

DESCRIPTION

Species Reactivity	Viral
Specificity	Detects viral CCI in direct ELISAs and Western blots.
Source	Monoclonal Mouse IgG ₁ Clone # 112803
Purification	Protein A or G purified from ascites
Immunogen	<i>S. frugiperda</i> insect ovarian cell line Sf 21-derived recombinant viral CCI Met17-Val258 Accession # P19063
Conjugate	Alexa Fluor 700 Excitation Wavelength: 675-700 nm Emission Wavelength: 723 nm
Formulation	Supplied 0.2mg/ml in 1X PBS with RDF1 and 0.09% Sodium Azide
*Contains <0.1% Sodium Azide, which is not hazardous at this concentration according to GHS classifications. Refer to the Safety Data Sheet (SDS) for additional information and handling instructions.	

APPLICATIONS

Please Note: Optimal dilutions should be determined by each laboratory for each application. [General Protocols](#) are available in the Technical Information section on our website.

Neutralization	Optimal dilution of this antibody should be experimentally determined.
Western Blot	Optimal dilution of this antibody should be experimentally determined.

PREPARATION AND STORAGE

Shipping	The product is shipped with polar packs. Upon receipt, store it immediately at the temperature recommended below.
Stability & Storage	Protect from light. Do not freeze. 12 months from date of receipt, 2 to 8 °C as supplied

BACKGROUND

The family of T1/35 kDa proteins are secreted, soluble proteins encoded by the '35K' virulence gene of many poxviruses. These proteins have been shown to bind CC-chemokines with high affinity and are now termed viral chemokine inhibitor (vCCI). Viral CCI from various poxviruses share multiple stretches of identical sequence motif and eight conserved cysteine residues. The vaccinia virus (strain Lister) vCCI cDNA encodes a 258 amino acid (aa) residue protein with a putative 17 amino acid residue signal peptide. Vaccinia virus (strain Lister) vCCI shows greater than 90% aa sequence identity with vCCI from other orthopoxviruses and approximately 40% aa sequence identity with the leporipoxvirus T-1 proteins.

Recombinant vCCI has been shown to be a potent general inhibitor of CC-chemokine activity *in vitro* and blocks binding of CC chemokines to cell surface chemokine receptors. In *in vivo* studies using a virus mutant in which the gene encoding the CCI has been deleted, leukocyte infiltration into the virus-infected areas is increased, suggesting that CCI can modulate the influx of inflammatory cells into virus-infected tissues.

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