

Mouse Anti-Respiratory Syncytial Virus (RSV) F Glycoprotein [5A6]: MC0204

Intended Use: For Research Use Only

Description: Respiratory Syncytial Virus (RSV) Fusion (F) Glycoprotein is a Class I viral fusion protein. The Glycoprotein has at least 3 conformational states: pre-fusion native state, pre-hairpin intermediate state, and post-fusion hairpin state. During viral and target cell membrane fusion, the heptad repeat regions assume a trimer-of-hairpins structure, positioning the fusion peptide in close proximity to the C-terminal region of the ectodomain. The formation of this structure appears to drive apposition and subsequent fusion of viral and target cell membranes, directs fusion of viral and cellular membranes leading to delivery of the nucleocapsid into the cytoplasm. This fusion is pH independent and occurs directly at the outer cell membrane. The trimer of F1-F2 (protein F) interacts with glycoprotein G at the virion surface. Upon binding of G to heparan sulfate, the hydrophobic fusion peptide is unmasked and interacts with the cellular membrane, inducing the fusion between host cell and virion membranes. RSV fusion protein is able to interact directly with heparan sulfate and therefore actively participates in virus attachment. Furthermore, the F2 subunit was identified as the major determinant of RSV host cell specificity. Later in infection, proteins F expressed at the plasma membrane of infected cells mediate fusion with adjacent cells to form syncytia, a cytopathic effect that could lead to tissue necrosis. The fusion protein is also able to trigger p53-dependent apoptosis. The antibody is suitable for the detection of RSV protein of Respiratory Syncytial Virus origin. Respiratory Syncytial Virus (RSV) is a major cause of respiratory illness in children who have not received the vaccine or treatment. Respiratory Syncytial Virus is a negative sense, enveloped, RNA virus. The virion has an average diameter between 120 and 300 nm. The fusion protein of the RSS 2 strain (subtype A) directs fusion of viral and cellular membranes, results in viral penetration, and can form syncytia or multi-nucleated giant cells. The matrix protein plays a role in viral assembly and has been observed to traffic into and out of the nucleus at specific times during the respiratory infectious cycle. The matrix protein has also been shown to be able to inhibit transcription, which may be a key to respiratory pathogenesis.

Specifications

Clone: 5A6
 Source: Mouse
 Isotype: IgG2a
 Reactivity: Respiratory Syncytial Virus
 Immunogen: hRSV strain A2 infected HeLa cells
 Localization: Virion membrane, Single-pass type I membrane protein, host cell membrane
 Formulation: Antibody in PBS pH7.4, containing BSA and $\leq 0.09\%$ sodium azide (NaN₃)
 Storage: Store at 2°- 8°C
 Applications: IHC, ELISA, IF, WB
 Package:

Description	Catalog No.	Size
Respiratory Syncytial Virus (RSV) F Glycoprotein Concentrated	MC0204	1 ml

IHC Procedure*

Positive Control Tissue: Human RSV A2 infected HeLa cells
 Concentrated Dilution: 10-100
 Pretreatment: None
 Incubation Time and Temp: Overnight @ 4°C
 Detection: Refer to the detection system manual

* Result should be confirmed by an established diagnostic procedure.

References:

1. The Secretome Profiling of a Pediatric Airway Epithelium Infected with hRSV Identified Aberrant Apical/Basolateral Trafficking and Novel Immune Modulating (CXCL6, CXCL16, CSF3) and Antiviral (CEACAM1) Proteins. Touzelet O, et al. Mol Cell Proteomics 19:793-807, 2020.
2. Suppression of IRG-1 Reduces Inflammatory Cell Infiltration and Lung Injury in Respiratory Syncytial Virus Infection by Reducing Production of Reactive Oxygen Species. Ke Ren, et al. J Virol. Jul 27;90(16):7313-7322, 2016.
3. Baicalin from Scutellaria baicalensis blocks respiratory syncytial virus (RSV) infection and reduces inflammatory cell infiltration and lung injury in mice. Hengfei Shi, et al. Sci Re. Oct 21;6:35851, 2016.