



## Rabbit Anti-Hepatitis C Virus 1b Core protein p19 antibody

SL16472R

<b>Product Name:</b>	Hepatitis C Virus 1b Core protein p19
<b>Chinese Name:</b>	丙型肝炎病毒1b抗体
<b>Alias:</b>	Core protein p19; HCV core antigen; HCV core protein; Hepatitis C Virus core protein; polyprotein [Hepatitis C virus subtype 1b].
<b>Organism Species:</b>	Rabbit
<b>Clonality:</b>	Polyclonal
<b>React Species:</b>	Hepatitis C Virus 1b
<b>Applications:</b>	WB=1:500-2000ELISA=1:500-1000IHC-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.
<b>Molecular weight:</b>	7.6/21kDa
<b>Form:</b>	Lyophilized or Liquid
<b>Concentration:</b>	1mg/ml
<b>immunogen:</b>	KLH conjugated synthetic peptide derived from Hepatitis C Virus 1b Core protein p19:71-170/201
<b>Lsotype:</b>	IgG
<b>Purification:</b>	affinity purified by Protein A
<b>Storage Buffer:</b>	0.01M TBS(pH7.4) with 1% BSA, 0.03% Proclin300 and 50% Glycerol.
<b>Storage:</b>	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.
<b>PubMed:</b>	<a href="#">PubMed</a>
<b>Product Detail:</b>	Core protein packages viral RNA to form a viral nucleocapsid, and promotes virion budding. Modulates viral translation initiation by interacting with HCV IRES and 40S ribosomal subunit. Also regulates many host cellular functions such as signaling pathways and apoptosis. Prevents the establishment of cellular antiviral state by

blocking the interferon-alpha/beta (IFN-alpha/beta) and IFN-gamma signaling pathways and by inducing human STAT1 degradation. Plays an important role in virus-mediated cell transformation leading to hepatocellular carcinomas. Interacts with, and activates STAT3 leading to cellular transformation. May repress the promoter of p53, and sequester CREB3 and SP110 isoform3/Sp110b in the cytoplasm. Also represses cell cycle negative regulating factor CDKN1A, thereby interrupting an important check point of normal cell cycle regulation. Targets transcription factors involved in the regulation of inflammatory responses and in the immune response: suppresses NK-kappaB activation, and activates AP-1. Mediates apoptotic pathways through association with TNF-type receptors TNFRSF1A and LTBR, although its effect on death receptors-induced apoptosis remains controversial. Enhances TRAIL mediated apoptosis, suggesting that it might play a role in mediated apoptosis, suggesting that it might play a role in immune-mediated liver cell injury. Secreted core protein is able to bind C1QR1 at the T-cell surface, resulting in down-regulation of T-lymphocytes proliferation. May transactivate human MYC, Rous sarcoma virus LTR, and SV40 promoters. May suppress the human FOS and HIV-1 LTR activity. May alter lipid metabolism by interacting with hepatocellular proteins involved in lipid accumulation and storage.

**Function:**

Core protein packages viral RNA to form a viral nucleocapsid, and promotes virion budding. Modulates viral translation initiation by interacting with HCV IRES and 40S ribosomal subunit. Also regulates many host cellular functions such as signaling pathways and apoptosis. Prevents the establishment of cellular antiviral state by blocking the interferon-alpha/beta (IFN-alpha/beta) and IFN-gamma signaling pathways and by inducing human STAT1 degradation. Thought to play a role in virus-mediated cell transformation leading to hepatocellular carcinomas. Interacts with, and activates STAT3 leading to cellular transformation. May repress the promoter of p53, and sequester CREB3 and SP110 isoform 3/Sp110b in the cytoplasm. Also represses cell cycle negative regulating factor CDKN1A, thereby interrupting an important check point of normal cell cycle regulation. Targets transcription factors involved in the regulation of inflammatory responses and in the immune response: suppresses NK-kappaB activation, and activates AP-1. Could mediate apoptotic pathways through association with TNF-type receptors TNFRSF1A and LTBR, although its effect on death receptor-induced apoptosis remains controversial. Enhances TRAIL mediated apoptosis, suggesting that it might play a role in immune-mediated liver cell injury. Seric core protein is able to bind C1QR1 at the T-cell surface, resulting in down-regulation of T-lymphocytes proliferation. May transactivate human MYC, Rous sarcoma virus LTR, and SV40 promoters. May suppress the human FOS and HIV-1 LTR activity. Alters lipid metabolism by interacting with hepatocellular proteins involved in lipid accumulation and storage. Core protein induces up-regulation of FAS promoter activity, and thereby probably contributes to the increased triglyceride accumulation in hepatocytes (steatosis).

**Subunit:**

Core protein is a homomultimer that binds the C-terminal part of E1 and interacts with

numerous cellular proteins. Interaction with human STAT1 SH2 domain seems to result in decreased STAT1 phosphorylation, leading to decreased IFN-stimulated gene transcription. In addition to blocking the formation of phosphorylated STAT1, the core protein also promotes ubiquitin-mediated proteasome-dependent degradation of STAT1. Interacts with, and constitutively activates human STAT3. Associates with human LTBR and TNFRSF1A receptors and possibly induces apoptosis. Binds to human SP110 isoform 3/Sp110b, HNRPK, C1QR1, YWHAЕ, UBE3A/E6AP, DDX3X, APOA2 and RXRA proteins. Interacts with human CREB3 nuclear transcription protein, triggering cell transformation. May interact with human p53. Also binds human cytokeratins KRT8, KRT18, KRT19 and VIM (vimentin). E1 and E2 glycoproteins form a heterodimer that binds to human LDLR, CLDN1, CD81 and SCARB1 receptors. E2 binds and inhibits human EIF2AK2/PKR. Also binds human CD209/DC-SIGN and CLEC4M/DC-SIGNR. p7 forms a homoheptamer in vitro. NS2 forms a homodimer containing a pair of composite active sites at the dimerization interface. NS2 seems to interact with all other non-structural (NS) proteins. NS4A interacts with NS3 serine protease and stabilizes its folding. NS3-NS4A complex is essential for the activation of the latter and allows membrane anchorage of NS3. NS3 interacts with human TANK-binding kinase TBK1 and MAVS. NS4B and NS5A form homodimers and seem to interact with all other non-structural (NS) proteins. NS5A also interacts with human EIF2AK2/PKR, FKBP8, GRB2, BIN1, PIK3R1, SRCAP, VAPB and with most Src-family kinases. NS5B is a homooligomer and interacts with human VAPB, HNRNPA1 and SEPT6.

**Subcellular Location:**

Core protein p21: Host endoplasmic reticulum membrane; Single-pass membrane protein. Host mitochondrion membrane; Single-pass type I membrane protein. Host lipid droplet. Note=The C-terminal transmembrane domain of core protein p21 contains an ER signal leading the nascent polyprotein to the ER membrane. Only a minor proportion of core protein is present in the nucleus and an unknown proportion is secreted.

Core protein p19: Virion. Host cytoplasm. Host nucleus. Secreted.

**Post-translational modifications:**

Specific enzymatic cleavages in vivo yield mature proteins. The structural proteins, core, E1, E2 and p7 are produced by proteolytic processing by host signal peptidases. The core protein is synthesized as a 21 kDa precursor which is retained in the ER membrane through the hydrophobic signal peptide. Cleavage by the signal peptidase releases the 19 kDa mature core protein. The other proteins (p7, NS2-3, NS3, NS4A, NS4B, NS5A and NS5B) are cleaved by the viral proteases.

Envelope E1 and E2 glycoproteins are highly N-glycosylated.

Core protein is phosphorylated by host PKC and PKA.

NS5A is phosphorylated in a basal form termed p56. p58 is an hyperphosphorylated form of p56. p56 and p58 coexist in the cell in roughly equivalent amounts.

Hyperphosphorylation is dependent on the presence of NS4A. Human AKT1, RPS6KB1/p70S6K, MAP2K1/MEK1, MAP2K6/MKK6 and CSNK1A1/CKI-alpha kinases may be responsible for NS5A phosphorylation.

NS4B is palmitoylated. This modification may play a role in its polymerization or in protein-protein interactions.

The N-terminus of a fraction of NS4B molecules seems to be relocated post-translationally from the cytoplasm to the ER lumen, with a 5th transmembrane segment. The C-terminus of NS2 may be luminal with a fourth transmembrane segment.

Core protein is ubiquitinated; mediated by UBE3A and leading to core protein subsequent proteasomal degradation.

**Similarity:**

Contains 1 peptidase C18 domain.

Contains 1 peptidase S29 domain.

Contains 1 RdRp catalytic domain.

**SWISS:**

P26662

**Gene ID:**

951475

**Database links:**

[Entrez Gene: 951475](#) Hepatitis C Virus genotype 1b

[SwissProt: P26662](#) Hepatitis C Virus genotype 1b

[SwissProt: P26663](#) Hepatitis C Virus genotype 1b

[SwissProt: Q9WMX2](#) Hepatitis C Virus genotype 1b

**Important Note:**

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