

HAV-P2C (residues 1121-1234) Hepatitis A Virus Core Protein P2C recombinant, *E. coli*

Cat. No.	Amount
PR-1115	100 µg

For *in vitro* use only
Quality guaranteed for 12 months
Store at -20°C

Avoid freeze / thaw cycles

Form

Liquid. Supplied in 10 mM CBB pH 9.6, 0.1% SDS and 50% glycerol.

Protein synonyms/aliases

Genome polyprotein.

Application

Recombinant HAV-P2C may be used in ELISA and Western blots, excellent for detection of HAV with minimal specificity problems.

Specificity

Immunoreactive with sera of HAV-infected individuals.

Molecular Weight

38.5 kDa

Purity

>90% by SDS-PAGE

Description

The E.Coli derived recombinant 38.5 kDa protein contains the P2C immunodominant regions, amino acids 1121-1234.

HAV core proteins are purified by proprietary chromatographic techniques.

Background

Forty-two antigenic domains were identified across the hepatitis A virus (HAV) polyprotein by using a set of 237 overlapping 20-mer synthetic peptides spanning the entire HAV polyprotein and a panel of serum samples from acutely HAV-infected patients. The term "antigenic domain" is used in this study to define a protein region spanned with consecutive overlapping immunoreactive peptides. Nineteen antigenic domains were found within the structural proteins, and 22 were found within the nonstructural proteins, with 1 domain spanning the junction of VP1 and P2A proteins. Five of these domains were considered immunodominant, as judged by both the breadth and the strength of their immunoreactivity. One domain is located within the VP2 protein at position 57-90 aa. A second domain, located at position 767-842 aa, contains the C-terminal part of the VP1 protein and the entire P2A protein. A third domain, located at position 1403-1456 aa, comprises the C-terminal part of the P2C protein and the N-terminal half of the P3A protein. The fourth domain, located at position 1500-1519 aa, includes almost the entire P3B, and the last domain, located at position 1719-1764 aa, contains the C-terminal region of the P3C protein and the N-terminal region of the P3D protein. It is interesting to note that four of the five most immunoreactive domains are derived from small HAV proteins and/or encompass protein cleavage sites separating different HAV proteins.

Selected References:

- Kanda *et al.* (2004) Interference of hepatitis A virus replication by small interfering RNAs. *Biochem. Biophys. Res. Commun.* **318**:341.
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Hu *et al.* (2002) Mutational characteristics in consecutive passage of rapidly replicating variants of hepatitis A virus strain H2 during cell culture adaptation. *World J. Gastroenterol.* **8**:872.
Emerson *et al.* (2002) Identification of VP1/2A and 2C as virulence genes of hepatitis A virus and demonstration of genetic instability of 2C. *J. Virol.* **76**:8551.
Frings *et al.* (2001) Adaptation of primate cell-adapted hepatitis A virus strain HM175 to growth in guinea pig cells is independent of mutations



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in the 5' nontranslated region. *J. Gen. Virol.* **82**:597.