

**Avian Influenza Neuraminidase Antibody**  
**Catalog # ASC10281****Specification****Avian Influenza Neuraminidase Antibody - Product Information**

Application	E
Primary Accession	<a href="#">Q710U6</a>
Other Accession	<a href="#">CAC95053</a> , <a href="#">39840718</a>
Reactivity	Virus
Host	Rabbit
Clonality	Polyclonal
Isotype	IgG
Application Notes	Avian influenza neuraminidase antibody can be used for the detection of the avian influenza neuraminidase protein from the H5N1 strain of Avian influenza A in ELISA. It will detect 10 ng of free peptide at 1 µg/mL.

**Avian Influenza Neuraminidase Antibody - Additional Information****Other Names**

Avian Influenza Neuraminidase Antibody: Neuraminidase, Neuraminidase

**Target/Specificity**

NA;

**Reconstitution & Storage**

Avian Influenza Neuraminidase antibody can be stored at 4°C for three months and -20°C, stable for up to one year. As with all antibodies care should be taken to avoid repeated freeze thaw cycles. Antibodies should not be exposed to prolonged high temperatures.

**Precautions**

Avian Influenza Neuraminidase Antibody is for research use only and not for use in diagnostic or therapeutic procedures.

**Avian Influenza Neuraminidase Antibody - Protein Information**

**Name** NA {ECO:0000255|HAMAP-Rule:MF\_04071}

**Function**

Catalyzes the removal of terminal sialic acid residues from viral and cellular glycoconjugates. Cleaves off the terminal sialic acids on the glycosylated HA during virus budding to facilitate virus release. Additionally helps virus spread through the circulation by further removing sialic acids from the cell surface. These cleavages prevent self-aggregation and ensure the efficient spread of the progeny virus from cell to cell. Otherwise, infection would be limited to one round of replication. Described as a receptor-destroying enzyme because it cleaves a terminal sialic acid from the cellular receptors. May facilitate viral invasion of the upper airways by cleaving the sialic

acid moieties on the mucin of the airway epithelial cells. Likely to play a role in the budding process through its association with lipid rafts during intracellular transport. May additionally display a raft- association independent effect on budding. Plays a role in the determination of host range restriction on replication and virulence. Sialidase activity in late endosome/lysosome traffic seems to enhance virus replication.

#### **Cellular Location**

Virion membrane {ECO:0000255|HAMAP- Rule:MF\_04071}. Host apical cell membrane {ECO:0000255|HAMAP- Rule:MF\_04071}; Single-pass type II membrane protein {ECO:0000255|HAMAP- Rule:MF\_04071}. Note=Preferentially accumulates at the apical plasma membrane in infected polarized epithelial cells, which is the virus assembly site. Uses lipid rafts for cell surface transport and apical sorting. In the virion, forms a mushroom-shaped spike on the surface of the membrane. {ECO:0000255|HAMAP- Rule:MF\_04071}

### **Avian Influenza Neuraminidase Antibody - Protocols**

Provided below are standard protocols that you may find useful for product applications.

- [Western Blot](#)
- [Blocking Peptides](#)
- [Dot Blot](#)
- [Immunohistochemistry](#)
- [Immunofluorescence](#)
- [Immunoprecipitation](#)
- [Flow Cytometry](#)
- [Cell Culture](#)

### **Avian Influenza Neuraminidase Antibody - Images**

### **Avian Influenza Neuraminidase Antibody - Background**

Avian Influenza Neuraminidase Antibody: Influenza A virus is a major public health threat, killing more than 30,000 people per year in the USA. Novel influenza virus strains 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. During 1997, an H5N1 avian influenza virus was determined to be the cause of death in 6 of 18 infected patients in Hong Kong. There was some evidence of human to human spread of this virus, but it is thought that the transmission efficiency was fairly low. Although it has been known that cleavage site and glycosylation patterns of the HA protein play important roles in determining the pathogenicity of H5 avian influenza viruses, it has only recently been shown that an additional glycosylation site within the globular head of the NA protein also contributes to the high virulence of the H5N1 virus.

### **Avian Influenza Neuraminidase Antibody - References**

Thompson WW, Shay DK, Weintraub, et al. Mortality associated with influenza and respiratory syncytial virus in the United States. JAMA 2003; 289:179-86.

Alexander DJ. A review of avian influenza. Proceedings of the European Society for Veterinary Virology (ESVV) Symposium on Influenza Viruses of Wild and Domestic Animals. Vet. Microbiol. 2000; 74:3-13.

Shortridge KF, Zhou NN, Guan Y, et al. Characterization of avian H5N1 influenza viruses from poultry in Hong Kong. Virol. 1998; 252:331-42.

Buxton Bridges C, Katz JM, Seto WH, et al. Risk of influenza A (H5N1) infection among health care workers exposed to patients with influenza A (H5N1), Hong Kong. J. Inf. Dis. 2000; 181:344-8.