

## Barmah forest virus (BFV) p130 Protein

### General Information

Synonyms: Structural polyprotein;p130

Protein Construction: 801-1239 aa

Species: BFV

Expression Host: E. coli

Accession: P89946

Molecular Weight: 50.7 kDa (predicted)

AA Sequence:

```
YEHSTTMPNQVGIPFKALIERPGYAGLPLSLVVIKSELVPSLVQDYITCNYKTVVPSPIKCCGGAEC SHKNEAD
YKCSVFTGVYPFMWGGAYCFCDTENSQMSEVYVTRGESCEADHAIAYQVHTASLKAQVMISIGELNQTVDVF
VNGDSPARIQQSKFILGPISAWSPFDHKVIVYRDEVYNEDYAPYGSQAGRFGDIQSRTVNSTDVYANTNLK
LKR PASGNVHVPYTQTPSGFSYWKKEKGVPLNRNAPFGCIKVNVPVRAENCVYGNIPISMDIADAHFTRIDESP
SVSLKACEVQSCTYSSDFGGVASISYTSNKVKGCAIHSNSATMKDSVQDVQESGALSFFATSSVEPNFVV
QVCNARITCHGKCEPPKDHIVPYAAKHND AEFPSISTTAWQWLAHTTSGPLTILVVAIIVVVVVSI VVCARH
```

### QC Testing

Biological Activity: Activity has not been tested. It is theoretically active, but we cannot guarantee it. If you require protein activity, we recommend choosing the eukaryotic expression version first.

Purity: > 85% as determined by SDS-PAGE.

Endotoxin: < 1.0 EU/µg of the protein as determined by the LAL method.

Formulation: If the delivery form is liquid, the default storage buffer is Tris/PBS-based buffer, 5%-50% glycerol. If the delivery form is lyophilized powder, the buffer before lyophilization is Tris/PBS-based buffer, 6% Trehalose, pH 8.0.

### Preparation and Storage

Reconstitution:

Reconstitute the lyophilized protein in sterile deionized water. The product concentration should not be less than 100 µg/mL. Before opening, centrifuge the tube to collect powder at the bottom. After adding the reconstitution buffer, avoid vortexing or pipetting for mixing.

Stability & Storage:

Lyophilized powders can be stably stored for over 12 months, while liquid products can be stored for 6-12 months at -80°C. For reconstituted protein solutions, the solution can be stored at -20°C to -80°C for at least 3 months. Please avoid multiple freeze-thaw cycles and store products in aliquots.

Actual storage temperature shall be subject to the COA.

Shipping:

In general, lyophilized powders are shipped with blue ice, while solutions are shipped with dry ice.

**Protein Background**

Forms an icosahedral capsid with a T=4 symmetry composed of 240 copies of the capsid protein surrounded by a lipid membrane through which penetrate 80 spikes composed of trimers of E1-E2 heterodimers. The capsid protein binds to the viral RNA genome at a site adjacent to a ribosome binding site for viral genome translation following genome release. Possesses a protease activity that results in its autocatalytic cleavage from the nascent structural protein. Following its self-cleavage, the capsid protein transiently associates with ribosomes, and within several minutes the protein binds to viral RNA and rapidly assembles into icosahedric core particles. The resulting nucleocapsid eventually associates with the cytoplasmic domain of the spike glycoprotein E2 at the cell membrane, leading to budding and formation of mature virions. In case of infection, new virions attach to target cells and after clathrin-mediated endocytosis their membrane fuses with the host endosomal membrane. This leads to the release of the nucleocapsid into the cytoplasm, followed by an uncoating event necessary for the genomic RNA to become accessible. The uncoating might be triggered by the interaction of capsid proteins with ribosomes. Binding of ribosomes would release the genomic RNA since the same region is genomic RNA-binding and ribosome-binding. Specifically inhibits interleukin-1 receptor-associated kinase 1/IRAK1-dependent signaling during viral entry, representing a means by which the alphaviruses may evade innate immune detection and activation prior to viral gene expression.; Provides the signal sequence for the translocation of the precursor of protein E3/E2 to the host endoplasmic reticulum. Furin-cleaved E3 remains associated with spike glycoprotein E1 and mediates pH protection of the latter during the transport via the secretory pathway. After virion release from the host cell, the assembly protein E3 is gradually released in the extracellular space.; Plays a role in viral attachment to target host cell, by binding to the cell receptor. Synthesized as a p62 precursor which is processed by furin at the cell membrane just before virion budding, giving rise to E2-E1 heterodimer. The p62-E1 heterodimer is stable, whereas E2-E1 is unstable and dissociate at low pH. p62 is processed at the last step, presumably to avoid E1 fusion activation before its final export to cell surface. E2 C-terminus contains a transitory transmembrane that would be disrupted by palmitoylation, resulting in reorientation of the C-terminal tail from lumenal to cytoplasmic side. This step is critical since E2 C-terminus is involved in budding by interacting with capsid proteins. This release of E2 C-terminus in cytoplasm occurs late in protein export, and precludes premature assembly of particles at the endoplasmic reticulum membrane.; Constitutive membrane protein involved in virus glycoprotein processing, cell permeabilization, and the budding of viral particles. Disrupts the calcium homeostasis of the cell, probably at the endoplasmic reticulum level. This leads to cytoplasmic calcium elevation. Because of its lipophilic properties, the 6K protein is postulated to influence the selection of lipids that interact with the transmembrane domains of the glycoproteins, which, in turn, affects the deformability of the bilayer required for the extreme curvature that occurs as budding proceeds. Present in low amount in virions, about 3% compared to viral glycoproteins.; Class II viral fusion protein. Fusion activity is inactive as long as E1 is bound to E2 in mature virion. After virus attachment to target cell and endocytosis, acidification of the endosome would induce dissociation of E1/E2 heterodimer and concomitant trimerization of the E1 subunits. This E1 trimer is fusion active, and promotes release of viral nucleocapsid in cytoplasm after endosome and viral membrane fusion. Efficient fusion requires the presence of cholesterol and sphingolipid in the target membran

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