Pan ras 抗体

产品货号： mIR1515

英文名称： Ras

中文名称： Pan ras 抗体

别　　名： Pan-ras; Pan ras; K-Ras; P21 protein; p21ras; H-Ras-1; c-H-ras; N-Ras; c bas/has; c H ras; c has/bas p21 protein; C K RAS; c K ras2 protein; c Ki ras; c Kirsten ras protein; c ras Ki 2 protein; C-BAS/HAS; c-H-ras; C-HA-RAS1; Cellular c Ki ras2 proto oncogene; CTLO; G1III6 N ras; GTPase HRas; GTPase KRas; GTPase NRas; H Ras 1; H RasIDX; H-Ras-1; H-RASIDX; Ha Ras; Ha-Ras; HAMSV; HRAS 1; HRAS; HRAS1; K Ras 2; K ras; K ras p21 protein; K RAS2A; K RAS2B; K RAS4A; K RAS4B; K-RAS; KI RAS; Kirsten rat sarcoma 2 viral (v Ki ras2) oncogene homolog; KRAS 1; KRAS 2; KRAS; KRAS1; KRAS2; N ras; N ras oncogene; N-RAS; N-terminally processed; Neuroblastoma RAS viral (v ras) oncogene homolog; NRAS 1; NRAS; NRAS1; NS3; Oncogene KRAS2; p21 protein; p21ras; PR310 c K ras oncogene; RASH 1; RASH_HUMAN; RASH1; RASK 2; RASK2; Transforming protein N Ras; Transforming protein p21; v Ha ras Harvey rat sarcoma viral oncogene homolog; v Ki ras2 Kirsten rat sarcoma 2 viral oncogene homolog; v Ki ras2 Kirsten rat sarcoma viral oncogene homolog; v ras neuroblastoma RAS viral oncogene homolog.

研究领域： 肿瘤

抗体来源： Rabbit

克隆类型： Polyclonal

交叉反应： Human, Mouse, Rat, Dog, Pig, Cow, Horse, Sheep,

产品应用： WB=1:500-2000 ELISA=1:500-1000 IHC-F=1:400-800 IF=1:100-500 （石蜡切片需做抗原修复）
not yet tested in other applications
optimal dilutions/concentrations should be determined by the end user.

分 子 量： 21kDa

细胞定位： 细胞浆 细胞膜

性　　状： Lyophilized or Liquid

浓　　度： 1mg/ml

免 疫 原： KLH conjugated synthetic peptide derived from human ras:

亚　　型： IgG

纯化方法： affinity purified by Protein A

储 存 液： 0.01M TBS(pH7.4) with 1% BSA, 0.03% Proclin300 and 50% Glycerol.

保存条件： Store at -20 ° C for one year. Avoid repeated freeze/thaw cycles. The lyophilized antibody is stable at room temperature for at least one month and for greater than a year when kept at -20° C. When reconstituted in sterile pH 7.4 0.01M PBS or diluent of antibody the antibody is stable for at least two weeks at 2-4 ° C.

PubMed： PubMed

产品介绍： RAS superfamily comprises around 50 related genes encoding GTP-binding domain (G-proteins) involved in signal transduction. The main genes are HRAS, NRAS and KRAS. Ras proteins are membrane-bound GTPases. The inactive form is GDP-bound. They are activated by ligand-binding receptor tyrosine kinases such as EGFR, PDGFR, colony-stimulating factor and fibroblast growth factor. These kinases transiently convert RAS-GDP to RAS-GTP, the active form of RAS. Single amino acid substitutions can activate RAS making it highly oncogenic. Such mutations generally reduce the GTPase activity of RAS, prolonging it in its active GTP-bound form. The consequence of this is sustained activation of the RAF1-MAPK signalling pathway. RAS mutations are found in 10-15% of tumours. A high incidence of RAS mutations is found in pancreatic cancers.

Function:

Ras proteins bind GDP/GTP and possess intrinsic GTPase activity.

Subcellular Location:

Cell membrane. Golgi apparatus membrane. The active GTP-bound form is localized most strongly to membranes than the inactive GDP-bound form (By similarity). Shuttles between the plasma membrane and the Golgi apparatus.

Post-translational modifications:

Palmitoylated by the ZDHHC9-GOLGA7 complex. A continuous cycle of de- and re-palmitoylation regulates rapid exchange between plasma membrane and Golgi. S-nitrosylated; critical for redox regulation. Important for stimulating guanine nucleotide exchange. No structural perturbation on nitrosylation.

DISEASE:

Defects in HRAS are the cause of faciocutaneoskeletal syndrome (FCSS) [MIM:218040]. A rare condition characterized by prenatally increased growth, postnatal growth deficiency, mental retardation, distinctive facial appearance, cardiovascular abnormalities (typically pulmonic stenosis, hypertrophic cardiomyopathy and/or atrial tachycardia), tumor predisposition, skin and musculoskeletal abnormalities.

Defects in HRAS are the cause of congenital myopathy with excess of muscle spindles (CMEMS) [MIM:218040]. CMEMS is a variant of Costello syndrome. Defects in HRAS may be a cause of susceptibility to Hurthle cell thyroid carcinoma (HCTC) [MIM:607464]. Hurthle cell thyroid carcinoma accounts for approximately 3% of all thyroid cancers. Although they are classified as variants of follicular neoplasms, they are more often multifocal and somewhat more aggressive and are less likely to take up iodine than are other follicular neoplasms.

Note=Mutations which change positions 12, 13 or 61 activate the potential of HRAS to transform cultured cells and are implicated in a variety of human tumors.

Defects in HRAS are a cause of susceptibility to bladder cancer (BLC) [MIM:109800]. A malignancy originating in tissues of the urinary bladder. It often presents with multiple tumors appearing at different times and at different sites in the bladder. Most bladder cancers are transitional cell carcinomas. They begin in cells that normally make up the inner lining of the bladder. Other types of bladder cancer include squamous cell carcinoma (cancer that begins in thin, flat cells) and adenocarcinoma (cancer that begins in cells that make and release mucus and other fluids). Bladder cancer is a complex disorder with both genetic and environmental influences.

Note=Defects in HRAS are the cause of oral squamous cell carcinoma (OSCC).

Similarity:

Belongs to the small GTPase superfamily. Ras family.

SWISS:

P01111

Gene ID:

3265

Important Note:

This product as supplied is intended for research use only, not for use in human, therapeutic or diagnostic applications.