

CC10 (T-18): sc-9772



The Power to Question

BACKGROUND

Clara cell 10 (CC10) protein, a homologue of rabbit uteroglobin, is a phospholipase A2 inhibitor. CC10 is regulated by AP-1, octamer, and hepatocyte nuclear factor-3 (HNF-3) family transcription factors. CC10 expression changes in relation to the ovarian menstrual cycle, and expression in human endometrium may be stimulated by progesterone, suggesting that CC10 may regulate eicosanoid levels in the human uterus. CC10 is expressed in nonciliated airway epithelial cells in the lung and in urogenital secretions. CC10 is involved in modulating inflammation in airway passages and may play a role in asthma. Overexpression of CC10 in the non-small cell lung cancer cell line A549 was shown to result in the near absence of MMP-2 and MMP-9 matrix metalloproteinases and a reduction in invasiveness, indicating that loss of CC10 may contribute to carcinogenesis.

CHROMOSOMAL LOCATION

Genetic locus: SCGB1A1 (human) mapping to 11q12.3; Scgb1a1 (mouse) mapping to 19 A.

SOURCE

CC10 (T-18) is an affinity purified goat polyclonal antibody raised against a peptide mapping near the C-terminus of CC10 of mouse origin.

PRODUCT

Each vial contains 200 µg IgG in 1.0 ml of PBS with < 0.1% sodium azide and 0.1% gelatin.

Blocking peptide available for competition studies, sc-9772 P, (100 µg peptide in 0.5 ml PBS containing < 0.1% sodium azide and 0.2% BSA).

APPLICATIONS

CC10 (T-18) is recommended for detection of CC10 of mouse, rat and human origin by Western Blotting (starting dilution 1:200, dilution range 1:100-1:1000), immunoprecipitation [1-2 µg per 100-500 µg of total protein (1 ml of cell lysate)], immunofluorescence (starting dilution 1:50, dilution range 1:50-1:500), immunohistochemistry (including paraffin-embedded sections) (starting dilution 1:50, dilution range 1:50-1:500) and solid phase ELISA (starting dilution 1:30, dilution range 1:30-1:3000).

Suitable for use as control antibody for CC10 siRNA (h): sc-29954, CC10 siRNA (m): sc-29955, CC10 shRNA Plasmid (h): sc-29954-SH, CC10 shRNA Plasmid (m): sc-29955-SH, CC10 shRNA (h) Lentiviral Particles: sc-29954-V and CC10 shRNA (m) Lentiviral Particles: sc-29955-V.

Molecular Weight of CC10: 10 kDa.

Positive Controls: WI-38 whole cell lysate: sc-364260.

RESEARCH USE

For research use only, not for use in diagnostic procedures.

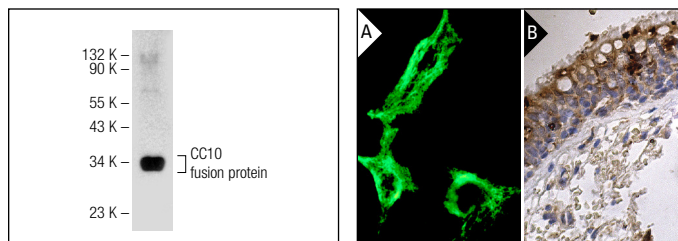
PROTOCOLS

See our web site at www.scbt.com or our catalog for detailed protocols and support products.

STORAGE

Store at 4° C, ****DO NOT FREEZE****. Stable for one year from the date of shipment. Non-hazardous. No MSDS required.

DATA



CC10 (T-18): sc-9772. Western blot analysis of human recombinant CC10 fusion protein.

CC10 (T-18): sc-9772. Immunofluorescence staining of methanol-fixed A549 cells showing cytoplasmic localization (A). Immunoperoxidase staining of formalin fixed, paraffin-embedded human bronchus tissue showing cytoplasmic staining of respiratory epithelial cells (B).

SELECT PRODUCT CITATIONS

- Bonner, J.C., et al. 2002. Susceptibility of cyclooxygenase-2-deficient mice to pulmonary fibrogenesis. *Am. J. Pathol.* 161: 459-470.
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- Uchimura, T., et al. 2009. Bmp2 and Bmp4 genetically interact to support multiple aspects of mouse development including functional heart development. *Genesis* 47: 374-384.
- Perl, A.K., et al. 2010. Conditional depletion of airway progenitor cells induces peribronchiolar fibrosis. *Am. J. Respir. Crit. Care Med.* 183: 511-521.
- O'Brien, K.B., et al. 2010. CARM1 is required for proper control of proliferation and differentiation of pulmonary epithelial cells. *Development* 137: 2147-2156.
- Pacheco-Pinedo, E.C., et al. 2011. Wnt/ β -catenin signaling accelerates mouse lung tumorigenesis by imposing an embryonic distal progenitor phenotype on lung epithelium. *J. Clin. Invest.* 121: 1935-1945.
- Volckaert, T., et al. 2011. Parabronchial smooth muscle constitutes an airway epithelial stem cell niche in the mouse lung after injury. *J. Clin. Invest.* 121: 4409-4419.
- Shannahan, J.H., et al. 2012. Subchronic pulmonary pathology, iron overload, and transcriptional activity after Libby amphibole exposure in rat models of cardiovascular disease. *Environ. Health Perspect.* 120: 85-91.

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