

# HSV-1 ICP4 (H943): sc-69809

## BACKGROUND

Infected-cell polypeptide 4 (ICP4) of herpes simplex virus type 1 (HSV-1) is one of five immediate early transcriptional regulatory proteins produced promptly upon infection. ICP4 is required for the adequate transcription of early and late viral genes. Necessary for viral growth, ICP4 Immediate early protein functions to amplify the rates of transcription of viral genes during viral infection by activating gene expression. ICP4 Immediate early protein also initiates transcription in reconstituted transcription reactions. By either increasing or decreasing the rate of formation of transcription initiation complexes mediated by RNA polymerase II, transcription is activated through a set of general transcription factors (GTFs). ICP4 immediate early protein specifically promotes transcription PIC (preinitiation complexes) formation by increasing the binding of TFIID to the TATA box. Data suggests that upon infection, the ICP4 protein also retains a critical role in directing the endless looped conformation of the HSV-1 genome.

## REFERENCES

1. Bartholomew, C., et al. 1987. Transmission of HTLV-I and HIV among homosexual men in Trinidad. *JAMA* 257: 2604-2608.
2. Smith, C.A., et al. 1993. ICP4, the major transcriptional regulatory protein of herpes simplex virus type 1, forms a tripartite complex with TATA-binding protein and TFIIB. *J. Virol.* 67: 4676-4687.
3. Gu, B., et al. 1995. Repression of activator-mediated transcription by herpes simplex virus ICP4 via a mechanism involving interactions with the basal transcription factors TATA-binding protein and TFIIB. *Mol. Cell. Biol.* 15: 3618-3626.

## SOURCE

HSV-1 ICP4 (H943) is a mouse monoclonal antibody raised against herpes virus.

## PRODUCT

Each vial contains 100 µg IgG<sub>1</sub> kappa light chain in 1.0 ml of PBS with < 0.1% sodium azide and 0.1% gelatin.

## APPLICATIONS

HSV-1 ICP4 (H943) is recommended for detection of ICP4 of HSV-1 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 HSV-1 ICP4: 175 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. Yao, F., et al. 2010. Development of a regulatable oncolytic herpes simplex virus type 1 recombinant virus for tumor therapy. *J. Virol.* 84: 8163-8171.
2. Nagel, C.H., et al. 2011. Herpes simplex virus immediate-early protein ICP0 is targeted by SIAH-1 for proteasomal degradation. *J. Virol.* 85: 7644-7657.
3. Qiu, M., et al. 2013. Zinc ionophores pyrithione inhibits herpes simplex virus replication through interfering with proteasome function and NFκB activation. *Antiviral Res.* 100: 44-53.
4. Jamin, A., et al. 2014. Barrier to auto integration factor becomes dephosphorylated during HSV-1 infection and can act as a host defense by impairing viral DNA replication and gene expression. *PLoS ONE* 9: e100511.
5. Lee, C.J., et al. 2014. Cathelicidin LL-37 and HSV-1 corneal infection: peptide versus gene therapy. *Transl. Vis. Sci. Technol.* 3: 4.
6. Zhang, W., et al. 2015. A novel oHSV-1 targeting telomerase reverse transcriptase-positive cancer cells via tumor-specific promoters regulating the expression of ICP4. *Oncotarget* 6: 20345-20355.
7. Crow, M.S. and Cristea, I.M. 2017. Human antiviral protein IFIX suppresses viral gene expression during HSV-1 infection and is counteracted by virus-induced proteasomal degradation. *Mol. Cell. Proteomics* 16: S200-S214.
8. Wang, P., et al. 2019. IL-36 promotes anti-viral immunity by boosting sensitivity to IFN-α/β in IRF1 dependent and independent manners. *Nat. Commun.* 10: 4700.
9. Faccin-Galhardi, L.C., et al. 2019. Assessment of antiherpetic activity of nonsulfated and sulfated polysaccharides from *Azadirachta indica*. *Int. J. Biol. Macromol.* 137: 54-61.
10. Han, M., et al. 2019. Synthetic lethality of cytolytic HSV-1 in cancer cells with ATRX and PML deficiency. *J. Cell Sci.* 132: jcs222349.
11. Adlakha, M., et al. 2020. The HSV-1 immediate early protein ICP22 is a functional mimic of a cellular J protein. *J. Virol.* 94: e01564-19.
12. Luo, Y., et al. 2020. Tumor-targeting oncolytic virus elicits potent immunotherapeutic vaccine responses to tumor antigens. *Oncoimmunology* 9: 1726168.
13. Shen, Y., et al. 2020. The interferon-inducible protein TDRD7 inhibits AMP-activated protein kinase and thereby restricts autophagy-independent virus replication. *J. Biol. Chem.* 295: 6811-6822.
14. Wang, W., et al. 2020. Near-atomic cryo-electron microscopy structures of varicella-zoster virus capsids. *Nat. Microbiol.* 5: 1542-1552.

## RESEARCH USE

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