Data Sheet (Cat.No.TMAY-01542)

TargetMol

Anti-Human respiratory syncytial virus (RSV) Fusion Glycoprotein/RSV-F Antibody (1A462)

Product Details

Ig Type:	Rabbit IgG
Conjugation:	Unconjugated
Clone:	1A462
Purification:	Protein A

Applications

Application:	ELISA,LFA(Cap),LFA(Det)
Recommended	ELISA: 1:5000-1:10000; ELISA(Cap): 1 mg/mL; LFA(Det): 1-2 mg/mL; ELISA(Det): 1 mg/mL; LFA (Det): 1-2 mg/mL

Properties

Stability & Storage:	Store at 2°C-8°C for 1 month. Store at -20°C or -80°C for 12 months. Avoid repeated freeze-thaw cycles. Preservative-Free.
Shipping:	Shipping with blue ice.

Antigen Details

Immunogen:	Recombinant Protein: Human RSV Fusion Glycoprotein / RSV-F (TMPY-01078)
Antigen Species:	RSV

Research Background

Human respiratory syncytial virus (HRSV) is the most common etiological agent of acute lower respiratory tract disease in infants and can cause repeated infections throughout life. It is classified within the genus pneumovirus of the family paramyxoviridae. Like other members of the family, HRSV has two major surface glycoproteins (G and F) that play important roles in the initial stages of the infectious cycle. The G protein mediates attachment of the virus to cell surface receptors, while the F protein promotes fusion of the viral and cellular membranes, allowing entry of the virus ribonucleoprotein into the cell cytoplasm. The fusion (F) protein of RSV is synthesized as a nonfusogenic precursor protein (F), which during its migration to the cell surface is activated by cleavage into the disulfide-linked F1 and F2 subunits. This fusion is pH independent and occurs directly at the outer cell membrane, and the F2 subunit was identifed as the major determinant of RSV host cell specificity. The trimer of F1-F2 interacts with glycoprotein G at the virion surface. Upon binding of G to heparan sulfate, the hydrophobic fusion peptide is unmasked and induces the fusion between host cell and virion membranes. Notably, RSV fusion protein is unique in that it is able to interact directly with heparan sulfate and therefore is sufficient for virus infection. Furthermore, the fusion protein is also able to trigger p53-dependent apoptosis.

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