

## Summary

Production Name	K-Ras Rabbit Polyclonal Antibody
Description	Rabbit Polyclonal Antibody
Host	Rabbit
Application	WB,IHC-P,IF-P,IF-F,ICC/IF,ELISA
Reactivity	Human, Mouse, Rat

### Performance

Conjugation	Unconjugated
Modification	Unmodified
lsotype	lgG
Clonality	Polyclonal
Form	Liquid
Storage	Store at 4°C short term. Aliquot and store at -20°C long term. Avoid freeze/thaw
	cycles.
Buffer	Liquid in PBS containing 50% glycerol, 0.5% BSA and 0.02% New type preservative N.
Purification	Affinity purification

### Immunogen

Gene Name	KRAS
Alternative Names	GTPase KRas (K-Ras 2;Ki-Ras;c-K-ras;c-Ki-ras) [Cleaved into: GTPase KRas, N-terminally
	processed]
Gene ID	3845.0
SwissProt ID	P01116.The antiserum was produced against synthesized peptide derived from the C-
	terminal region of human KRAS. AA range:150-189

# Application

Dilution Ratio	IHC-P 100-300.WB 1:500-1:2000, ELISA 1:10000, IF-P/IF-F/ICC/IF 1:50-200
Molecular Weight	22kDa



### Background

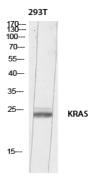
This gene, a Kirsten ras oncogene homolog from the mammalian ras gene family, encodes a protein that is a member of the small GTPase superfamily. A single amino acid substitution is responsible for an activating mutation. The transforming protein that results is implicated in various malignancies, including 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, Jul 2008], alternative products: Isoforms differ in the C-terminal region which is encoded by two alternative exons (IVA and IVB), disease: Defects in KRAS are a cause of acute myelogenous leukemia (AML) [MIM:601626]. AML is a malignant disease in which hematopoietic precursors are arrested in an early stage of development., disease: Defects in KRAS are a cause of cardiofaciocutaneous syndrome (CFC syndrome) [MIM:115150]; also known as cardio-facio-cutaneous syndrome. CFC syndrome is characterized by a distinctive facial appearance, heart defects and mental retardation. Heart defects include pulmonic stenosis, atrial septal defects and hypertrophic cardiomyopathy. Some affected individuals present with ectodermal abnormalities such as sparse, friable hair, hyperkeratotic skin lesions and a generalized ichthyosis-like condition. Typical facial features are similar to Noonan syndrome. They include high forehead with bitemporal constriction, hypoplastic supraorbital ridges, downslanting palpebral fissures, a depressed nasal bridge, and posteriorly angulated ears with prominent helices. The inheritance of CFC syndrome is autosomal dominant., disease: Defects in KRAS are a cause of juvenile myelomonocytic leukemia (JMML) [MIM:607785]. JMML is a pediatric myelodysplastic syndrome that constitutes approximately 30% of childhood cases of myelodysplastic syndrome (MDS) and 2% of leukemia. It is characterized by leukocytosis with tissue infiltration and in vitro hypersensitivity of myeloid progenitors to granulocyte-macrophage colony stimulating factor, disease: Defects in KRAS are the cause of Noonan syndrome 3 (NS3) [MIM:609942]. Noonan syndrome (NS) [MIM:163950] is a disorder characterized by dysmorphic facial features, short stature, hypertelorism, cardiac anomalies, deafness, motor delay, and a bleeding diathesis. It is a genetically heterogeneous and relatively common syndrome, with an estimated incidence of 1 in 1000-2500 live births. Rarely, NS is associated with juvenile myelomonocytic leukemia (JMML). NS3 inheritance is autosomal dominant., disease: KRAS mutations are involved in cancer development.,enzyme regulation: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 GTPaseactivating protein (GAP)., function: Ras proteins bind GDP/GTP and possess intrinsic GTPase activity., online information: The Singapore human mutation and polymorphism database, similarity: Belongs to the small GTPase superfamily. Ras family., subunit: Interacts with PHLPP.,

### **Research Area**

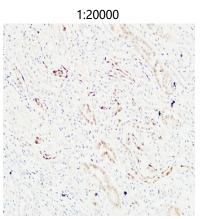
MAPK\_ERK\_Growth;MAPK\_G\_Protein;ErbB\_HER;Chemokine;Dorso-ventral axis formation;Axon guidance;VEGF;Tight junction;Gap junction;Natural killer cell mediated cytotoxicity;T\_Cell\_Receptor;B\_Cell\_Antigen;Fc epsilon RI;Long-term potentiation;Neurotrophin;Long-term depression;Regulates Actin and Cytoskeleton;Insulin\_Receptor;GnRH;Progesterone-mediated oocyte maturation;Melanogenesis;Aldosterone-regulated sodium reabsorption;Pathways in cancer;Colorectal cancer;Renal cell carcinoma;Pancreatic cancer;Endometrial cancer;Glioma;Prostate cancer;Thyroid cancer;Melanoma;Bladder cancer;Chronic myeloid leukemia;Acute myeloid leukemia;Non-small cell lung cancer;



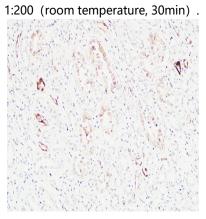
#### Image Data



Western blot analysis of 293T lysis using KRAS antibody. Antibody was diluted at 1:500. Secondary antibody was diluted at

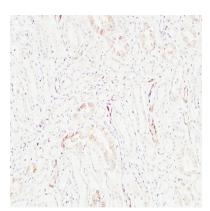


Immunohistochemical analysis of paraffin-embedded Human kidney. 1, Antibody was diluted at 1:200 (4°,overnight) . 2, High-pressure and temperature EDTA, pH8.0 was used for antigen retrieval. 3,Secondary antibody was diluted at



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**Note** For research use only.