



MONOCLONAL ANTIBODY

For research use only. Not for clinical diagnosis

Catalog No. PRPG-VS-M02

Anti- Versican [CSPG2] (4C5)

BACKGROUND

Versican, originally 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 that of brain. It was first isolated from the bovine aorta by Dick Heinegard's and Anders Malmstrom's groups (1982) and shortly after isolated from the chick embryonic limbs by Koji Kimata's group (1986). 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. *

Product type	Primary antibodies
Immunogen	Versican-enriched proteoglycan preparation from adult bovine aorta
Rased in	Mouse
Myeloma	-
Clone number	4C5
Isotype	IgM
Host	-
Source	Hybridoma cell culture
Purification	-
Form	Liquid
Storage buffer	Supernatant supplemented with 0.05% NaN ₃
Concentration	ND
Volume	2 mL
Label	Unlabeled
Specificity	Versican V0, V1 and V2 isoforms (V3 isoform recognition not ascertained)
Cross reactivity	Human, Bovine Other species have not been tested.
Storage	Store at 4°C for short-term storage and -20°C for prolonged storage Aliquot to avoid cycles of freeze / thaw.
Other	Data Link : UniProtKB/Swiss-Prot P81282 (CSPG2_BOVIN)

Application notes	WB, IHC(F), ELISA
Recommended dilutions	• Western blotting, 1/20 - 1/40 • Immunohistochemistry, 1/25 - 1/50 (PFA-frozen sections) * • ELISA, 1/50 - 1/150 *<Staining Pattern> Antibody 4C5 stains ubiquitously connective tissue ECMs and detects versican glycosylation isoforms of V0-V2 that are different from those recognized by antibody 5C12 and are highly concentrated in the vasculature and generally highly vascularized organs. Chondroitinase ABC pre-digestion of the sections and various unmasking procedures may affect the staining pattern. Reactivity of the antibody on fixed and paraffin-embedded material has not been fully established.. Other applications have not been tested. Optimal dilutions/concentrations should be determined by the end user.

References	1) Mazzucato, M., <i>et al.</i>, 2002. Vascular PG-M/versican variants promote platelet adhesion at low shear rates and cooperate with collagens to induce aggregation. <i>FASEB J.</i> 16, 1903-1916. 2) Cattaruzza S, <i>et al.</i>, 2002. Distribution of PG-M/versican variants in human tissues and de novo expression of isoform V3 upon endothelial cell activation and neoangiogenesis. <i>J.Biol.Chem.</i> 277, 47626-47635. 3) Cattaruzza S, Perris R. 2005. Proteoglycan control of cell movement during wound healing and cancer. <i>Matrix Biol.</i> 24, 400-417.
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ANTIBODY CHARACTERIZATION

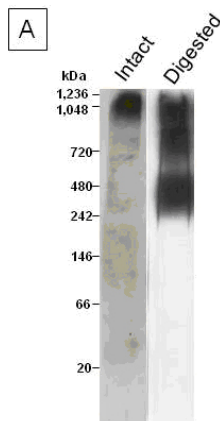


Fig.1 Immunoblotting of intact versican (mixture of V1 and V2 isoforms) prior to (Intact) and after combined chondroitinase ABC (Chase ABC) and endo- β -galactosidase-digestion (Digested) form. The proteoglycan was resolved by SDS-PAGE under reducing conditions on 3-8% linear gradient gels (MW, HiMark Unstained Protein Standard).

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

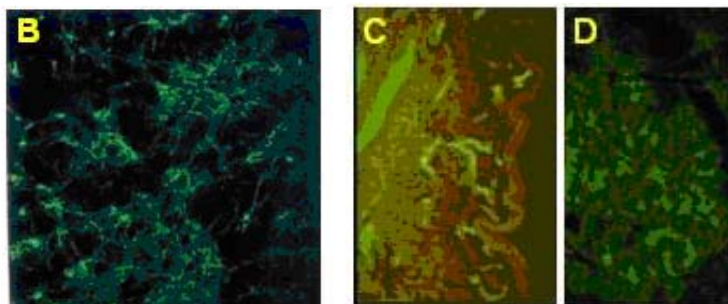


Fig.2 (B) Immunocytochemistry on cultured smooth muscle cells showing versican distribution in the ECM deposited by the cells.
(C) Immunostaining of versican in normal human skin.
(D) Immunostaining of versican distribution in the Bowman capsule of a normal human kidney (PFA-fixed frozen section).

RELATED PRODUCTS:

Product Name	Maker	Cat#
Anti Aggrecan (6F4) Monoclonal Antibody	CAC	PRPG-AG-M01
Anti Aggrecan (5D3) Monoclonal Antibody	CAC	PRPG-AG-M02
Anti Aggrecan (5G2) Monoclonal Antibody	CAC	PRPG-AG-M03
Anti Aggrecan (7B7) Monoclonal Antibody	CAC	PRPG-AG-M04
Anti Versican/CSPG2 (5C12) Monoclonal Antibody	CAC	PRPG-VS-M01
Anti Versican/CSPG2 (4C5) Monoclonal Antibody	CAC	PRPG-VS-M02
Anti NG2 / CSPG4 (2164H5) Monoclonal Antibody	CAC	PRPG-NG-M01
Anti COMP (484D1) Monoclonal Antibody	CAC	PRPG-CP-M01
Anti COMP (490D11) Monoclonal Antibody	CAC	PRPG-CP-M02
Anti Keratan sulfate (373E1) Monoclonal Antibody	CAC	PRPG-KS-M01
Anti Decorin (889C7) Monoclonal Antibody	CAC	PRPG-DC-M01
Anti Fibromodulin (636B12) Monoclonal Antibody	CAC	PRPG-FBM-M01
Anti Biglycan (905A7) Monoclonal Antibody	CAC	PRPG-BG-M01
Anti XTP1 (2191H1) Monoclonal Antibody	CAC	PRPG-XTP-M01
Anti SDP35 (2200D12) Monoclonal Antibody	CAC	PRPG-SDP-M01
Anti Laminin $\alpha 4$ (652C4) Monoclonal Antibody	CAC	PRPG-LA4-M01
Anti Collagen 12 (378D5) Monoclonal Antibody	CAC	PRPG-CO12-M01

* < BACKGROUND : Versican [CSPG2] >

Versican, originally 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 that of brain. It was first isolated from the bovine aorta by Dick Heinegard's and Anders Malmstrom's groups (1982) and shortly after isolated from the chick embryonic limbs by Koji Kimata's group (1986). 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 generated by duplication of the gene.

Versican splicing forms are generated through differential utilization of the central core protein exons 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 case of a GAG-free proteoglycan. The splicing pattern of the *VCAN/CSPG2* gene implies that core proteins of the different *versican* isoforms have a molecular mass range of 50-550 kDa. When taking 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. To note, because of its complex glycosylation/glycanation pattern and the complex molecular interactions that *versican* engages and it is extremely to purify native versican from mammalian and human adult tissues to homogeneity. The C-terminal (G3) globular domain of *versican* consists of one or two EGF repeats, a C-type lectin module and a 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 connective tissue ECMs.

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 of murine embryos harboring the *VCAN/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, while the transcript emerges as one of the most modulated with respect to healthy tissue. *Versican* is a primary component of the intralesional stroma where it may also contribute to progression of the tumour.

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