**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% NaN3  
**Concentration**  | ND  
**Volume**  | 2 mL  
**Label**  | Unlabeled  
**Specificity**  | Versican V0, V1 and V2 isoforms (V3 isoform recognition not ascertained)  
**Cross reactivity**  | Human, Bovine  
**Storage**  | Store at 4°C for short-term storage and -20°C for prolonged storage  
**Other**  | Aliquot to avoid cycles of freeze / thaw.  

**Application notes**  
**Recommended dilutions**  
- Western blotting, 1/20 - 1/40  
- Immunohistochemistry, 1/25 - 1/50  
- ELISA, 1/50 - 1/150  

*<Staining Pattern>*  
Antibody 4C5 stains ubiquitously connective tissue ECMs and detects versican glycosylation isoforms 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**

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).
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 intraliesional stroma were it may also contribute to progression of the tumour.

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