

HCK Antibody

Catalog No: #32589

Package Size: #32589-1 50ul #32589-2 100ul

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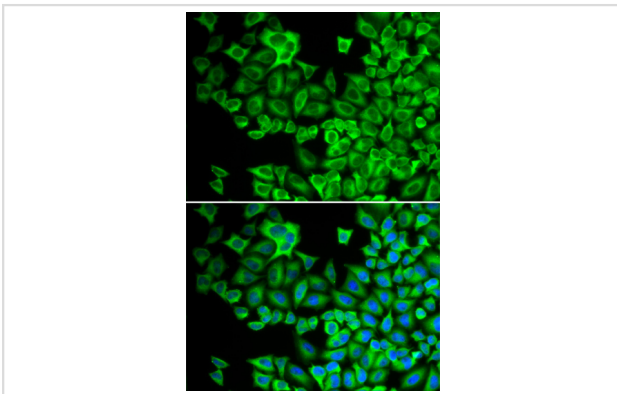
Description

Product Name	HCK Antibody
Host Species	Rabbit
Clonality	Polyclonal
Purification	Antibodies were purified by affinity purification using immunogen.
Applications	WB,IHC,IF
Species Reactivity	Human
Specificity	The antibody detects endogenous level of total HCK protein.
Immunogen Type	Recombinant Protein
Immunogen Description	Recombinant protein of human HCK.
Target Name	HCK
Other Names	JTK9;
Accession No.	Swiss-Prot:P08631NCBI Gene ID:3055
Uniprot	P08631
GeneID	3055;
SDS-PAGE MW	57KD
Concentration	1.0mg/ml
Formulation	Supplied at 1.0mg/mL in phosphate buffered saline (without Mg ²⁺ and Ca ²⁺), pH 7.4, 150mM NaCl, 0.02% sodium azide and 50% glycerol.
Storage	Store at -20°C

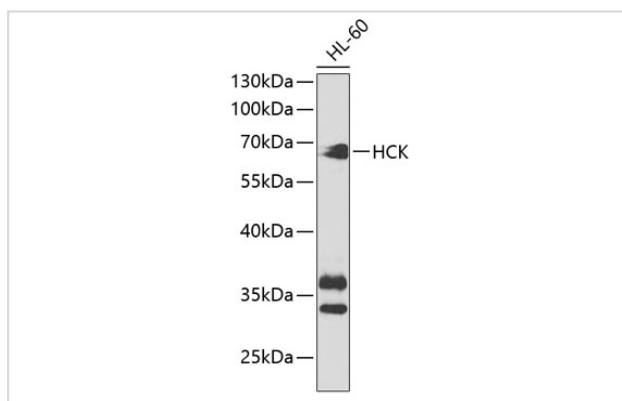
Application Details

WB □ 1:500 - 1:2000 IHC □ 1:50 - 1:200 IF □ 1:10 - 1:100

Images



Immunofluorescence analysis of A549 cells using HCK . Blue: DAPI for nuclear staining.



Western blot analysis of extracts of HL-60 cells, using HCK at 1:1000 dilution.

Background

Hck (hemopoietic cell kinase) is a protein tyrosine kinase of the Src family prominently expressed in the lymphoid and myeloid lineages of hemopoiesis (1). It participates in transducing a variety of extracellular signals, which ultimately affect cellular processes including proliferation, differentiation and migration. The well-defined modular structure of Hck comprises a relatively divergent, NH₂-terminal "unique" domain, which is subject to post-translational lipid modifications thereby targeting Hck to the plasma membrane. Src homology 3 (SH3) and 2 (SH2) domains, and a tyrosine kinase catalytic domain follow the "unique" domain. The catalytic activity of Hck is regulated, both positively and negatively, by tyrosine phosphorylation of highly conserved tyrosine (Y) residues. Phosphorylation of a single conserved Tyr499 residue in the COOH terminus of Hck by the protein kinase Csk renders Hck inactive as a result of an intramolecular interaction between the phosphorylated tyrosine (pY) residue and its own SH2 domain. Disruption of this interaction, either as a result of dephosphorylation, or substitution of the COOH-terminal regulatory Y residue with phenylalanine (F; e.g., HckY499F), or COOH-terminal truncation mutations as observed in the virally transduced v-Src oncoprotein, results in constitutive activation of Hck. In contrast to phosphorylation of the COOH-terminal regulatory tyrosine residue, autophosphorylation of a tyrosine residue (Tyr388) within the kinase domain of Hck acts to positively regulate its catalytic activity. Thus, activation of Hck requires both disruption of the COOH-terminal regulatory tyrosine-SH2 domain interaction and autophosphorylation of the regulatory tyrosine residue within the kinase domain (2, 3). The dysfunction or dysregulation of Hck may contribute to the pathogenesis of some human leukemias (4).

Note: This product is for in vitro research use only