalization-based signaling system. Oncogenic Ras can deregulate processes that control both cell proliferation and apoptosis.

Specifications

Clone:	Polyclonal
Source:	Rabbit
Isotype:	IgG
Reactivity:	Human, mouse, rat
Immunogen:	Recombinant human KRAS protein
Localization:	Membrane
Formulation:	Purified antibody in PBS pH7.4, containing BSA and $\leq 0.09\%$ sodium azide (NaN ₃)
Storage:	Store at 2°- 8°C
Applications:	IHC, ICC/IF, WB
Package:	

Description	Catalog No.	Size
KRAS Concentrated	RC0311	1 ml
KRAS Prediluted	RC0311RTU7	7 ml

IHC Procedure*

Positive Control Tissue:	Lung cancer, pancreas cancer
Concentrated Dilution:	25-50
Pretreatment:	Tris EDTA pH9.0, 15 minutes Pressure Cooker or 30-60 minutes water bath at 95°-99°C
Incubation Time and Temp:	Overnight at 4°C
Detection:	Refer to the detection system manual
* Result should be confirmed by an established diagnostic procedure.	



Human placenta FFPE tissue stained with anti-KRAS using AEC

References:

1. CSE1L modulates Ras-induced cancer cell invasion: correlation of K-Ras mutation and CSE1L expression in colorectal cancer progression. Jiang MC, et al. Am J Surg. Sep;206(3):418-27, 2013.
2. A detailed immunohistochemical analysis of the PI3K/AKT/mTOR pathway in lung cancer: correlation with PIK3CA, AKT1, K-RAS or PTEN mutational status and clinicopathological features. Trigka EA, et al. Oncol Rep. Aug;30(2):623-36, 2013.

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