Anti-Human respiratory syncytial virus(RSV) Fusion glycoprotein/RSV-F Neutralizing Antibody

catalog number: E-AB-V1272

Note: *Centrifuge before opening to ensure complete recovery of vial contents.*

Description

Reactivity	RSV
Immunogen	Recombinant RSV(A2) Fusion glycoprotein / RSV-F Protein (His Tag)
Host	Rabbit
Isotype	IgG
Clone	R338
Purification	Protein A Affinity
Buffer	0.2 μm filtered solution in PBS.

Applications	Recommended Dilution
ELISA	1:1000-1:10000

Preparation & Storage

Storage	Store at -20°C Valid for 12 months. Avoid freeze / thaw cycles.
Shipping	The product is shipped with ice pack,upon receipt,store it immediately at the temperature recommended.

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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