SANTA CRUZ BIOTECHNOLOGY, INC.

# VE-cadherin (C-19): sc-6458

SANTA CRUZ BIOTECHNOLOGY

*The Power to Question*

## BACKGROUND

The cadherins are a family of $Ca^{2+}$-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 (C-19) is an affinity purified goat polyclonal antibody raised against a peptide mapping at the C-terminus of VE-cadherin of human origin.

## PRODUCT

Each vial contains 200 µg IgG in 1.0 ml of PBS with < 0.1% sodium azide and 0.1% gelatin.

Blocking peptide available for competition studies, sc-6458 P, (100 µg peptide in 0.5 ml PBS containing < 0.1% sodium azide and 0.2% BSA).

Available as phycoerythrin (sc-6458 PE), fluorescein (sc-6458 FITC), PerCP (sc-6458 PerCP) or PerCP-Cy5.5 (sc-6458 PCPC5) conjugates for flow cytometry 100 tests.

## APPLICATIONS

VE-cadherin (C-19) 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), immunofluorescence (starting dilution 1:50, dilution range 1:50-1:500), flow cytometry (1 µg per 1 x $10^6$ cells) and solid phase ELISA (starting dilution 1:30, dilution range 1:30-1:3000).

VE-cadherin (C-19) is also recommended for detection of VE-cadherin in additional species, including equine, bovine and porcine.

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: rat placenta extract: sc-364808 or HUV-EC-C whole cell lysate: sc-364180.

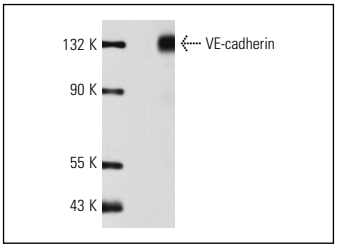
## RESEARCH USE

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

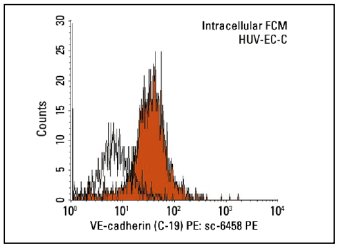
## STORAGE

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

## DATA




VE-cadherin (C-19): sc-6458. Western blot analysis of VE-cadherin expression in rat placenta extract.




VE-cadherin (C-19) PE: sc-6458 PE. Intracellular FCM analysis of methanol permeabilized HUV-EC-C cells. Solid black line histogram represents control goat IgG.

## SELECT PRODUCT CITATIONS


1. Tinsley, J.H., et al. 1999. Activated neutrophils induce hyperpermeability and phosphorylation of adherens junction proteins in coronary venular endothelial cells. J. Biol. Chem. 274: 24930-24934.
2. Hoang, M.V., et al. 2011. Active Rac1 improves pathologic VEGF neovessel architecture and reduces vascular leak: mechanistic similarities with angiopoietin-1. Blood 117: 1751-1760.
3. Youn, S.W., et al. 2011. COMP-Ang1 stimulates HIF-1α-mediated SDF-1 overexpression and recovers ischemic injury through BM-derived progenitor cell recruitment. Blood 117: 4376-4386.
4. Beard, R.S., et al. 2011. Hyperhomocysteinemia increases permeability of the blood-brain barrier by NMDA receptor-dependent regulation of adherens and tight junctions. Blood 118: 2007-2014.
5. Beard, R.S., et al. 2012. Metabotropic glutamate receptor 5 mediates phosphorylation of vascular endothelial cadherin and nuclear localization of β-catenin in response to homocysteine. Vascul. Pharmacol. 56: 159-167.
6. 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.
7. Huang, W., et al. 2012. HMGB1 increases permeability of the endothelial cell monolayer via RAGE and Src family tyrosine kinase pathways. Inflammation 35: 350-362.
8. Armstrong, S.M., et al. 2012. Influenza infects lung microvascular endothelium leading to microvascular leak: role of apoptosis and claudin-5. PLoS ONE 7: e47323.