

VE-cadherin (BV9): sc-52751

BACKGROUND

The cadherins are a family of Ca²⁺-dependent adhesion molecules that function to mediate cell-cell binding critical to the maintenance of tissue structure and morphogenesis. Cadherins each contain a large extracellular domain at the amino-terminus, which is characterized by a series of five homologous repeats, the most distal of which is thought to be responsible for binding specificity. The relatively short carboxy-terminal, intracellular domain interacts with a variety of cytoplasmic proteins, including β -catenin, to regulate cadherin function. VE-cadherin (for vascular endothelial cadherin, also designated cadherin-5) is localized at intercellular junctions of endothelial cells, where it is thought to play a role in the cohesion and organization of intercellular junctions.

CHROMOSOMAL LOCATION

Genetic locus: CDH5 (human) mapping to 16q21; Cdh5 (mouse) mapping to 8 D3.

SOURCE

VE-cadherin (BV9) is a mouse monoclonal antibody raised against recombinant VE-Cadherin within an extracellular domain of human origin.

PRODUCT

Each vial contains 200 μ g IgG_{2a} kappa light chain in 1.0 ml of PBS with < 0.1% sodium azide and 0.1% gelatin.

VE-cadherin (BV9) is available conjugated to agarose (sc-52751 AC), 500 μ g/0.25 ml agarose in 1 ml, for IP; to HRP (sc-52751 HRP), 200 μ g/ml, for WB, IHC(P) and ELISA; to either phycoerythrin (sc-52751 PE), fluorescein (sc-52751 FITC), Alexa Fluor[®] 488 (sc-52751 AF488), Alexa Fluor[®] 546 (sc-52751 AF546), Alexa Fluor[®] 594 (sc-52751 AF594) or Alexa Fluor[®] 647 (sc-52751 AF647), 200 μ g/ml, for WB (RGB), IF, IHC(P) and FCM; and to either Alexa Fluor[®] 680 (sc-52751 AF680) or Alexa Fluor[®] 790 (sc-52751 AF790), 200 μ g/ml, for Near-Infrared (NIR) WB, IF and FCM.

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APPLICATIONS

VE-cadherin (BV9) is recommended for detection of VE-cadherin of mouse, rat and human origin by Western Blotting (starting dilution 1:200, dilution range 1:100-1:1000), immunoprecipitation [1-2 μ g per 100-500 μ g of total protein (1 ml of cell lysate)], immunofluorescence (starting dilution 1:50, dilution range 1:50-1:500) and flow cytometry (1 μ g per 1 x 10⁶ cells).

Suitable for use as control antibody for VE-cadherin siRNA (h): sc-36814, VE-cadherin siRNA (m): sc-36813, VE-cadherin shRNA Plasmid (h): sc-36814-SH, VE-cadherin shRNA Plasmid (m): sc-36813-SH, VE-cadherin shRNA (h) Lentiviral Particles: sc-36814-V and VE-cadherin shRNA (m) Lentiviral Particles: sc-36813-V.

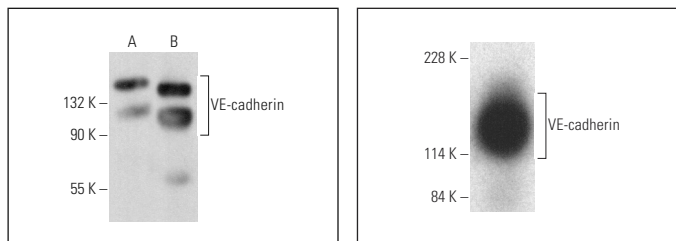
Molecular Weight of VE-cadherin: 130 kDa.

Positive Controls: human lung extract: sc-363767, NCI-H292 whole cell lysate: sc-364179 or HUV-EC-C whole cell lysate: sc-364180.

STORAGE

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

DATA



VE-cadherin (BV9): sc-52751. Western blot analysis of VE-cadherin expression in NCI-H292 whole cell lysate (A) and human lung tissue extract (B).

VE-cadherin (BV9): sc-52751. Direct western blot analysis of VE-cadherin expression in human lung tissue extract.

SELECT PRODUCT CITATIONS

1. Ferreira, L.S., et al. 2007. Bioactive hydrogel scaffolds for controllable vascular differentiation of human embryonic stem cells. *Biomaterials* 28: 2706-2717.
2. Sidibé, A., et al. 2012. Soluble VE-cadherin in rheumatoid arthritis patients correlates with disease activity: evidence for tumor necrosis factor α -induced VE-cadherin cleavage. *Arthritis Rheum.* 64: 77-87.
3. Dave, J.M., et al. 2013. Proteomic profiling of endothelial invasion revealed receptor for activated C kinase 1 (RACK1) complexed with vimentin to regulate focal adhesion kinase (FAK). *J. Biol. Chem.* 288: 30720-30733.
4. Gong, H., et al. 2015. HIF2 α signaling inhibits adherens junctional disruption in acute lung injury. *J. Clin. Invest.* 125: 652-664.
5. Wilson, H.K., et al. 2016. Cryopreservation of brain endothelial cells derived from human induced pluripotent stem cells is enhanced by rho-associated coiled coil-containing kinase inhibition. *Tissue Eng. Part C Methods* 22: 1085-1094.
6. Bartolomé, R.A., et al. 2017. VE-cadherin RGD motifs promote metastasis and constitute a potential therapeutic target in melanoma and breast cancers. *Oncotarget* 8: 215-227.
7. Lant, B., et al. 2018. Interrogating the ccm-3 gene network. *Cell Rep.* 24: 2857-2868.
8. Kouam, P.N., et al. 2019. Ionizing radiation increases the endothelial permeability and the transendothelial migration of tumor cells through ADAM10-activation and subsequent degradation of VE-cadherin. *BMC Cancer* 19: 958.
9. Maroufi, N.F., et al. 2020. Inhibitory effect of melatonin on hypoxia-induced vasculogenic mimicry via suppressing epithelial-mesenchymal transition (EMT) in breast cancer stem cells. *Eur. J. Pharmacol.* 881: 173282.

RESEARCH USE

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