

## ETHE1 Antibody (Center)

Purified Rabbit Polyclonal Antibody (Pab)
Catalog # AP6641c

#### **Specification**

## ETHE1 Antibody (Center) - Product Information

Application WB, IHC-P, FC,E

Primary Accession <u>095571</u>
Other Accession <u>09DCM0</u>

Reactivity Human, Mouse

Host Rabbit
Clonality Polyclonal
Isotype Rabbit IgG
Calculated MW 27873
Antigen Region 103-130

#### ETHE1 Antibody (Center) - Additional Information

#### **Gene ID 23474**

#### **Other Names**

Persulfide dioxygenase ETHE1, mitochondrial, Ethylmalonic encephalopathy protein 1, Hepatoma subtracted clone one protein, Sulfur dioxygenase ETHE1, ETHE1, HSCO

#### Target/Specificity

This ETHE1 antibody is generated from rabbits immunized with a KLH conjugated synthetic peptide between 103-130 amino acids from the Central region of human ETHE1.

#### **Dilution**

WB~~1:1000 IHC-P~~1:50~100 FC~~1:10~50

#### **Format**

Purified polyclonal antibody supplied in PBS with 0.09% (W/V) sodium azide. This antibody is prepared by Saturated Ammonium Sulfate (SAS) precipitation followed by dialysis against PBS.

#### **Storage**

Maintain refrigerated at 2-8°C for up to 2 weeks. For long term storage store at -20°C in small aliquots to prevent freeze-thaw cycles.

#### **Precautions**

ETHE1 Antibody (Center) is for research use only and not for use in diagnostic or therapeutic procedures.

#### ETHE1 Antibody (Center) - Protein Information

## Name ETHE1



## **Synonyms HSCO**

**Function** Sulfur dioxygenase that plays an essential role in hydrogen sulfide catabolism in the mitochondrial matrix. Hydrogen sulfide (H(2)S) is first oxidized by SQRDL, giving rise to cysteine persulfide residues. ETHE1 consumes molecular oxygen to catalyze the oxidation of the persulfide, once it has been transferred to a thiophilic acceptor, such as glutathione (R-SSH). Plays an important role in metabolic homeostasis in mitochondria by metabolizing hydrogen sulfide and preventing the accumulation of supraphysiological H(2)S levels that have toxic effects, due to the inhibition of cytochrome c oxidase. First described as a protein that can shuttle between the nucleus and the cytoplasm and suppress p53-induced apoptosis by sequestering the transcription factor RELA/NFKB3 in the cytoplasm and preventing its accumulation in the nucleus (PubMed:12398897).

**Cellular Location** 

Cytoplasm. Nucleus. Mitochondrion matrix

**Tissue Location** 

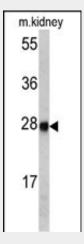
Ubiquitously expressed.

#### ETHE1 Antibody (Center) - Protocols

Provided below are standard protocols that you may find useful for product applications.

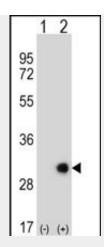
- Western Blot
- Blocking Peptides
- Dot Blot
- Immunohistochemistry
- Immunofluorescence
- Immunoprecipitation
- Flow Cytomety
- Cell Culture

#### ETHE1 Antibody (Center) - Images



Western blot analysis of ETHE1 antibody (Center) (Cat. #AP6641c) in mouse kidney tissue lysates (35ug/lane). ETHE1 (arrow) was detected using the purified Pab.

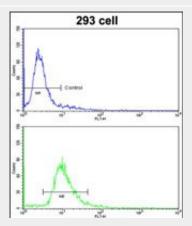




Western blot analysis of ETHE1 (arrow) using rabbit polyclonal ETHE1 Antibody (Center) (Cat. #AP6641c). 293 cell lysates (2 ug/lane) either nontransfected (Lane 1) or transiently transfected (Lane 2) with the ETHE1 gene.



ETHE1 Antibody (Center) (Cat. #AP6641c) immunohistochemistry analysis in formalin fixed and paraffin embedded human colon carcinoma followed by peroxidase conjugation of the secondary antibody and DAB staining. This data demonstrates the use of the ETHE1 Antibody (Center) for immunohistochemistry. Clinical relevance has not been evaluated.



Flow cytometric analysis of 293 cells using ETHE1 Antibody (Center)(bottom histogram) compared to a negative control cell (top histogram). FITC-conjugated goat-anti-rabbit secondary antibodies were used for the analysis.

## ETHE1 Antibody (Center) - Background

ETHE1 is a sulfur dioxygenase that localizes within the mitochondrial matrix. The enzyme functions





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in sulfide catabolism. Mutations in its gene result in ethylmalonic encephalopathy.

# **ETHE1** Antibody (Center) - References

Tiranti, V., Nat. Med. 15 (2), 200-205 (2009) Mineri, R., J. Med. Genet. 45 (7), 473-478 (2008)