磷酸化 Rho 相关蛋白激酶 1 抗体

产品货号： mlR10239

英文名称： phospho-ROCK1 (Thr455+Ser456)

中文名称： 磷酸化 Rho 相关蛋白激酶 1 抗体

别　　名： p-ROCK1(Thr455/Ser456); p160 ROCK1; p160ROCK; Renal carcinoma antigen NY REN 35; Rho associated coiled coil containing protein kinase 1; Rho associated protein kinase 1; Rho-associated coiled-coil containing protein kinase 1; ROCK1_HUMAN.

产品类型： 磷酸化抗体

研究领域： 信号转导　激酶和磷酸酶

抗体来源： Rabbit

克隆类型： Polyclonal

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

产品应用： ELISA=1:500-1000 IHC-P=1:400-800 IHC-F=1:400-800 ICC=1:100-500 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： 158kDa

细胞定位： 细胞浆

性　　状： Lyophilized or Liquid

浓　　度： 1mg/ml

免 疫 原 ： KLH conjugated synthesised phosphopeptide derived from human ROCK1 around the phosphorylation site of Thr455+Ser456:CR(p-T)(p-S)NI

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： The protein encoded by this gene binds copper and zinc ions and is one of two isozymes responsible for destroying free superoxide radicals in the body. The encoded isozyme is a soluble cytoplasmic protein, acting as a homodimer to convert naturally-occuring but harmful superoxide radicals to molecular oxygen and hydrogen peroxide. The other isozyme is a mitochondrial protein. Mutations in this gene have been implicated as causes of familial amyotrophic lateral sclerosis. Rare transcript variants have been reported for this gene. [provided by RefSeq, Jul 2008]

Function:

Destroys radicals which are normally produced within the cells and which are toxic to biological systems.

Subunit:

Homodimer; non-disulfide linked. Homodimerization may take place via the ditryptophan cross-link at Trp-33. The pathogenic variants ALS1 Arg-38, Arg-47, Arg-86 and Ala-94 interact with RNF19A, whereas wild-type protein does not. The pathogenic variants ALS1 Arg-86 and Ala-94 interact with MARCH5, whereas wild-type protein does not.

Subcellular Location:

Cytoplasm. Note=The pathogenic variants ALS1 Arg-86 and Ala-94 gradually aggregates and accumulates in mitochondria.

Post-translational modifications:

Unlike wild-type protein, the pathogenic variants ALS1 Arg-38, Arg-47, Arg-86 and Ala-94 are polyubiquitinated by RNF19A leading to their proteasomal degradation. The pathogenic variants ALS1 Arg-86 and Ala-94 are ubiquitinated by MARCH5 leading to their proteasomal degradation.

The ditryptophan cross-link at Trp-33 is responsible for the non-disulfide-linked homodimerization. Such modification might only occur in extreme conditions and additional experimental evidence is required.

DISEASE:

Defects in SOD1 are the cause of amyotrophic lateral sclerosis type 1 (ALS1) [MIM:105400]. ALS1 is a familial form of amyotrophic lateral sclerosis, a neurodegenerative disorder affecting upper and lower motor neurons and resulting in fatal paralysis. Sensory abnormalities are absent. Death usually occurs within 2 to 5 years. The etiology of amyotrophic lateral sclerosis is likely to be multifactorial, involving both genetic and environmental factors. The disease is inherited in 5-10% of cases leading to familial forms.

Similarity:

Belongs to the Cu-Zn superoxide dismutase family.

SWISS:

Q13464

Gene ID:

6093

Important Note:

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

产品图片