H1N1 Hemagglutinin 2 Antibody FITC Conjugated

Catalog No: #C03864F



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Description	
Product Name	H1N1 Hemagglutinin 2 Antibody FITC Conjugated
Host Species	Rabbit
Clonality	Polyclonal
Isotype	IgG
Purification	Purified by Protein A.
Applications	IF
Species Reactivity	Influenza A Virus H1N1
Crossing Reactivity	Influenza A virus H1N1 (strain swl A California 04 2009 H1N1)
Immunogen Description	KLH conjugated synthetic peptide aa 480-530 566 derived from Influenza A Virus Hemagglutinin (strain swl A
	California 04 2009 H1N1)
Conjugates	FITC
Target Name	H1N1 Hemagglutinin 2
Other Names	HA; HA2; Hemagglutinin; Influenza A Virus (strain swl A California 04 2009 H1N1)
Accession No.	Swiss-Prot#C3W5S1
Uniprot	C3W5S1
Excitation Emission	494nm 518nm
Cell Localization	Cell membrane
Concentration	1mg ml
Formulation	0.01M TBS(pH7.4) with 1% BSA, 0.03% Proclin300 and 50% Glycerol.
Storage	Shipped at 4°C. Store at -20°C for one year. Avoid repeated freeze/thaw cycles.

Application Details

IF=1:50-200

Background

Influenza A virus is a major public health threat. Novel influenza virus strains caused by genetic drift and viral recombination emerge periodically to which humans have little or no immunity, resulting in devastating pandemics. Influenza A can exist in a variety of animals; however it is in birds that all subtypes can be found. These subtypes are classified based on the combination of the virus coat glycoproteins hemagglutinin (HA) and neuraminidase (NA) subtypes. HA interacts with cell surface proteins containing oligosaccharides with terminal sialyl residues. Binds to sialic acid-containing receptors on the cell surface, bringing about the attachment of the virus particle to the cell. This attachment induces virion internalization of about two third of the virus particles through clathrin-dependent endocytosis and about one third through a clathrin- and caveolin-independent pathway. Plays a major role in the determination of host range restriction and virulence. Class I viral fusion protein. Responsible for penetration of the virus into the cell cytoplasm by mediating the fusion of the membrane of the endocytosed virus particle with the endosomal membrane. Low pH in endosomes induces an irreversible conformational change in HA2, releasing the fusion hydrophobic peptide. Several trimers are required to form a competent fusion pore. Influenza A Virus (strain swl A California 04 2009 H1N1)

Note: This product is for in vitro research use only