Product name: Anti-Respiratory Syncytial Virus Fusion (F) Glycoprotein antibody [RSV5A6] ab94968

Description: Mouse monoclonal [RSV5A6] to Respiratory Syncytial Virus Fusion (F) Glycoprotein

Host species: Mouse

Tested applications: Suitable for: ELISA, WB, ICC/IF, IHC-P, IHC-Fr

Species reactivity: Reacts with: Other species

Immunogen: Human Respiratory Syncytial Virus strain A2 infected HeLa cells

General notes: Fusion partner: PS-NS/1-Ag4

ab94968 is useful for the identification and location of expression of the fusion glycoprotein of Human Respiratory Syncytial Virus of both sub-groups A and B.

Form: Liquid

Storage instructions: Shipped at 4°C. Upon delivery aliquot and store at -20°C or -80°C. Avoid repeated freeze / thaw cycles.

Storage buffer: Constituent: PBS

Purity: Protein A purified

Primary antibody notes: ab94968 is useful for the identification and location of expression of the fusion glycoprotein of Human Respiratory Syncytial Virus of both sub-groups A and B.

Clonality: Monoclonal

Clone number: RSV5A6

Isotype: IgG2a

Applications:
Our Abpromise guarantee covers the use of ab94968 in the following tested applications.
The application notes include recommended starting dilutions; optimal dilutions/concentrations should be determined by the end user.
Respiratory Syncytial Virus (RSV) Fusion (F) Glycoprotein is a Class I viral fusion protein. Under the current model, the protein 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 (HR) 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. Notably, 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.

**Cellular localization**

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**Target**

**Relevance**
Respiratory Syncytial Virus (RSV) Fusion (F) Glycoprotein is a Class I viral fusion protein. Under the current model, the protein 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 (HR) 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. Notably, 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.

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**Application**

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<th>Application</th>
<th>Abreviews</th>
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<tbody>
<tr>
<td>ELISA</td>
<td>Use at an assay dependent concentration.</td>
<td></td>
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<tr>
<td>WB</td>
<td>Use at an assay dependent concentration. Predicted molecular weight: 63 kDa.</td>
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<tr>
<td>ICC/IF</td>
<td>Use at an assay dependent concentration.</td>
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<tr>
<td>IHC-P</td>
<td>Use at an assay dependent concentration.</td>
<td></td>
</tr>
<tr>
<td>IHC-Fr</td>
<td>Use at an assay dependent concentration. Fix with Acetone.</td>
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</tbody>
</table>

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**Target**

**Relevance**
Respiratory Syncytial Virus (RSV) Fusion (F) Glycoprotein is a Class I viral fusion protein. Under the current model, the protein 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 (HR) 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. Notably, 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.

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