



Rabbit Anti-EV71 polyprotein VP4 antibody

SL0852R

Product Name:	EV71 polyprotein VP4
Chinese Name:	肠道病毒71型/手足口病病毒抗体
Alias:	EV71; Enterovirus 71; VP4 [Human enterovirus 71]; polyprotein; VP4 capsid protein; capsid protein; polyprotein VP0.
Organism Species:	Rabbit
Clonality:	Polyclonal
React Species:	EV71
Applications:	ELISA=1:500-1000IHC-P=1:400-800IHC-F=1:400-800ICC=1:100-500IF=1:100-500 (Paraffin sections need antigen repair) not yet tested in other applications. optimal dilutions/concentrations should be determined by the end user.
Molecular weight:	7.6kDa
Form:	Lyophilized or Liquid
Concentration:	1mg/ml
immunogen:	KLH conjugated synthetic peptide derived from EV71 Polyprotein VP4:1-69/2193
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.
PubMed:	PubMed
Product Detail:	Enteroviruses, such as enterovirus 71, are classified to be in the picornavirus family, pico [small] + RNA [ribonucleic acid] + virus. Picornaviruses are among the smallest and simplest ribonucleic acid containing viruses known (1). The RNA for many enteroviruses have now been cloned and complete genomic sequences have been obtained. The RNA from all sequenced enteroviruses are similar in length, about 7400 nucleotides, and have identical organization (1). The human alimentary tract is the predominant site of enterovirus replication and these viruses were first isolated from

enteric specimens. These viruses are the cause of paralytic poliomyelitis, aseptic meningitis-encephalitis, myocarditis, pleurodynia, hand-foot-and-mouth disease, conjunctivitis, and numerous other syndromes associated with extra-intestinal target organs. There are 67 numbered types of enteroviruses in the enterovirus family (1): three polioviruses, twenty-three coxsackieviruses A, six coxsackieviruses B, thirty-one echoviruses, and four other enteroviruses.

Function:

Protein VP1: Forms, together with VP2 and VP3, an icosahedral capsid (pseudo T=3), 300 Angstroms in diameter, composed of 60 copies of each capsid protein and enclosing the viral positive strand RNA genome. Protein VP1 mainly forms the vertices of the capsid. VP1 interacts with host cell receptor to provide virion attachment to target cell. After binding to its receptor, the capsid undergoes conformational changes. VP1 N-terminus (that contains an amphipathic alpha-helix) is externalized, VP4 is released and together, they shape a virion-cell connecting channel and a pore in the host membrane through which RNase-protected transfer of the viral genome takes place. After genome has been released, the channel shrinks.

Protein VP2: Forms, together with VP1 and VP3, an icosahedral capsid (pseudo T=3), 300 Angstroms in diameter, composed of 60 copies of each capsid protein and

Protein VP3: Forms, together with VP1 and VP2, an icosahedral capsid (pseudo T=3), 300 Angstroms in diameter, composed of 60 copies of each capsid protein and enclosing the viral positive strand RNA genome.

Protein VP4: Lies on the inner surface of the capsid shell. After binding to the host receptor, the capsid undergoes conformational changes. VP4 is released, VP1 N-terminus is externalized, and together, they shape a virion-cell connecting channel and a pore in the host membrane through which RNase-protected transfer of the viral genome takes place. After genome has been released, the channel shrinks.

Protein VP0: Protein VP0: VP0 precursor is a component of immature procapsids, which gives rise to VP4 and VP2 after maturation. Allows the capsid to remain inactive before the maturation step.

Protease 2A: cysteine protease that is responsible for the cleavage between the P1 and P2 regions. It cleaves the host translation initiation factor EIF4G1, in order to shut off the capped cellular mRNA transcription (By similarity).

Protein 2B: Affects membrane integrity and cause an increase in membrane permeability.

Protein 2C: Associates with and induces structural rearrangements of intracellular membranes. It displays RNA-binding, nucleotide binding and NTPase activities.

Protein 3A, via its hydrophobic domain, serves as membrane anchor. It also inhibits endoplasmic reticulum-to-Golgi transport.

Protease 3C: cysteine protease that generates mature viral proteins from the precursor polyprotein. In addition to its proteolytic activity, it binds to viral RNA, and thus influences viral genome replication. RNA and substrate bind cooperatively to the protease.

RNA-directed RNA polymerase 3D-POL replicates genomic and antigenomic RNA by recognizing replications specific signals.

Subunit:

Protein 2C N-terminus interacts with human RTN3. This interaction is important for viral replication.

Subcellular Location:

Protein VP2: Virion. Host cytoplasm (Potential).

Protein VP3: Virion. Host cytoplasm (Potential).

Protein VP1: Virion. Host cytoplasm (Potential).

Protein 2B: Host cytoplasmic vesicle membrane; Peripheral membrane protein; Cytoplasmic side (Potential). Note=Probably localizes to the surface of intracellular membrane vesicles that are induced after virus infection as the site for viral RNA replication. These vesicles are derived from the endoplasmic reticulum.

Protein 2C: Host cytoplasmic vesicle membrane; Peripheral membrane protein; Cytoplasmic side (Potential). Note=Probably localizes to the surface of intracellular membrane vesicles that are induced after virus infection as the site for viral RNA replication. These vesicles are derived from the endoplasmic reticulum.

Protein 3A: Host cytoplasmic vesicle membrane; Peripheral membrane protein; Cytoplasmic side (Potential). Note=Probably localizes to the surface of intracellular membrane vesicles that are induced after virus infection as the site for viral RNA replication. These vesicles are derived from the endoplasmic reticulum.

Protein 3B: Virion (Potential).

Protease 3C: Host cytoplasm (Potential).

RNA-directed RNA polymerase 3D-POL: Host cytoplasmic vesicle membrane; Peripheral membrane protein; Cytoplasmic side (Potential). Note=Probably localizes to the surface of intracellular membrane vesicles that are induced after virus infection as the site for viral RNA replication. These vesicles are derived from the endoplasmic reticulum.

Post-translational modifications:

Specific enzymatic cleavages in vivo by the viral proteases yield a variety of precursors and mature proteins. Polyprotein processing intermediates such as VP0 which is a VP4-VP2 precursor are produced. During virion maturation, non-infectious particles are rendered infectious following cleavage of VP0. This maturation cleavage is followed by a conformational change of the particle.

VPg is uridylylated by the polymerase and is covalently linked to the 5'-end of genomic RNA. This uridylylated form acts as a nucleotide-peptide primer for the polymerase. Myristoylation of VP4 is required during RNA encapsidation and formation of the mature virus particle.

Similarity:

Belongs to the picornaviruses polyprotein family.

Contains 2 peptidase C3 domains.

Contains 1 RdRp catalytic domain.

Contains 1 SF3 helicase domain.

SWISS:

Q66478

Gene ID:

N/A

Database links:

[SwissProt: Q66478](#) HE71B

Important Note:

This product as supplied is intended for research use only, not for use in human, therapeutic or diagnostic applications.

肠道病毒71型(Human enterovirus

71)简称EV71。肠病毒在病毒学上的分类是属于微小病毒科(picornaviridae)中的肠病毒群(enterovirus)。

EV

71为目前肠病毒群中最晚发现的病毒，其感染性强且致病率高，尤其是神经系统方面的并发症。EV71主要引起手足口病，还可引起无菌性脑膜炎、脑干脑炎和脊髓灰质炎样的麻痹等多种神经系统疾病。手足口病和中枢神经系统感染是EV71感染而引起的两大常见临床症状。

肠道病毒71型病毒从咽部或肠道侵入，在局部黏膜或淋巴组织中繁殖，并由局部排出，此时可引起局部症状。继而病毒又侵入局部淋巴结，并由此进入血液循环导致第一次病毒血症。病毒经血循环侵入网状内皮组织、深层淋巴结、肝、脾、骨髓等处大量繁殖并由此进入血液循环，引起第二次病毒血症。病毒可随血流进入全身各器官，如中枢神经系统、皮肤黏膜、心脏等处，进一步繁殖并引起病变。