

Rabbit Anti-DENV-2 E antibody

SL0171R

Product Name	DENV-2 E
Chinese Name	登革热病毒包膜 glycoproteinE 抗体
Alias	DENV2; Dengue virus type 2 (strain China/D2-04); Dengue virus 2; DENV-2; Dengue virus type 2 (strain D2-04); Dengue virus type 2 China/D2-04; Dengue virus 2 China/D2-04; POLG_DEN2D; Genome polyprotein; DEN2;
Research Area	Bacteria and viruses
Immunogen Species	Rabbit
Clonality	Polyclonal
React Species	(predicted:Dengue virus type-2) WB=1:500-2000,ELISA=1:5000-10000
Applications	not yet tested in other applications. optimal dilutions/concentrations should be determined by the end user.
Theoretical molecular weight	54kDa
Cellular localization	cytoplasmic The cell membrane Secretory protein
Form	Liquid
Concentration	1mg/ml
immunogen	KLH conjugated synthetic peptide derived from Dengue virus type-2 envelope glycoprotein E: 501-600/3390
Lsotype	IgG
Purification	affinity purified by Protein A
Buffer Solution	(predicted:Dengue virus type-2)1M TBS(pH7.4) with 1% BSA, (predicted:Dengue virus type-2)3% Proclin300 and 50% Glycerol.
Storage	Shipped at 4°C. Store at -20 °C for one year. Avoid repeated freeze/thaw cycles.
Attention	This product as supplied is intended for research use only, not for use in human, therapeutic or diagnostic applications.
PubMed	PubMed
Product Detail	Envelope protein E binding to host cell surface receptor is followed by virus

internalization through clathrin-mediated endocytosis. Envelope protein E is subsequently involved in membrane fusion between virion and host late endosomes. Synthesized as a homodimer with prM which acts as a chaperone for envelope protein E. After cleavage of prM, envelope protein E dissociate from small envelope protein M and homodimerizes.

Function:

prM acts as a chaperone for envelope protein E during intracellular virion assembly by masking and inactivating envelope protein E fusion peptide. prM is matured in the last step of virion assembly, presumably to avoid catastrophic activation of the viral fusion peptide induced by the acidic pH of the trans-Golgi network. After cleavage by host furin, the pr peptide is released in the extracellular medium and small envelope protein M and envelope protein E homodimers are dissociated (By similarity).

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Non-structural protein 1 is involved in virus replication and regulation of the innate immune response. Soluble and membrane-associated NS1 may activate human complement and induce host vascular leakage. This effect might explain the clinical manifestations of dengue hemorrhagic fever and dengue shock syndrome (By similarity).

Non-structural protein 2A may be involved viral RNA replication and capsid assembly (Potential).

Non-structural protein 2B is a required cofactor for the serine protease function of NS3 (By similarity).

Serine protease NS3 displays three enzymatic activities: serine protease, NTPase and RNA helicase. NS3 serine protease, in association with NS2B, performs its autocleavage and cleaves the polyprotein at dibasic sites in the cytoplasm: C-prM, NS2A-NS2B, NS2B-NS3, NS3-NS4A, NS4A-2K and NS4B-NS5. NS3 RNA helicase binds RNA and unwinds dsRNA in the 3' to 5' direction (By similarity).

Non-structural protein 4A induces host endoplasmic reticulum membrane rearrangements leading to the formation of virus-induced membranous vesicles hosting the dsRNA and polymerase, functioning as a replication complex. NS4A might also regulate the ATPase activity of the NS3 helicase (By similarity).

Peptide 2k functions as a signal peptide for NS4B and is required for the interferon antagonism activity of the latter (By similarity).

Non-structural protein 4B inhibits interferon (IFN)-induced host STAT1 phosphorylation and nuclear translocation, thereby preventing the

establishment of cellular antiviral state by blocking the IFN-alpha/beta pathway (By similarity).

RNA-directed RNA polymerase NS5 replicates the viral (+) and (-) genome, and performs the capping of genomes in the cytoplasm. NS5 methylates viral RNA cap at guanine N-7 and ribose 2'-O positions. Besides its role in genome replication, also prevents the establishment of cellular antiviral state by blocking the interferon-alpha/beta (IFN-alpha/beta) signaling pathway. Inhibits host TYK2 and STAT2 phosphorylation, thereby preventing activation of JAK-STAT signaling pathway.

Subcellular Location:

Capsid protein C: Virion (Potential).

Peptide pr: Secreted.

Small envelope protein M: Virion membrane; Multi-pass membrane protein. Host endoplasmic reticulum membrane; Multi-pass membrane protein.

Envelope protein E: Virion membrane; Multi-pass membrane protein. Host endoplasmic reticulum membrane; Multi-pass membrane protein.

Non-structural protein 1: Secreted. Host endoplasmic reticulum membrane; Peripheral membrane protein; Luminal side.

Non-structural protein 2A-alpha: Host endoplasmic reticulum membrane; Multi-pass membrane protein (Potential).

Non-structural protein 2A: Host endoplasmic reticulum membrane; Multi-pass membrane protein (Potential).

Serine protease subunit NS2B: Host endoplasmic reticulum membrane; Peripheral membrane protein; Cytoplasmic side.

Serine protease NS3: Host endoplasmic reticulum membrane; Peripheral membrane protein; Cytoplasmic side (By similarity). Note=Remains non-covalently associated to NS3 protease (By similarity).

Non-structural protein 4A: Host endoplasmic reticulum membrane; Multi-pass membrane protein (By similarity). Note=Located in RE-associated vesicles hosting the replication complex.

Non-structural protein 4B: Host endoplasmic reticulum membrane; Multi-pass membrane protein (By similarity).

RNA-directed RNA polymerase NS5: Host endoplasmic reticulum membrane; Peripheral membrane protein; Cytoplasmic side. Host nucleus. Note=Located in RE-associated vesicles hosting the replication complex.

Post-translational modifications:

Specific enzymatic cleavages in vivo yield mature proteins. The nascent protein C contains a C-terminal hydrophobic domain that act as a signal sequence for translocation of prM into the lumen of the ER. Mature protein C is cleaved at a site upstream of this hydrophobic domain by NS3. prM is cleaved in post-Golgi vesicles by a host furin, releasing the mature small envelope protein M, and peptide pr. Non-structural protein 2A-alpha, a

C-terminally truncated form of non-structural protein 2A, results from partial cleavage by NS3. Peptide 2K acts as a signal sequence and is removed from the N-terminus of NS4B by the host signal peptidase in the ER lumen. Signal cleavage at the 2K-4B site requires a prior NS3 protease-mediated cleavage at the 4A-2K site.

RNA-directed RNA polymerase NS5 is phosphorylated on serines residues. This phosphorylation may trigger NS5 nuclear localization.

Envelope protein E and non-structural protein 1 are N-glycosylated.

Similarity:

In the N-terminal section; belongs to the class I-like SAM-binding methyltransferase superfamily. mRNA cap 0-1 NS5-type methyltransferase family.

Contains 1 helicase ATP-binding domain.

Contains 1 helicase C-terminal domain.

Contains 1 mRNA cap 0-1 NS5-type MT domain.

Contains 1 peptidase S7 domain.

Contains 1 RdRp catalytic domain.

SWISS:

P30026

Gene ID:

1494449

Database links:

[SwissProt: P29990](#) Dengue virus type 2

[SwissProt: P30026](#) Dengue virus type 2

登革病毒(Dengue Virus, DEN)属黄病毒科(flaviviridae)又称汉坦病毒、黄病毒, 是重要的虫媒病毒之一, 是引起流行性出血热的主要原因。