SunLong Biotech Co.,LTD
Tel: 0086-571- 56623320 Fax:0086-571- 56623318
E-mail:sales@sunlongbiotech.com
www.sunlongbiotech.com

Rabbit Anti-Dengue virus envelope glycoprotein E/FITC Conjugated antibody

SL0171R-FITC

Product Name:	Anti-Dengue virus envelope glycoprotein E/FITC
Chinese Name:	FITC标记的登革热病毒包膜glycoproteinE抗体
Alias:	Dengue virus; DV1_gp1; Genome polyprotein; envelope glycoprotein (Dengue virus type-2); Dengue virus envelope glycoprotein E; Polyprotein; DEN polyprotein.
Organism Species:	Rabbit
Clonality:	Polyclonal
React Species:	Dengue virus type-2
Applications:	IF=1:50-200 not yet tested in other applications. optimal dilutions/concentrations should be determined by the end user.
Molecular weight:	54kDa
Cellular localization:	The cell membrane
Form:	Lyophilized or Liquid
Concentration:	1mg/ml
immunogen:	KLH conjugated synthetic peptide derived from Dengue virus type-2 envelope glycoprotein E
Lsotype:	IgG
Purification:	affinity purified by Protein A
Storage Buffer:	0.01M TBS(pH7.4) with 1% BSA, 0.03% Proclin300 and 50% Glycerol.
Storage:	Store at -20 °C for one year. Avoid repeated freeze/thaw cycles. The lyophilized antibody is stable at room temperature for at least one month and for greater than a year when kept at -20°C. When reconstituted in sterile pH 7.4 0.01M PBS or diluent of antibody the antibody is stable for at least two weeks at 2-4 °C.
Product Detail:	**background:** 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). 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 (By similarity). 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; Lumenal 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. **Database links:** Entrez Gene: 1494449 Dengue virus 2 **Important Note:** This product as supplied is intended for research use only, not for use in human, therapeutic or diagnostic applications. 登革病毒(Dengue Virus，DEN)属黄病毒科(flavlviridae)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。