CELL APPLICATIONS, INC.

TIMP-3 Antibody

Cat. No. CA0577

Applications:	**WB, IHC**
Detected MW:	**24 kDa**
Species & Reactivity:	**Human, Mouse, Rat**
Isotype:	**Rabbit IgG**

BACKGROUND

The TIMPs are well-studied inhibitors of MMPs and consist of a family of four structurally related proteins (TIMP-1–4), with core proteins of ~21 kDa. TIMPs inhibit MMP activity by a common mechanism involving interaction of the amino-terminal cysteine residue with the zinc atom at the MMP active site. The TIMPs inhibit MMP activity associated with tumor invasion and angiogenesis. In addition to their MMP-inhibitory activity, it is now widely appreciated that TIMPs have direct effects on cellular behaviors such as cell growth, apoptosis, migration, and differentiation.[1]

The TIMP-3 polypeptide sequence is 37% and 42% similar to the sequences of TIMP-1 and TIMP-2, respectively. The TIMP-3 protein has 188 amino acids. It has a conserved glycosylation site near the C-terminus. Characterisation of the human recombinant TIMP-3 reveals that it has both a 27 kDa glycosylated and a 24 kDa unglycosylated species. Unlike the other TIMPs, which are soluble, TIMP-3 is unique in being a component of extracellular matrix (ECM). TIMP-3 is localised to the ECM in both its glycosylated and unglycosylated forms. In addition, TIMP-3 is the only TIMP to inhibit members of the ADAM (a disintegrin and metalloprotease domain) family such as tumor necrosis factor-α-converting enzyme; this may account for its ability to induce apoptosis. It is the only TIMP to inhibit shedding of L-selectin and interleukin-6 receptors. Moreover, TIMP-3 is the only TIMP directly implicated in a disease process: Ser-Cys mutants of TIMP-3 accumulate in Bruch's membrane of the eye and cause Sorsby's fundus dystrophy. TIMP-3 also promotes the detachment of transformed cells from the ECM and is involved in the formation, branching, and expansion of epithelial tubes and in regulating trophoblast invasion of the uterus.[2] Mutations in the human TIMP-3 gene cause a dominantly inherited, adult-onset blindness (Sorsby's fundus dystrophy or SFD).[3] It was demonstrated that increased expression of TIMP-3 resulted in a statistically significant suppression of tumor growth. Deposition of TIMP-3 in the surrounding ECM by tumor cells may inhibit tumor growth by preventing local expansion of tumor, retarding the release of growth factors sequestered in ECM, or inhibiting angiogenesis. TIMP-3 over-expression had no effect on the growth of the two tumor cell lines in vitro. Because recombinant TIMP-3 inhibits endothelial cell migration and tube formation in response to angiogenic factors, thus, the effect of TIMP-3 on tumor growth seen in this study may be a consequence of its angiostatic action.[4]

References:

1. Gomez, D.E.: Eur J Cell Biol. 74:111-22, 1997
2. Yu, W.H. et al: J. Biol. Chem. 275:31226-32, 2000
3. Weber, B.H.F. et al: Nature Genetics 8:352-56, 1994
4. Anand-Apte, B. et al: Biochem. Cell Biol. 74:853–62, 1996

TECHNICAL INFORMATION

Source: Anti-SCN1B is a rabbit polyclonal antibody raised against a peptide mapping at the C-terminal end of TIMP-3 of human origin different to the related rat sequence on one amino acid.

Specificity and Sensitivity: Reacts specifically with TIMP-3 of human, mouse, and rat origin in immunohistochemical staining and western blotting, no cross-reactivity with other members of the family.

Storage Buffer: PBS and 30% glycerol.

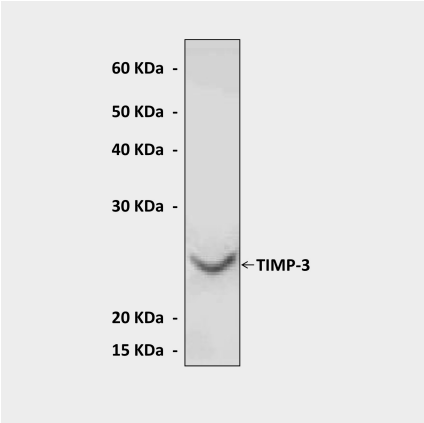
Storage: Store at -20°C for at least one year. Store at 4°C for frequent use. Avoid repeated freeze-thaw cycles

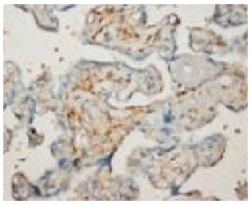
APPLICATIONS

Application:	*Dilution:
WB	1:500 – 1:1000
IP	n/d
IHC	1:25 – 1:100
ICC	n/d
FACS	n/d

**Optimal dilutions must be determined by end user.*

QUALITY CONTROL DATA





Top: Detection of TIMP-3 from rat kidney tissue lysate in Western blot assay, using Anti- TIMP-3. **Bottom:** Immunohistochemical staining of paraffin-embedded human placental tissue, using Anti- TIMP-3.