SANTA CRUZ BIOTECHNOLOGY, INC.

EBV ZEBRA (BZ1): sc-53904

SANTA CRUZ BIOTECHNOLOGY

The Power to Question

BACKGROUND

Epstein-Barr virus, frequently referred to as EBV, is a member of the herpesvirus family and one of the most common human viruses. The EBV latent membrane protein 1, otherwise known as LMP1, plays a central role in the transformation process. LMP1 mimics activated receptors of the tumor necrosis factor receptor superfamily to promote cell growth and antiapoptotic mechanisms. LMP1 and other EBV latent proteins upregulate cytokines and growth factors which participate in autocrine and paracrine loops that are likely to promote cell transformation and modulate immune responses. In addition, the crucial Epstein-Barr virus (EBV) gene, ZEBRA, acts as a switch between latency and replication of this herpesvirus. During latency of EBV, ZEBRA expression is repressed. Inducing stimuli cause synthesis of ZEBRA which, in turn, activates expression of several individual EBV early genes. The ZEBRA polypeptide is a site-specific DNA binding protein that is likely to function as a transcriptional transactivator.

REFERENCES

1. Young, L.S., et al. 1988. Epstein-Barr virus gene expression in nasopharyngeal carcinoma. J. Gen. Virol. 5: 1051-1065.
2. Miller, G. 1990. The switch between latency and replication of Epstein-Barr virus. J. Infect. Dis. 5: 833-844.

SOURCE

EBV ZEBRA (BZ1) is a mouse monoclonal antibody raised against full-length recombinant EBV ZEBRA protein.

PRODUCT

Each vial contains 200 µg IgG_1 kappa light chain in 1.0 ml of PBS with < 0.1% sodium azide and 0.1% gelatin.

EBV ZEBRA (BZ1) is available conjugated to agarose (sc-53904 AC), 500 µg/ 0.25 ml agarose in 1 ml, for IP; to HRP (sc-53904 HRP), 200 µg/ml, for WB, IHC(P) and ELISA; to either phycoerythrin (sc-53904 PE), fluorescein (sc-53904 FITC), Alexa Fluor® 488 (sc-53904 AF488), Alexa Fluor® 546 (sc-53904 AF546), Alexa Fluor® 594 (sc-53904 AF594) or Alexa Fluor® 647 (sc-53904 AF647), 200 µg/ml, for WB (RGB), IF, IHC(P) and FCM; and to either Alexa Fluor® 680 (sc-53904 AF680) or Alexa Fluor® 790 (sc-53904 AF790), 200 µg/ml, for Near-Infrared (NIR) WB, IF and FCM.

APPLICATIONS

EBV ZEBRA (BZ1) is recommended for detection of ZEBRA of EBV by Western Blotting (starting dilution 1:200, dilution range 1:100-1:1000), immunoprecipitation [1-2 µg per 100-500 µg of total protein (1 ml of cell lysate)], immunofluorescence (starting dilution 1:50, dilution range 1:50-1:500) and solid phase ELISA (starting dilution 1:30, dilution range 1:30-1:3000).

Molecular Weight of EBV ZEBRA: 38 kDa.

RESEARCH USE

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

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. Jiang, Y., et al. 2008. Mutual inhibition between Kaposi's sarcoma-associated herpesvirus and Epstein-Barr virus lytic replication initiators in dually-infected primary effusion lymphoma. PLoS ONE 3: e1569.
2. Guo, Q., et al. 2010. Transactivators Zta and Rta of Epstein-Barr virus promote G_0/G_1 to S transition in Raji cells: a novel relationship between lytic virus and cell cycle. Mol. Immunol. 47: 1783-1792.
3. Robinson, A.R., et al. 2011. Cellular transcription factor Oct-1 interacts with the Epstein-Barr virus BRLF1 protein to promote disruption of viral latency. J. Virol. 85: 8940-8953.
4. Sivachandran, N., et al. 2012. Functions of the Epstein-Barr virus EBNA1 protein in viral reactivation and lytic infection. J. Virol. 86: 6146-6158.
5. Robinson, A.R., et al. 2012. The B-cell specific transcription factor, Oct-2, promotes Epstein-Barr virus latency by inhibiting the viral immediate-early protein, BZLF1. PLoS Pathog. 8: e1002516.
6. Sun, X., et al. 2013. Hsp90 inhibitor 17-DMAG decreases expression of conserved herpesvirus protein kinases and reduces virus production in Epstein-Barr virus-infected cells. J. Virol. 87: 10126-10138.
7. Iempridee, T., et al. 2014. Epstein-Barr virus utilizes Ikaros in regulating its latent-lytic switch in B cells. J. Virol. 88: 4811-4827.
8. Granato, M., et al. 2014. Epstein-Barr virus blocks the autophagic flux and appropriates the autophagic machinery to enhance viral replication. J. Virol. 88: 12715-12726.
9. Reusch, J.A., et al. 2015. Cellular differentiation regulator BLIMP1 induces Epstein-Barr virus lytic reactivation in epithelial and B cells by activating transcription from both the R and Z promoters. J. Virol. 89: 1731-1743.
10. Strong, M.J., et al. 2015. Latent expression of the Epstein-Barr virus (EBV)-encoded major histocompatibility complex class I TAP inhibitor, BNLF2a, in EBV-positive gastric carcinomas. J. Virol. 89: 10110-10114.
11. Li, Y., et al. 2015. Epstein-Barr virus BZLF1-mediated downregulation of proinflammatory factors is essential for optimal lytic viral replication. J. Virol. 90: 887-903.
12. Wille, C.K., et al. 2015. 5-hydroxymethylation of the EBV genome regulates the latent to lytic switch. Proc. Natl. Acad. Sci. USA 112: E7257-E7265.
13. Kraus, R.J., et al. 2017. Hypoxia-inducible factor-1α plays roles in Epstein-Barr virus's natural life cycle and tumorigenesis by inducing lytic infection through direct binding to the immediate-early BZLF1 gene promoter. PLoS Pathog. 13: e1006404.

PROTOCOLS

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