

TIMP-3 (Tissue Inhibitor of Metalloproteinase-3) Antibody - With BSA and Azide
Rabbit Polyclonal Antibody
Catalog # AH10766**Specification****TIMP-3 (Tissue Inhibitor of Metalloproteinase-3) Antibody - With BSA and Azide - Product Information**

Application	IHC-P, IF, FC
Primary Accession	P35625
Other Accession	7078 , 644633 , 714168
Reactivity	Human, Mouse, Rat, Bovine, Horse, Dog
Host	Rabbit
Clonality	Polyclonal
Isotype	Rabbit / IgG
Calculated MW	30kDa kDa

TIMP-3 (Tissue Inhibitor of Metalloproteinase-3) Antibody - With BSA and Azide - Additional Information**Gene ID** 7078**Other Names**

Metalloproteinase inhibitor 3, Protein MIG-5, Tissue inhibitor of metalloproteinases 3, TIMP-3, TIMP3

Application Note

IHC-P ~ ~ N/A
IF ~ ~ 1:50 ~ 200
FC ~ ~ 1:10 ~ 50

Format

200ug/ml of Ab purified from rabbit anti-serum by Protein A. Prepared in 10mM PBS with 0.05% BSA & 0.05% azide. Also available WITHOUT BSA at 1.0mg/ml.

Storage

Store at 2 to 8°C. Antibody is stable for 24 months.

Precautions

TIMP-3 (Tissue Inhibitor of Metalloproteinase-3) Antibody - With BSA and Azide is for research use only and not for use in diagnostic or therapeutic procedures.

TIMP-3 (Tissue Inhibitor of Metalloproteinase-3) Antibody - With BSA and Azide - Protein Information**Name** TIMP3**Function**

Mediates a variety of processes including matrix regulation and turnover, inflammation, and angiogenesis, through reversible inhibition of zinc protease superfamily enzymes, primarily matrix

metalloproteinases (MMPs). Regulates extracellular matrix (ECM) remodeling through inhibition of matrix metalloproteinases (MMP) including MMP-1, MMP-2, MMP-3, MMP-7, MMP-9, MMP-13, MMP-14 and MMP-15. Additionally, modulates the processing of amyloid precursor protein (APP) and apolipoprotein E receptor ApoER2 by inhibiting two alpha- secretases ADAM10 and ADAM17 (PubMed:17913923). Functions as a tumor suppressor and a potent inhibitor of angiogenesis. Exerts its anti- angiogenic effect by directly interacting with vascular endothelial growth factor (VEGF) receptor-2/KDR, preventing its binding to the VEGFA ligand (PubMed:12652295). Selectively induces apoptosis in angiogenic endothelial cells through a caspase-independent cell death pathway (PubMed:25558000). Mechanistically, inhibits matrix-induced focal adhesion kinase PTK2 tyrosine phosphorylation and association with paxillin/PXN and disrupts the incorporation of ITGB3, PTK2 and PXN into focal adhesion contacts on the matrix (PubMed:25558000).

Cellular Location

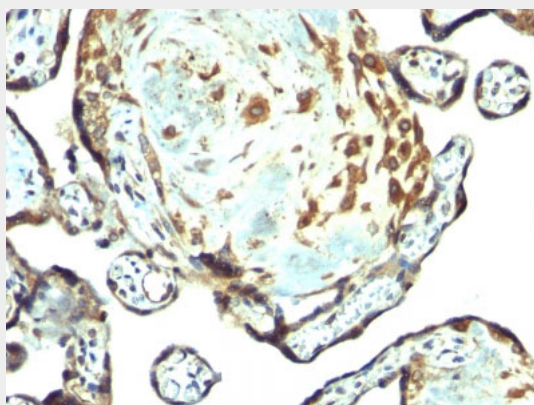
Secreted, extracellular space, extracellular matrix

TIMP-3 (Tissue Inhibitor of Metalloproteinase-3) Antibody - With BSA and Azide - 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](#)

TIMP-3 (Tissue Inhibitor of Metalloproteinase-3) Antibody - With BSA and Azide - Images



Formalin-fixed, paraffin-embedded human Placenta stained with TIMP3 Rabbit Polyclonal Antibody.

TIMP-3 (Tissue Inhibitor of Metalloproteinase-3) Antibody - With BSA and Azide - Background

TIMP3 (tissue inhibitor of metalloproteinases 3), along with family members TIMP1, TIMP2, and

TIMP4, are inhibitors of the matrix metalloproteinases (MMPs), a group of peptidases involved in degradation of the extracellular matrix (ECM). An imbalance between MMPs and the associated TIMPs may play a significant role in the invasive phenotype of malignant tumors. TIMP s inhibit the proteolytic invasiveness of tumor cells and normal placental trophoblast cells. TIMP-3 may be involved in regulating trophoblastic invasion of the uterus as well as in regulating remodeling of the extracellular matrix during the folding of epithelia, and in the formation, branching and expansion of epithelial tubes.

**TIMP-3 (Tissue Inhibitor of Metalloproteinase-3) Antibody - With BSA and Azide -
References**

Nagase, H. et al. (2006) Cardiovasc Res 69, 562-73. | Visse, R. and Nagase, H. (2003) Circ Res 92, 827-39. |