

DESCRIPTION

Species Reactivity	Human
Specificity	Detects human NOV/CCN3 in ELISAs and Western blots. In sandwich immunoassays, approximately 7% cross-reactivity with recombinant mouse NOV is observed.
Source	Polyclonal Goat IgG
Purification	Antigen Affinity-purified
Immunogen	Mouse myeloma cell line NS0-derived recombinant human NOV/CCN3 Thr32-Met357 Accession # P48745
Formulation	Lyophilized from a 0.2 µm filtered solution in PBS with BSA as a carrier protein. See Certificate of Analysis for details.

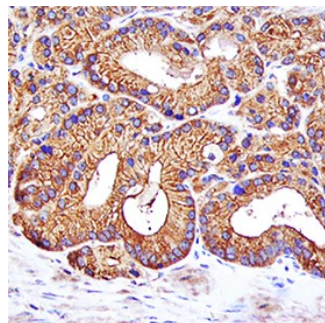
APPLICATIONS

Please Note: Optimal dilutions should be determined by each laboratory for each application. *General Protocols* are available in the *Technical Information* section on our website.

	Recommended Concentration	Sample
Western Blot	0.1 µg/mL	Recombinant Human NOV/CCN3 (Catalog # 1640-NV)
Immunohistochemistry	5-15 µg/mL	See Below
Human NOV/CCN3 Sandwich Immunoassay		Reagent
ELISA Capture	0.2-0.8 µg/mL	Human NOV/CCN3 Antibody (Catalog # AF1640)
ELISA Detection	0.1-0.4 µg/mL	Human NOV/CCN3 Biotinylated Antibody (Catalog # BAF1640)
Standard		Recombinant Human NOV/CCN3 (Catalog # 1640-NV)

DATA

Immunohistochemistry



NOV/CCN3 in Human Prostate.

NOV/CCN3 was detected in immersion fixed paraffin-embedded sections of human prostate using Goat Anti-Human NOV/CCN3 Biotinylated Antigen Affinity-purified Polyclonal Antibody (Catalog # BAF1640) at 15 µg/mL overnight at 4 °C. Tissue was stained using the Anti-Goat HRP-DAB Cell & Tissue Staining Kit (brown; Catalog # CTS008) and counterstained with hematoxylin (blue). Specific staining was localized to cytoplasm and plasma membrane. View our protocol for [Chromogenic IHC Staining of Paraffin-embedded Tissue Sections](#).

PREPARATION AND STORAGE

Reconstitution	Reconstitute at 0.2 mg/mL in sterile PBS.
Shipping	The product is shipped at ambient temperature. Upon receipt, store it immediately at the temperature recommended below.
Stability & Storage	<p>Use a manual defrost freezer and avoid repeated freeze-thaw cycles.</p> <ul style="list-style-type: none"> ● 12 months from date of receipt, -20 to -70 °C as supplied. ● 1 month, 2 to 8 °C under sterile conditions after reconstitution. ● 6 months, -20 to -70 °C under sterile conditions after reconstitution.

BACKGROUND

NOV, also called CCN3, is one of six CCN (CYR61/CTGF/NOV) secreted proteins which share a common multimodular organization (1-4). NOV/CCN3 contains an N-terminal IGF1R domain that appears to be non-functional and a vWF type C and thrombospondin type I domain which mediate oligomerization and matrix interactions, respectively (1, 2). The C-terminal cysteine knot domain interacts with several partners, including the matrix protein fibulin 1C (5), Notch-1 (6), and CCN2, with which it may heterodimerize (2). NOV/CCN3 also interacts with the gap junction protein Connexin43 and mediates suppression of proliferation (7). It also binds the calcium binding protein S100A4 and promotes calcium channel activation (8). The 357 amino acid (aa), 44 kDa human NOV/CCN3 shares 80% aa identity with mouse, rat and dog NOV/CCN3, and 78% aa identity with cow NOV/CCN3. NOV/CCN3 also shows 38-50% aa identity with other family members including WISP proteins, except for WISP-2/CCN5 which lacks the cysteine knot (1). NOV/CCN3 is widely expressed developmentally, especially in muscle, endothelium, nervous system, adrenal cortex and chondrocytes (1-4). In transformed cells, a 32 kDa N-terminally truncated form lacks the signal sequence is localized to the nucleus. Truncation allows a C-terminal nuclear localization sequence to be active (9). Nuclear NOV/CCN3 acts as a transcriptional repressor but promotes proliferation, presumably by interfering with growth control (9). Full length NOV/CCN3 is a secreted extracellular matrix protein which inhibits cell growth. Interaction of NOV/CCN3 with integrins $\alpha_5\beta_1$ and $\alpha_5\beta_3$ mediates endothelial cell adhesion, chemotaxis, and promotes angiogenesis (10, 11). Over-expression of NOV/CCN3 downregulates myogenic genes such as MyoD (12).

References:

1. Perbal, B. (2004) *Lancet* **363**:62.
2. Perbal, B. (2006) *Cell Commun. Signal.* **4**:3.
3. Martinerie, C. *et al.* (1994) *Oncogene* **9**:2729.
4. Snaith, M.R. *et al.* (1996) *Genomics* **38**:425.
5. Perbal, B. *et al.* (1999) *Proc. Natl. Acad. Sci. USA* **96**:869.
6. Sakamoto, K. *et al.* (2002) *J. Biol. Chem.* **277**:29399.
7. Fu, C.T. *et al.* (2004) *J. Biol. Chem.* **279**:36943.
8. Li, C.L. *et al.* (2002) *Mol. Pathol.* **55**:250.
9. Planque, N. *et al.* (2006) *J. Cell. Biochem.* **99**:105.
10. Lin, C.G. *et al.* (2003) *J. Biol. Chem.* **278**:24200.
11. Lin, C.G. *et al.* (2003) *J. Biol. Chem.* **280**:8229.
12. Calhabeu, F. *et al.* (2006) *Exp. Cell. Res.* **312**:1876.