#14643 Store at -20C

Hck (E1I7F) Rabbit mAb



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Applications: WB, IP	Reactivity: H	Sensitivity: Endogenous	MW (kDa): 59, 61	Rabbit IgG	UniProt ID: #P08631	Entrez-Gene Id: 3055
Product Usage Information	Application			Dilution		
	Western Blotting			1:1000		
	Immunoprecipitation			1:100		

 $\textbf{Storage} \hspace{1.5cm} \textbf{Supplied in 10 mM sodium HEPES (pH 7.5), 150 mM NaCl, 100 } \mu\text{g/ml BSA, 50\% glycerol and less than} \\$

0.02% sodium azide. Store at -20° C. Do not aliquot the antibody.

Specificity / Sensitivity Hck (E117F) Rabbit mAb recognizes endogenous levels of total Hck protein.

Source / Purification Monoclonal antibody is produced by immunizing animals with a synthetic peptide corresponding to

residues surrounding Lys60 of human Hck protein.

Background The hematopoietic cell kinase (Hck) is a Src family protein tyrosine kinase that is prominently expressed in

lymphoid and myeloid lineages of hematopoiesis (1). The Hck kinase participates in transduction of a variety of extracellular signals that affect cellular processes, including cell proliferation, differentiation, and migration. Hck protein structure includes a relatively divergent amino-terminal "unique" domain that is subject to post-translational lipid modifications and targets Hck to the plasma membrane. Src homology 3 (SH3) and 2 (SH2) domains, and a tyrosine kinase catalytic domain are adjacent to the "unique" domain. Research studies indicate that phosphorylation of conserved tyrosine residues positively and negatively regulate Hck catalytic activity. Phosphorylation of Hck at the conserved, carboxy-terminal Tyr499 by protein kinase Csk promotes an interaction between the phosphorylated tyrosine and the SH2 domain, rendering Hck inactive. Disruption of this interaction through dephosphorylation, the replacement of the Tyr522 with phenylalanine, or carboxy-terminal truncation mutations, results in constitutive activation of Hck. Autophosphorylation of Tyr411 within the kinase domain positively regulates Hck catalytic activity. Thus, the activation of Hck requires both disruption of the 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 forms of leukemia (4).

Background References

- 1. Karasek, M. et al. (1978) Cell Tissue Res 195, 547-56.
- 2. Ziegler, S.F. et al. (1989) Mol Cell Biol 9, 2724-7.
- 3. Kefalas, P. et al. (1995) Int J Biochem Cell Biol 27, 551-63.
- 4. Hu, Y. et al. (2004) Nat Genet 36, 453-61.

Species Reactivity Species reactivity is determined by testing in at least one approved application (e.g., western blot).

Western Blot Buffer IMPORTANT: For western blots, incubate membrane with diluted primary antibody in 5% w/v BSA, 1X TBS,

0.1% Tween® 20 at 4°C with gentle shaking, overnight.

Applications Key WB: Western Blotting IP: Immunoprecipitation

Cross-Reactivity Key H: human M: mouse R: rat Hm: hamster Mk: monkey Vir: virus Mi: mink C: chicken Dm: D. melanogaster

X: Xenopus Z: zebrafish B: bovine Dg: dog Pg: pig Sc: S. cerevisiae Ce: C. elegans Hr: horse

GP: Guinea Pig Rab: rabbit All: all species expected

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