SANTA CRUZ BIOTECHNOLOGY, INC.

SP-C (M-20): sc-7706

BACKGROUND

Pulmonary surfactant is primarily responsible for lowering the surface tension at the air-liquid interface in the alveoli, a process that is essential for normal respiration. Pulmonary surfactant is a mixture of phospholipids and proteins, including four distinct surfactant-associated proteins (SPs), SP-A, SP-B, SP-C, SP-D. SP-B and SP-C are predominantly hydrophobic proteins that associate with lipids to promote the absorption of surfactant phospholipids and to reduce the surface tension in the alveoli. SP-A and SP-D are large multimeric proteins belonging to the family of calcium-dependent lectins, designated collectins, which contribute to the innate immune system. Both SP-A and SP-D have been shown to protect against microbial challenge through binding to the lipid components of the bacterial cell wall and facilitating the rapid removal of microbials.

REFERENCES

1. Glasser, S.W., et al. 1990. Structure and expression of the pulmonary surfactant protein SP-C gene in the mouse. J. Biol. Chem. 265: 21986-21991.
2. Hawgood, S., et al. 1991. Structures and properties of the surfactant-associated proteins. Annu. Rev. Physiol. 53: 375-394.

CHROMOSOMAL LOCATION

Genetic locus: SFTPC (mouse) mapping to 14 D2.

SOURCE

SP-C (M-20) is an affinity purified goat polyclonal antibody raised against a peptide mapping near the C-terminus of SP-C 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-7706 P, (100 µg peptide in 0.5 ml PBS containing < 0.1% sodium azide and 0.2% BSA).

APPLICATIONS

SP-C (M-20) is recommended for detection of SP-C precursor of mouse, rat and mink 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) and solid phase ELISA (starting dilution 1:30, dilution range 1:30-1:3000); non cross-reactive with mature SP-C.

Suitable for use as control antibody for SP-C siRNA (m): sc-36540, SP-C shRNA Plasmid (m): sc-36540-SH and SP-C shRNA (m) Lentiviral Particles: sc-36540-V.

Molecular Weight of SP-C precursor: 21 kDa.

Molecular Weight of mature SP-C: 4-11 kDa.

Positive Controls: rat lung extract: sc-2396 or mouse lung extract: sc-2390.

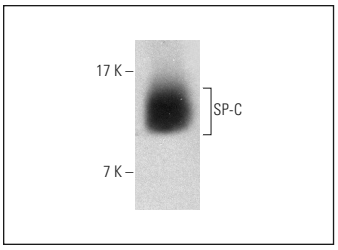
RESEARCH USE

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

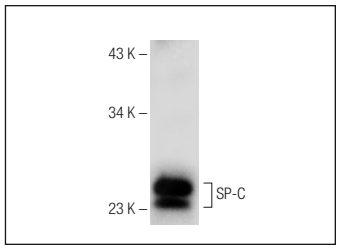
STORAGE

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

DATA



SP-C (M-20): sc-7706. Western blot analysis of SP-C expression in rat lung tissue extract.



SP-C (M-20)-R: sc-7706-R. Western blot analysis of SP-C expression in P 23 whole cell lysate.

SELECT PRODUCT CITATIONS

1. Lin, Y., et al. 2001. Induced repatterning of type XVIII collagen expression in ureter bud from kidney to lung type: association with sonic hedgehog and ectopic surfactant protein C. Development 128: 1573-1585.
2. Ramirez, M.I., et al. 2003. T1α, a lung type I cell differentiation gene, is required for normal lung cell proliferation and alveolus formation at birth. Dev. Biol. 256: 61-72.
3. Lesur, O., et al. 2003. Role of IFN-γ and IL-2 in rat lung epithelial cell migration and apoptosis after oxidant injury. Am. J. Physiol. Lung Cell. Mol. Physiol. 286: L4-L14.
4. Berg, T., et al. 2006. Ectopic expression of C/EBPα in the lung epithelium disrupts late lung development. Am. J. Physiol. Lung Cell. Mol. Physiol. 291: L683-L693.
5. Tang, J.R., et al. 2007. Early inhaled nitric oxide treatment decreases apoptosis of endothelial cells in neonatal rat lungs after vascular endothelial growth factor inhibition. Am. J. Physiol. Lung Cell. Mol. Physiol. 293: L1271-L1280.
6. Jay, P.Y., et al. 2007. Impaired mesenchymal cell function in Gata4 mutant mice leads to diaphragmatic hernias and primary lung defects. Dev. Biol. 301: 602-614.
7. Danielian, P.S., et al. 2007. E2f4 is required for normal development of the airway epithelium. Dev. Biol. 305: 564-576.
8. Didon, L., et al. 2010. Lung-specific inactivation of CCAAT/enhancer binding protein α causes a pathological pattern characteristic of COPD. Eur. Respir. J. 35: 186-197.
9. Kishimoto, K., et al. 2011. Indispensable role of factor for adipocyte differentiation 104 (fad104) in lung maturation. Exp. Cell Res. 317: 2110-2123.
10. Sun, F., et al. 2012. Differential expression of coxsackievirus and adenovirus receptor on alveolar epithelial cells between fetal and adult mice determines their different susceptibility to coxsackievirus B infection. Arch. Virol. 157: 1101-1111.