SANTA CRUZ BIOTECHNOLOGY, INC.

HHV-8 K-bZIP (F33P1): sc-69797

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The Power to Question

BACKGROUND

HHV-8 (human herpes virus type 8), also designated Kaposi's sarcoma-associated herpesvirus, is associated with multicentric Castleman's disease and primary effusion lymphoma (PEL), a rare type of non-Hodgkin lymphoma affecting body cavities. HHV-8 encodes a viral cyclin that is homologous to cellular D-type cyclins (cyclin D1-D3), a class of positive cell cycle mediators that are physiologically regulated by p27 (a cell cycle inhibitor). Although related to D-type cyclins, HHV-8 cyclin is not sensitive to p27, which may explain the coexistence of p27 and HHV-8 that is observed in individuals affected by PEL. HHV-8 K-bZIP, also known simply as K-bZIP, is a 237 amino acid homodimeric phosphorprotein that regulates HHV-8 viral transcription. Specifically, HHV-8 K-bZIP functions as a transactivator that, via a prototypic basic leucine zipper domain at its carboxy terminus, can bind to a variety of promoters, thereby inducing the viral replication cycle. In addition, HHV-8 K-bZIP is thought to repress p53-mediated apoptosis, further facilitating viral transcription and replication.

REFERENCES

1. Liao, W., et al. 2003. K-bZIP of Kaposi's sarcoma-associated herpesvirus/human herpesvirus 8 (KSHV/ HHV-8) binds KSHV/HHV-8 Rta and represses Rta-mediated transactivation. J. Virol. 77: 3809-3815.
2. Tomita, M., et al. 2004. The Kaposi's sarcoma-associated herpesvirus K-bZIP protein represses transforming growth factor β signaling through interaction with CREB-binding protein. Oncogene 23: 8272-8281.

SOURCE

HHV-8 K-bZIP (F33P1) is a mouse monoclonal antibody raised against HHV-8.

PRODUCT

Each vial contains 100 µg IgG_1 kappa light chain in 1.0 ml of PBS with < 0.1% sodium azide and 0.1% gelatin.

APPLICATIONS

HHV-8 K-bZIP (F33P1) is recommended for detection of sumoylated and non-sumoylated K-bZIP of HHV-8 origin by Western Blotting (starting dilution 1:200, dilution range 1:100-1:1000) and immunofluorescence (starting dilution 1:50, dilution range 1:50-1:500).

Molecular Weight of HHV-8 K-bZIP: 37 kDa.

RECOMMENDED SUPPORT REAGENTS

To ensure optimal results, the following support reagents are recommended: 1) Western Blotting: use m-IgGκ BP-HRP: sc-516102 or m-IgGκ BP-HRP (Cruz Marker): sc-516102-CM (dilution range: 1:1000-1:10000), Cruz Marker™ Molecular Weight Standards: sc-2035, UltraCruz® Blocking Reagent: sc-516214 and Western Blotting Luminol Reagent: sc-2048. 2) Immunofluorescence: use m-IgGκ BP-FITC: sc-516140 or m-IgGκ BP-PE: sc-516141 (dilution range: 1:50-1:200) with UltraCruz® Mounting Medium: sc-24941 or UltraCruz® Hard-set Mounting Medium: sc-359850.

STORAGE

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

SELECT PRODUCT CITATIONS

1. Haas, D.A., et al. 2013. The inflammatory kinase MAP4K4 promotes reactivation of Kaposi's sarcoma herpesvirus and enhances the invasiveness of infected endothelial cells. PLoS Pathog. 9: e1003737.
2. Granato, M., et al. 2015. The activation of KSHV lytic cycle blocks autophagy in PEL cells. Autophagy 11: 1978-1986.
3. Zhang, G., et al. 2016. Cytoplasmic isoforms of Kaposi sarcoma herpesvirus LANA recruit and antagonize the innate immune DNA sensor cGAS. Proc. Natl. Acad. Sci. USA 113: E1034-E1043.
4. Granato, M., et al. 2017. Quercetin induces apoptosis and autophagy in primary effusion lymphoma cells by inhibiting PI3K/Akt/mTOR and Stat3 signaling pathways. J. Nutr. Biochem. 41: 124-136.
5. Granato, M., et al. 2017. Metformin triggers apoptosis in PEL cells and alters bortezomib-induced unfolded protein response increasing its cytotoxicity and inhibiting KSHV lytic cycle activation. Cell. Signal. 40: 239-247.
6. Gonnella, R., et al. 2017. Oxidant species are involved in T/B-mediated ERK1/2 phosphorylation that activates p53-p21 axis to promote KSHV lytic cycle in PEL cells. Free Radic. Biol. Med. 112: 327-335.
7. Abere, B., et al. 2017. The Kaposi's sarcoma-associated herpesvirus (KSHV) non-structural membrane protein K15 is required for viral lytic replication and may represent a therapeutic target. PLoS Pathog. 13: e1006639.
8. Abere, B., et al. 2018. Kaposi's sarcoma-associated herpesvirus nonstructural membrane protein pK15 recruits the class II phosphatidylinositol 3-kinase PI3K-C2α to activate productive viral replication. J. Virol. 92: e00544-18.
9. Granato, M., et al. 2018. Cytotoxic drugs activate KSHV lytic cycle in latently infected PEL cells by inducing a moderate ROS increase controlled by HSF1, Nrf2 and p62/SQSTM1. Viruses 11: 8.
10. Santarelli, R., et al. 2019. Stat3 phosphorylation affects p53/p21 axis and KSHV lytic cycle activation. Virology 528: 137-143.
11. Koch, S., et al. 2019. Kaposi's sarcoma-associated herpesvirus vIRF2 protein utilizes an IFN-dependent pathway to regulate viral early gene expression. PLoS Pathog. 15: e1007743.
12. Gilardini Montani, M.S., et al. 2019. Kaposi sarcoma herpes virus (KSHV) infection inhibits macrophage formation and survival by counteracting macrophage colony-stimulating factor (M-CSF)-induced increase of reactive oxygen species (ROS), c-Jun N-terminal kinase (JNK) phosphorylation and autophagy. Int. J. Biochem. Cell Biol. 114: 105560.

RESEARCH USE

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