www.neobiolab.com
info@neobiolab.com
888.754.5670, +1 617.500.7103 United States
0800.088.5164, +44 020.8123.1558 United Kingdom

HCK

Reactivity: Human Mouse Rat

Tested applications:WB IHC IF

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

Calculated MW:57kDa

Observed MW: Refer to Figures

Immunogen:

Recombinant protein of human HCK

Storage Buffer:

Store at -20. Avoid freeze / thaw cycles. Buffer: PBS with 0.02% sodium azide, 50% glycerol, pH7.3.

Synonym:

JTK9

Catalog #:A2083

Antibody Type:

Polyclonal Antibody

Species: Rabbit

Gene ID:3055

Isotype: IgG

Swiss Prot:P08631

Purity: Affinity purification

For research use only.

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).

To place an order, please Click HERE.