Human Adenosylhomocysteinase/AHCY Alexa Fluor® 594-conjugated Antibody

Antigen Affinity-purified Polyclonal Sheep IgG
Catalog Number: AF6466T
100 µg

DESCRIPTION

Species Reactivity	Human
Specificity	Detects human Adenosylhomocysteinase/AHCY in direct ELISAs and Western blots.
Source	Polyclonal Sheep IgG
Purification	Antigen Affinity-purified
Immunogen	*E. coli*-derived recombinant human Adenosylhomocysteinase/AHCY Ser2-Tyr432 Accession # P23526
Conjugate	Alexa Fluor 594 Excitation Wavelength: 590 nm Emission Wavelength: 617 nm
Formulation	Supplied 0.2mg/ml in 1X PBS with RDF1 and 0.09% Sodium Azide *Contains <0.1% Sodium Azide, which is not hazardous at this concentration according to GHS classifications. Refer to the Safety Data Sheet (SDS) for additional information and handling instructions.

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.*

Western Blot	Optimal dilution of this antibody should be experimentally determined.

PREPARATION AND STORAGE

Shipping	The product is shipped with polar packs. Upon receipt, store it immediately at the temperature recommended below.
Stability & Storage	Protect from light. Do not freeze. 12 months from date of receipt, 2 to 8 °C as supplied

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-adonosyl-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).

PRODUCT SPECIFIC NOTICES

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