

DESCRIPTION

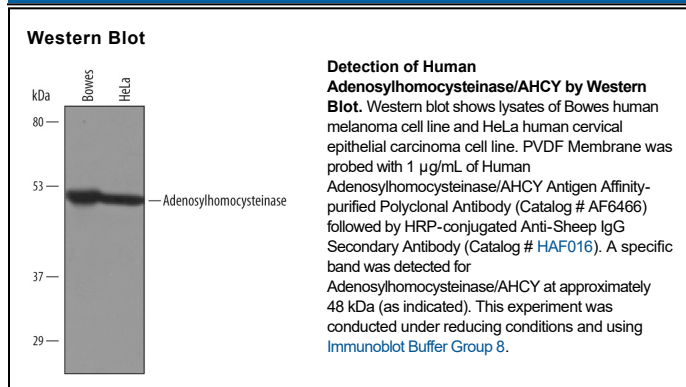
Species Reactivity	Human
Specificity	Detects human Adenosylhomocysteinase/AHCY in direct ELISAs and Western blots.
Source	Polyclonal Sheep IgG
Purification	Antigen Affinity-purified
Immunogen	<i>E. coli</i> -derived recombinant human Adenosylhomocysteinase/AHCY Ser2-Tyr432 Accession # P23526
Formulation	Lyophilized from a 0.2 µm filtered solution in PBS with Trehalose. See Certificate of Analysis for details. *Small pack size (-SP) is supplied either lyophilized or as a 0.2 µm filtered solution in PBS.

APPLICATIONS

Please Note: Optimal dilutions should be determined by each laboratory for each application. *General Protocols* are available in the *Technical Information* section on our website.

	Recommended Concentration	Sample
Western Blot	1 µg/mL	See Below

DATA



PREPARATION AND STORAGE

Reconstitution	Sterile PBS to a final concentration of 0.2 mg/mL.
Shipping	The product is shipped at ambient temperature. Upon receipt, store it immediately at the temperature recommended below. *Small pack size (-SP) is shipped with polar packs. Upon receipt, store it immediately at -20 to -70 °C
Stability & Storage	Use a manual defrost freezer and avoid repeated freeze-thaw cycles. <ul style="list-style-type: none"> • 12 months from date of receipt, -20 to -70 °C as supplied. • 1 month, 2 to 8 °C under sterile conditions after reconstitution. • 6 months, -20 to -70 °C under sterile conditions after reconstitution.

BACKGROUND

Human S-Adenosylhomocysteinase (AHCY) is a cytoplasmic tetramer with a tightly bound NAD co-factor for each subunit (1, 2). It is the only known enzyme to catalyze the breakdown of S-adenosylhomocysteine (AdoHcy) to homocysteine and adenosine. AdoHcy hydrolysis is a reversible reaction with an equilibrium favoring AdoHcy formation, but hydrolysis prevails under physiological conditions due to the rapid removal of adenosine and homocysteine. Thus, AHCY's activity in mammals is directly related to homocysteine level, an independent risk factor for vascular disease (3). It also functions as a regulator of biological transmethylation by controlling the concentration of AdoHcy, a potent competitive inhibitor of all S-adenosyl-L-methionine methyltransferases (1). A mutation in the human AHCY results in AHCY deficiency with increase of plasma creatine kinase, methionine, S-adenosylmethionine and AdoHcy, delay of myelination, myopathy and psychomotor retardation (4, 5).

References:

1. Turner, M. A. *et al.* (2000) *Cell Biochem. Biophys.* **33**:101.
2. Takata, Y. *et al.* (2002) *J. Biol. Chem.* **277**:22670.
3. Gellekink, H. *et al.* (2004) *Eur. J. Hum. Genet.* **12**:942.
4. Baric, I. *et al.* (2004) *Proc. Natl. Acad. Sci. USA.* **101**:4234.
5. Fumic, K. *et al.* (2007) *Eur. J. Hum. Genet.* **15**:347.