ABCLONAL BIOTECHNOLOGY,INC.

Hck Rabbit pab Antibody

Anti Hck antibody

Catalog Number:	A0898	**Quantity:**	100ul
Lot Number:	A00009	**Species:**	Rabbit
Gene ID:	3055	**Swiss Prot:**	P08631

DESCRIPTION

Description Rabbit polyclonal to Human Hck
Species Rabbit
Applications WB ICC IP FC
Reactivity H
Immunogen A synthetic peptide of human Hck
Other Name HCK ;JTK9 ; Tyrosine-protein kinase HCK ; Hemopoietic cell kinase ;p59-HCK/p60-HCK ;

PROPERTIES

Form Liquid
Storage instructions Upon delivery aliquot and store at -20ºC or -80ºC.
Storage buffer PBS with 0.1% Sodium Azide, 50% Glycerol,
Purity Affinity purification
Clonality Polyclonal
Isotype IgG

APPLICATION

WB WB :1/1000-2000
ICC ICC:1/50-100
IP IP:1/20-50
FC FC:1/10-50

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).

1. Quintrell, N. et al. (1987) *Mol. Cell. Biol.* 7, 2267-2275.
2. Ziegler, C.A. et al. (1989) *Mol. Cell. Biol.* 9, 2724-2727.
3. Kefalas, P. et al. (1995) *Int. J. Biochem. Cell. Biol.* 27, 551-563.
4. Hu, Y. et al. (2004) *Nat. Genet.* 36, 453-461.