

DESCRIPTION

Species Reactivity	Human
Specificity	Detects recombinant human MTAP protein in Direct ELISA
Source	Monoclonal Mouse IgG _{2A} Clone # 1113601
Purification	Protein A or G purified from hybridoma culture supernatant
Immunogen	<i>E. coli</i> -derived recombinant human MTAP Accession # Q13126
Conjugate	Alexa Fluor 594 Excitation Wavelength: 590 nm Emission Wavelength: 617 nm
Formulation	Supplied 0.2 mg/mL in a saline solution containing BSA and 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.
Immunocytochemistry	Optimal dilution of this antibody should be experimentally determined.
Immunohistochemistry	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

Methyl-thioadenosine phosphorylase/MTAP is part of the PNP/MTAP phosphorylase family and catalyzes the reversible phosphorylation of S-methyl-5'-thioadenosine (MTA), a major byproduct of polyamine synthesis essential for cell growth and proliferation. MTAP also produces most of the free adenine generated in human cells through a salvage pathway and thus couples the purine salvage pathway with polyamine biosynthesis. MTAP forms an active trimer where each identical 32 kDa monomer contains a separate active site (1). Each active site contains three distinct regions required for base-, methylthioribose-, and sulfate/phosphate-binding (1). MTAP is cytosolic and abundantly expressed in normal cells and tissues (2). In contrast, deficient MTAP expression is observed in many types of tumors including lung, bladder, pancreatic, and endometrial cancer (3) due to hyper-methylation gene suppression (4) or gene deletion (3, 5, 6). MTA accumulation leads to an immunosuppressive tumor microenvironment and apoptotic resistance (7-9) and MTAP directly regulates the level of MTA present. MTAP has been reported as a tumor suppressor (6,10) that may also act in a manner that is independent of enzymatic activity (11) through signaling pathways such as the insulin-like growth factor-1 receptor pathway (12). Potential therapeutic strategies to exploit MTAP deficiency in tumors (13,14) or inhibit MTAP in tumors that express MTAP, such as prostate cancer, are under investigation (15).

References:

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