MONOCLONAL ANTIBODY

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Cat. No. AMS.PRPG-VS-M01

Anti-Versican (5C12)

BACKGROUND

Versican, also known as PG-M, and encoded by the VCAN/CSPG2 gene, is a large extracellular matrix chondroitin sulfate proteoglycan ubiquitously expressed in interstitial matrices of the human body, including brain ECM. It was first described in the bovine aorta by Dick Heinegard's and Anders Malmstrom's groups (1982) and shortly after isolated from the chick embryo by Koji Kimata's group. Cloning of the human VCAN/CSPG2 gene was accomplished in 1989 by Zimmermann and Ruoslahti, who also cognated the name versican in recognition of its versatile modular structure. Versican belongs to the *lectican* proteoglycan subgroup, to which aggrecan, brevican and neurocan also pertain and share the N-terminal (G1) globular domain. This consists of Ig-like loops and two link modules and is responsible for the binding to hyaluronan, which may or may not be further stabilized by link proteins. At least 4 different alternative spliced versican isoforms are known in higher vertebrates, denoted V0, V1, V2 and V3, while lower vertebrates may have additional ones in part by duplication of the gene. These isoforms are generated through differential utilization of the central core protein regions denoted GAG-α and GAG-β and encompassing the glycosaminoglycan (chondroitin sulfate) attachment sites. The V0 isoform is the parental one containing both the above "GAG-attachment" exons; the V1 isoforms has only the GAG-β domain; the V2 isoform has only the GAG-α domain; and the V3 isoform is void of any GAG attachment domain, and is therefore a GAG-free proteoglycan. This implies that the versican isoform core proteins have a molecular mass range of 50-550 kDa and, when taking also into consideration the extensive glycosylation of the versican core protein, the molecular weights of the different isoforms vary from about 60 kDa to 1,500-2,000 kDa. The C-terminal (G3) globular domain consists of one or two EGF repeats, a C-type lectin module and complement regulatory protein (CRP)-like domain. The C-terminal domain binds a variety of ligands in the ECM and thereby contributes to the macromolecular organization of versican.

The role of versican in ECM assembly (in particular elastic matrices), cell adhesion, cell migration, and cell proliferation is extensively described and its essential role during embryonic development is confirmed by the early lethality murine embryos with CSPG2 gene deletion. As many other large proteoglycans, versican is processed by multiple MMPs and ADAMTSs and its matrix deposition may be strongly down- or up-regulated in degenerative diseases and cancer. In some tumours its expression pattern has been proposed to have a prognostic value.

Product type	Primary antibody
Host	Mouse
Source	Hybridoma cell culture
Form	Liquid
	Supernatant with 0.05% NaN_3 as a preservative.
Volume	2 ml
Specificity	Versican V0, V1 and V2 isoforms (V3 isoform reactivity not ascertained)
Antigen	Versican-enriched proteoglycan preparation from bovine aorta
Isotype	IgM

Application notes

Recommended use

WB, IHC(P), ELISA

Recommended dilutions

Western blotting, 1/20 to 1/60

Banding pattern depends upon the isoforms and is often complex. In the intact forms, i.e. without removal of GAGs, V0, V1 and V2 do not enter acrylamide gels and therefore agarose gels are recommended. Following chondroitinase-digestion and extensive enzymatic deglycosylation, most isoforms still show complex, smeared banding patterns.

Immunohistochemistry, 1/25 to 1/75 (PFA-frozen sections)

ELISA, 1/50 – 1/150

Optimal dilutions/concentrations should be determined by the end user.

Staining Pattern

mAb 5C12 stains ubiquitously connective tissue ECMs and particularly concentrates in vascular structures. Chondroitinase ABC pre-digestion of the sections may affect the staining pattern.

Cross reactivity	Human, bovine
Storage	Store at 4°C (-20° C for prolonged storage) Aliquot to avoid cycles of freeze/thaw.

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References

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Cattaruzza S, Schiappacassi M, Ljungberg-Rose Å, Spessotto P, Perissinotto D, Mörgelin M, Mucignat MT, Colombatti A, Perris R. 2002. Distribution of PG-M/versican variants in human tissues and de novo expression of isoform V3 upon endothelial cell activation and neoangiogenesis. *J. Biol. Chem.* 277, 47626-47635.

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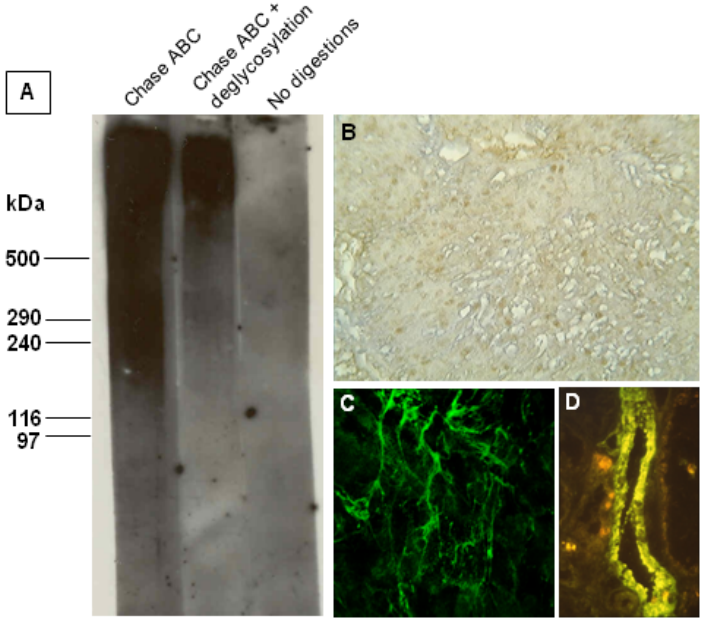
(A) Immunoblotting of intact versican (mixture of V1 and V2 isoforms) in its untreated and chondroitinase ABC (Chase ABC)-digested form, or after combined digestion with chondroitinase ABC and a number of exo- and endoglycosydases. The proteoglycan was resolved by SDS-PAGE under reducing conditions on 3-8% linear gradient gels (MW, HiMark Unstained Protein Standard). (**B**) immunohistochemistry on human normal urinary bladder; (**C**) immunostaining of versican in the matrix deposited in vitro of human microvascular endothelial cells after TNFα stimulation; (**D**) immunostaining of versican lining the wall of a larger vein in human kidney (PFA-fixed frozen section).



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