SANTA CRUZ BIOTECHNOLOGY, INC.

CPV CrmA (vN-13): sc-21361

SANTA CRUZ BIOTECHNOLOGY

The Power to Question

BACKGROUND

Poxviruses include some of the most virulent of all human pathogens, and this results, in part, from their abilities to counter host defenses against infection. One powerful way in which poxviruses subvert the host immune response is through the expression of a family of cytokine-response modifiers that affect host cytokine responses in various ways, including inhibition of synthesis and release of cytokines from infected cells, interference of the interaction between a cytokine and its receptor, and production of viral cytokines that antagonize the effects of host cytokines. Cytokine response modifier A (CrmA) is a well-studied member of this family of cowpox proteins, which targets members of the caspase family of proteases. Caspases initiate apoptosis of infected cells through proteolysis of active cell surface receptors and trigger activation of the pro-inflammatory cytokines interleukin-1β and interleukin-18. Also known as serine protease inhibitor-2 (SPI2), CrmA is a 38 kDa protein that has the typical fold of a cleaved serpin, despite the fact that it lacks the N-terminal half of the A helix, the entire D helix, and a portion of the E helix that are present on known host serpins. The minimal structure of the protein is an example of viral economy, resulting in a protein that retains its caspase docking ability but lacks regions not needed for structural integrity or inhibitory activity.

REFERENCES

1. Pickup, D. 1994. Poxviral modifiers of cytokine responses to infection. Infect. Agents Dis. 3: 116-127.
2. Bird, P. 1998. Serpins and regulation of cell death. Results Probl. Cell Differ. 24: 63-89.
3. Turner, S., Kenshole, B. and Ruby, J. 1999. Viral modulation of the host response via CrmA/SPI2 expression. Immunol. Cell. Biol. 77: 236-241.
4. Renatus, M., Zhou, Q., Stennicke, H., Snipas, S., Turk, D., Bankston, L., Liddington, R. and Salvesen, G. 2000. Crystal structure of the apoptotic suppressor CrmA in its cleaved form. Structure Fold. Des. 8: 789-797.
5. Snipas, S., Stennicke, H., Riedl, S., Potempa, J., Tavis, J., Barrett, A. and Salvesen, G. 2001. Inhibition of distant caspase homologues by natural caspase inhibitors. Biochem. J. 357: 575-580.

SOURCE

CPV CrmA (vN-13) is an affinity purified goat polyclonal antibody raised against a peptide mapping near the N-terminus of CrmA of cowpox virus origin.

PRODUCT

Each vial contains 200 µg IgG in 1.0 ml of PBS with < 0.1% sodium azide and 0.1% gelatin.

Blocking peptide available for competition studies, sc-21361 P, (100 µg peptide in 0.5 ml PBS containing < 0.1% sodium azide and 0.2% BSA).

STORAGE

Store at 4° C, **DO NOT FREEZE**. Stable for one year from the date of shipment. Non-hazardous. No MSDS required.

APPLICATIONS

CPV CrmA (vN-13) is recommended for detection of CrmA of cowpox virus origin by Western Blotting (starting dilution 1:200, dilution range 1:100-1:1000) and solid phase ELISA (starting dilution 1:30, dilution range 1:30-1:3000).

RECOMMENDED SECONDARY REAGENTS

To ensure optimal results, the following support (secondary) reagents are recommended: 1) Western Blotting: use donkey anti-goat IgG-HRP: sc-2020 (dilution range: 1:2000-1:100,000) or Cruz Marker™ compatible donkey anti-goat IgG-HRP: sc-2033 (dilution range: 1:2000-1:5000), Cruz Marker™ Molecular Weight Standards: sc-2035, TBS Blotto A Blocking Reagent: sc-2333 and Western Blotting Luminol Reagent: sc-2048.

RESEARCH USE

For research use only, not for use in diagnostic procedures.

PROTOCOLS

See our web site at www.scbt.com or our catalog for detailed protocols and support products.