SANTA CRUZ BIOTECHNOLOGY, INC.

Pneumolysin (1F11): sc-80500



BACKGROUND

Pneumolysin, also known as PLY or *Streptococcus pneumoniae* D39, is a pneumococcal intracellular toxin. It is an important virulence factor of pneumococcus and has proinflammatory and cytotoxic activities. Pneumolysin activates the classical complement pathway and stimulates the production of cytokines by monocytes and macrophages. It can affect polymorphonuclear cell activity such as chemotaxis, degranulation and bactericidal activity. At toxic levels, Pneumolysin binds to cholesterol-containing cell membranes and induces the formation of ring-shaped pores resulting in cell death. Pneumolysin promotes extra-pulmonary dissemination of the pneumococcus and is the major cause of bacterial meningitis. It permanently damages cochlear hair cells and leads to subsequent loss of hearing. An immune system response is activated by the recognition of Pneumolysin by the Toll-like receptor, TLR4. The production of Pneumolysin can be inhibited by erythromycin, clindamycin and rifampicin.

REFERENCES

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- Virolainen, A., et al. 1996. Comparison of serum antibodies to Pneumolysin with those to pneumococcal capsular polysaccharides in children with acute otitis media. Pediatr. Infect. Dis. J. 15: 128-133.
- Anderson, R., et al. 2007. Comparison of the effects of macrolides, amoxicillin, ceftriaxone, doxycycline, tobramycin and fluoroquinolones, on the production of Pneumolysin by *Streptococcus pneumoniae in vitro*. J. Antimicrob. Chemother. 60: 1155-1158.
- Spreer, A., et al. 2007. Influence of subinhibitory concentrations of protein-synthesis-inhibiting antibiotics on production and release of the pneumococcal virulence factor Pneumolysin *in vitro*. Chemotherapy 53: 327-331.
- Braun, J.S., et al. 2007. Pneumolysin causes neuronal cell death through mitochondrial damage. Infect. Immun. 75: 4245-4254.
- Goos, M., et al. 2007. Expression of a Cu,Zn superoxide dismutase typical for familial amyotrophic lateral sclerosis increases the vulnerability of neuroblastoma cells to infectious injury. BMC Infect. Dis. 7: 131.
- Propst-Graham, K.L., et al. 2007. Cirrhosis-induced defects in innate pulmonary defenses against *Streptococcus pneumoniae*. BMC Microbiol. 7: 94.
- Dessing, M.C., et al. 2008. Toll-like receptor 2 contributes to antibacterial defence against Pneumolysin-deficient pneumococci. Cell. Microbiol. 10: 237-246.
- Franco-Vidal, V., et al. 2008. Zinc protection against Pneumolysin toxicity on rat cochlear hair cells. Audiol. Neurootol. 13: 65-70.

SOURCE

Pneumolysin (1F11) is a mouse monoclonal antibody raised against Pneumolysin of *Streptococcus pneumoniae* origin.

PRODUCT

Each vial contains 100 $\mu g~lg G_1$ in 1.0 ml of PBS with < 0.1% sodium azide and 0.1% gelatin.

APPLICATIONS

Pneumolysin (1F11) is recommended for detection of Pneumolysin of *Streptococcus pneumoniae* origin by Western Blotting (starting dilution 1:200, dilution range 1:100-1:1000).

Molecular Weight of Pneumolysin: 53 kDa.

SELECT PRODUCT CITATIONS

- Karthikeyan, R.S., et al. 2013. Host response and bacterial virulence factor expression in *Pseudomonas aeruginosa* and *Streptococcus pneumoniae* corneal ulcers. PLoS ONE 8: e64867.
- Shak, J.R., et al. 2013. Novel role for the *Streptococcus pneumoniae* toxin Pneumolysin in the assembly of biofilms. mBio 4: e00655-13.
- Zhu, L., et al. 2015. Deletion analysis of *Streptococcus pneumoniae* late competence genes distinguishes virulence determinants that are dependent or independent of competence induction. Mol. Microbiol. 97: 151-165.
- 4. Rashwan, R., et al. 2018. *Streptococcus pneumoniae* potently induces cell death in mesothelial cells. PLoS ONE 13: e0201530.
- Surve, M.V., et al. 2018. Heterogeneity in Pneumolysin expression governs the fate of *Streptococcus pneumoniae* during blood-brain barrier trafficking. PLoS Pathog. 14: e1007168.
- Larpin, Y., et al. 2020. Bacterial pore-forming toxin Pneumolysin: cell membrane structure and microvesicle shedding capacity determines differential survival of cell types. FASEB J. 34: 1665-1678.
- Badgujar, D.C., et al. 2020. Structural insights into loss of function of a pore forming toxin and its role in pneumococcal adaptation to an intracellular lifestyle. PLoS Pathog. 16: e1009016.
- Lella, M., et al. 2022. Attenuating the *Streptococcus pneumoniae* competence regulon using urea-bridged cyclic dominant-negative competencestimulating peptide analogs. J. Med. Chem. 65: 6826-6839.

STORAGE

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

RESEARCH USE

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

PROTOCOLS

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