

**GSTA1 Antibody (ascites)**  
**Mouse Monoclonal Antibody (Mab)**  
Catalog # AM1932a

## Specification

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### GSTA1 Antibody (ascites) - Product Information

Application	WB,E
Primary Accession	<a href="#">P08263</a>
Other Accession	<a href="#">NP_665683.1</a>
Reactivity	Mouse
Host	Mouse
Clonality	Monoclonal
Isotype	IgM,k
Calculated MW	25631

### GSTA1 Antibody (ascites) - Additional Information

Gene ID 2938

#### Other Names

Glutathione S-transferase A1, GST HA subunit 1, GST class-alpha member 1, GST-epsilon, GSTA1-1, GTH1, Glutathione S-transferase A1, N-terminally processed, GSTA1

#### Target/Specificity

This GSTA1 monoclonal antibody is generated from mouse immunized with GSTA1 recombinant protein.

#### Dilution

WB~~1:1000~32000

#### Format

Mouse monoclonal antibody supplied in crude ascites with 0.09% (W/V) sodium azide.

#### 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

GSTA1 Antibody (ascites) is for research use only and not for use in diagnostic or therapeutic procedures.

### GSTA1 Antibody (ascites) - Protein Information

Name GSTA1

**Function** Glutathione S-transferase that catalyzes the nucleophilic attack of the sulfur atom of glutathione on the electrophilic groups of a wide range of exogenous and endogenous compounds (Probable). Involved in the formation of glutathione conjugates of both prostaglandin A2 (PGA2)

and prostaglandin J2 (PGJ2) (PubMed:[9084911](#)). It also catalyzes the isomerization of D5-androstene-3,17-dione (AD) into D4-androstene- 3,17-dione and may therefore play an important role in hormone biosynthesis (PubMed:[11152686](#)). Through its glutathione-dependent peroxidase activity toward the fatty acid hydroperoxide (13S)-hydroperoxy-(9Z,11E)-octadecadienoate/13-HPODE it is also involved in the metabolism of oxidized linoleic acid (PubMed:[16624487](#)).

#### Cellular Location

Cytoplasm.

#### Tissue Location

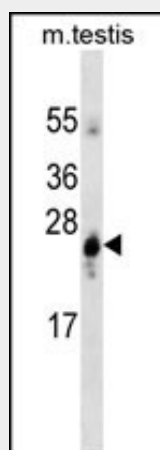
Liver.

### GSTA1 Antibody (ascites) - Protocols

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

- [Western Blot](#)
- [Blocking Peptides](#)
- [Dot Blot](#)
- [Immunohistochemistry](#)
- [Immunofluorescence](#)
- [Immunoprecipitation](#)
- [Flow Cytometry](#)
- [Cell Culture](#)

### GSTA1 Antibody (ascites) - Images



GSTA1 (Cat. #AM1932a) western blot analysis in mouse testis tissue lysates (35µg/lane). This demonstrates the GSTA1 antibody detected the GSTA1 protein (arrow).

### GSTA1 Antibody (ascites) - Background

Cytosolic and membrane-bound forms of glutathione S-transferase are encoded by two distinct supergene families. These enzymes function in the detoxification of electrophilic compounds, including carcinogens, therapeutic drugs, environmental toxins and products of oxidative stress, by conjugation with glutathione. The genes encoding these enzymes are known to be highly polymorphic. These genetic variations can change an individual's susceptibility to carcinogens and toxins as well as affect the toxicity and efficacy of some drugs. At present, eight distinct classes of the soluble cytoplasmic mammalian glutathione S-transferases have been identified: alpha, kappa,

mu, omega, pi, sigma, theta and zeta. This gene encodes a glutathione S-transferase belonging to the alpha class. The alpha class genes, located in a cluster mapped to chromosome 6, are the most abundantly expressed glutathione S-transferases in liver. In addition to metabolizing bilirubin and certain anti-cancer drugs in the liver, the alpha class of these enzymes exhibit glutathione peroxidase activity thereby protecting the cells from reactive oxygen species and the products of peroxidation.

#### **GSTA1 Antibody (ascites) - References**

Elhasid, R., et al. *Pediatr Blood Cancer* 55(6):1172-1179(2010) Hawken, S.J., et al. *Hum. Genet.* 128(1):89-101(2010) Oguztuzun, S., et al. *Folia Histochem. Cytobiol.* 48(1):122-127(2010) Eriksen, K.T., et al. *J. Toxicol. Environ. Health Part A* 73(9):583-595(2010) Nguyen, T.V., et al. *Oncol. Res.* 18(7):349-355(2010)