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SARS virus Spike Antibody (C-term V1158)

Purified Rabbit Polyclonal Antibody (Pab)
Catalog # AP6009g

Product Information

Application	E
Primary Accession	P59594
Reactivity	SARS
Host	Rabbit
Clonality	Polyclonal
Isotype	Rabbit IgG
Clone Names	RB3989-3990
Calculated MW	139125
Antigen Region	1150-1179

Additional Information

Other Names	Spike glycoprotein, S glycoprotein, E2, Peplomer protein, Spike protein S1, Spike protein S2, S
Target/Specificity	This SARS virus Spike antibody is generated from rabbits immunized with a KLH conjugated synthetic peptide between 1150~1179 amino acids from the C-terminal region of SARS virus Spike protein.
Dilution	E~~Use at an assay dependent concentration.
Format	Purified polyclonal antibody supplied in PBS with 0.09% (W/V) sodium azide. This antibody is prepared by Saturated Ammonium Sulfate (SAS) precipitation followed by dialysis against PBS.
Storage	Maintain refrigerated at 2-8°C for up to 2 weeks. For long term storage store at -20°C in small aliquots to prevent freeze-thaw cycles.
Precautions	SARS virus Spike Antibody (C-term V1158) is for research use only and not for use in diagnostic or therapeutic procedures.

Protein Information

Name	S {ECO:0000255|HAMAP-Rule:MF_04099}
Function	[Spike glycoprotein]: May down-regulate host tetherin (BST2) by lysosomal degradation, thereby counteracting its antiviral activity.
Cellular Location	Virion membrane {ECO:0000255|HAMAP-Rule:MF_04099, ECO:0000269|PubMed:15831954}; Single-pass type I membrane protein {ECO:0000255|HAMAP-Rule:MF_04099, ECO:0000269|PubMed:15831954}.

Host endoplasmic reticulum-Golgi intermediate compartment membrane {ECO:0000255|HAMAP-Rule:MF_04099, ECO:0000269|PubMed:20861307}; Single- pass type I membrane protein {ECO:0000255|HAMAP-Rule:MF_04099, ECO:0000269|PubMed:15831954}. Host cell membrane {ECO:0000255|HAMAP- Rule:MF_04099, ECO:0000269|PubMed:15831954}; Single-pass type I membrane protein {ECO:0000255|HAMAP-Rule:MF_04099, ECO:0000269|PubMed:15831954}. Note=Accumulates in the endoplasmic reticulum-Golgi intermediate compartment, where it participates in virus particle assembly. Colocalizes with S in the host endoplasmic reticulum-Golgi intermediate compartment (PubMed:20861307). Some S oligomers are transported to the host plasma membrane, where they may mediate cell-cell fusion. {ECO:0000255|HAMAP-Rule:MF_04099, ECO:0000269|PubMed:20861307}

Background

An outbreak of atypical pneumonia, referred to as severe acute respiratory syndrome (SARS) and first identified in Guangdong Province, China, has spread to several countries. The severity of this disease is such that the mortality rate appears to be ~3 to 6%. A number of laboratories worldwidehave undertaken the identification of the causative agent. The National Microbiology Laboratory in Canada obtained the Tor2 isolate from a patient in Toronto, and succeeded in growing a coronavirus-like agent in African Green Monkey Kidney (Vero E6) cells. This coronavirus has been named publicly by the World Health Organization and member laboratories as ?SARS virus? The SARS membrane proteins, including the major proteins S (Spike) and M (Membrane), are inserted into the endoplasmic reticulum Golgi intermediate compartment (ERGIC) while full length replicated RNA (+ strands) assemble with the N (nucleocapsid) protein. The virus then migrates through the Golgi complex and eventually exits the cell, likely by exocytosis. The site of viral attachment to the host cell resides within the S protein. Oligomeric spike (S) glycoproteins extend from SARS membranes. These integral membrane proteins assemble within the endoplasmic reticulum of infected cells and are subsequently endoproteolyzed in the Golgi, generating noncovalently associated S1 and S2 fragments. Once on the surface of infected cells and virions, peripheral S1 fragments bind carcinoembryonic antigen-related cell adhesion molecule (CEACAM) receptors, and this triggers membrane fusion reactions mediated by integral membrane S2 fragments.

References

He, R., et al., Biochem. Biophys. Res. Commun. 316(2):476-483 (2004).
Snijder, E.J., et al., J. Mol. Biol. 331(5):991-1004 (2003).
Marra, M.A., et al., Science 300(5624):1399-1404 (2003).
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