

Product Data Sheet



NA/Neuraminidase Protein, H1N1 (ACP41107, HEK293, Fc)

Cat. No.: HY-P73791

Synonyms: NA; Neuraminidase; NA/Neuraminidase Protein, H1N1 (A/California/04/2009, HEK293, Fc)

Species: HEK293 Source:

Accession: ACP41107 (H36-K469)

Gene ID:

Molecular Weight: 95-105 kDa

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Appearance	Lyophilized powder.
Formulation	Lyophilized from a 0.2 μ m filtered solution of PBS, pH 7.4. Normally 5 % - 8 % trehalose, mannitol and 0.01% Tween 80 are added as protectants before lyophilization.
Endotoxin Level	<1 EU/µg, determined by LAL method.
Reconsititution	It is not recommended to reconstitute to a concentration less than 100 $\mu g/mL$ in ddH ₂ O.
Storage & Stability	Stored at -20°C for 2 years. After reconstitution, it is stable at 4°C for 1 week or -20°C for longer (with carrier protein). It is recommended to freeze aliquots at -20°C or -80°C for extended storage.
Shipping	Room temperature in continental US; may vary elsewhere.

DESCRIPTION

Background

Neuraminidase (NA) plays a pivotal role in viral propagation by catalyzing the removal of terminal sialic acid residues from both viral and cellular glycoconjugates. Specifically, during virus budding, NA cleaves off terminal sialic acids from the glycosylated hemagglutinin (HA), facilitating the release of viral particles and enabling efficient virus spread through the circulation. By preventing self-aggregation and ensuring the removal of sialic acids from the cell surface, NA allows the progeny virus to disseminate efficiently from cell to cell, thereby avoiding limitations to a single round of replication. Described as a receptor-destroying enzyme, NA cleaves terminal sialic acids from cellular receptors, potentially facilitating viral invasion of the upper airways by targeting sialic acid moieties on airway epithelial cell mucin. Its association with lipid rafts during intracellular transport, and its potential raft-independent effect on budding, highlight the multifaceted role of NA in determining host range restriction, replication, and virulence. Moreover, the sialidase activity in late endosome/lysosome traffic appears to enhance virus replication^{[1][2][3]}.

Page 1 of 2 www.MedChemExpress.com $\label{lem:caution:Product} \textbf{Caution: Product has not been fully validated for medical applications. For research use only.}$

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Page 2 of 2 www.MedChemExpress.com