bs-4542R **[Primary Antibody]**

BRLF1 Rabbit pAb

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DATASHEET

Host: Rabbit **Isotype:** IgG

Clonality: Polyclonal

Target: BRLF1

Immunogen: KLH conjugated synthetic peptide derived from HHV4tp2 BRLF1: 81-180/605.

Purification: affinity purified by Protein A

Concentration: 1mg/ml

Storage: 0.01M TBS (pH7.4) with 1% BSA, 0.02% Proclin300 and 50% Glycerol.
Shipped at 4°C. Store at -20°C for one year. Avoid repeated freeze/thaw cycles.

Background: Expression of the Epstein-Barr virus (EBV) immediate-early (IE) protein BRLF1 induces the lytic form of viral replication in most EBV-positive cell lines. BRLF1 is a transcriptional activator that binds directly to a GC-rich motif present in some EBV lytic gene promoters. However, BRLF1 activates transcription of the other IE protein, BZLF1, through an indirect mechanism which we previously showed to require activation of the stress mitogen-activated protein kinases. Here we demonstrate that BRLF1 activates phosphatidylinositol-3 (PI3) kinase signaling in host cells. We show that the specific PI3 kinase inhibitor, LY294002, completely abrogates the ability of a BRLF1 adenovirus vector to induce the lytic form of EBV infection, while not affecting lytic infection induced by a BZLF1 adenovirus vector. Furthermore, we demonstrate that the requirement for PI3 kinase activation in BRLF1-induced transcriptional activation is promoter dependent. BRLF1 activation of the SM early promoter (which occurs through a direct binding mechanism) does not require PI3 kinase activation, whereas activation of the IE BZLF1 and early BMRF1 promoters requires PI3 kinase activation. Thus, there are clearly two separate mechanisms by which BRLF1 induces transcriptional activation.

Applications: **WB** (1:500-2000)
IHC-P (1:100-500)
IHC-F (1:100-500)
IF (1:100-500)
ELISA (1:5000-10000)

Reactivity: (predicted: HHV-4)

Predicted MW.: 66.5 kDa

SELECTED CITATIONS

- **[IF=11.205]** Ruo-Wen Xiao. et al. Rare POLN mutations confer risk for familial nasopharyngeal carcinoma through weakened Epstein-Barr virus lytic replication. EBIOMEDICINE. 2022 Oct;84:104267 WB ;Human. 36116213
- **[IF=3.329]** Hui D et al. CD44+CD24-/low sphere-forming cells of EBV-associated gastric carcinomas show immunosuppressive effects and induce Tregs partially through production of PGE2. Exp Cell Res. 2020 May 15;390(2):111968. WB ;human. 32197932
- **[IF=0]** Granato M et al. Quercetin Interrupts the Positive Feedback Loop Between STAT3 and IL-6, Promotes Autophagy, and Reduces ROS, Preventing EBV-Driven B Cell Immortalization. Biomolecules. 2019 Sep 12;9(9). pii: E482. WB ;Human. 31547402