

DESCRIPTION

Species Reactivity	Mouse
Specificity	Detects mouse VE-Cadherin in direct ELISAs and Western blots. In direct ELISAs, approximately 30% cross-reactivity with recombinant human VE-Cadherin is observed.
Source	Polyclonal Goat IgG
Purification	Antigen Affinity-purified
Immunogen	Mouse myeloma cell line NS0-derived recombinant mouse VE-Cadherin Asp46-Gln592 Accession # 2208309A
Endotoxin Level	<0.1 EU per 1 µg of the antibody by the LAL method.
Formulation	Lyophilized from a 0.2 µm filtered solution in PBS with Trehalose. See Certificate of Analysis for details.

APPLICATIONS

Please Note: Optimal dilutions should be determined by each laboratory for each application. *General Protocols* are available in the *Technical Information* section on our website.

	Recommended Concentration	Sample
Western Blot	0.1 µg/mL	Recombinant Mouse VE-Cadherin Fc Chimera (Catalog # 1002-VC)

PREPARATION AND STORAGE

Reconstitution	Reconstitute at 0.2 mg/mL in sterile PBS.
Shipping	The product is shipped at ambient temperature. Upon receipt, store it immediately at the temperature recommended below.
Stability & Storage	<p>Use a manual defrost freezer and avoid repeated freeze-thaw cycles.</p> <ul style="list-style-type: none"> ● 12 months from date of receipt, -20 to -70 °C as supplied. ● 1 month from date of receipt, 2 to 8 °C, reconstituted. ● 6 months from date of receipt, -20 to -70 °C, reconstituted.

BACKGROUND

The cadherin (Ca⁺⁺-dependent adherence) superfamily is a large group of membrane-associated glycoproteins that engage in homotypic, calcium-dependent, cell-cell adhesion events. The superfamily can be divided into at least five major subfamilies based on molecule gene structure, and/or extracellular (EC) and intracellular domains (1-4). Subfamilies include classical/type I, atypical/type II, and desmosomal-related cadherins (1-3). VE-Cadherin (vascular endothelial cadherin; also cadherin-5 and CD144) is a 125 kDa atypical/type II subfamily cadherin. Its subfamily classification is based principally on its genomic structure, as its physical structure is notably divergent from other type II subfamily members (2, 3). Mouse VE-Cadherin is synthesized as a 784 amino acid (aa) type I transmembrane (TM) preproprotein that contains a 24 aa signal peptide, a 21 aa prosequence, a 554 aa extracellular region (ECR), a 21 aa TM segment, and a 164 aa cytoplasmic domain (5, 6). The ECR contains five Ca⁺⁺-binding cadherin domains that are approximately 105 aa in length. Cadherin domains are comprised of two β-sheets that are oriented like bread in a sandwich. Although complex, the N-terminal cadherin domain mediates *trans* interactions, while the internal domains contribute to *cis* multimerizations (7). Mouse VE-Cadherin ECR is 92%, 77%, and 73% aa identical to rat, human and porcine VE-Cadherin ECR, respectively. VE-Cadherin is involved in the maintenance of endothelial permeability. In this regard, VE-Cadherin does not initiate new blood vessel formation; it maintains it once formed. Thus, when VE-Cadherin is downregulated, cells part and permeability increases (8). Notably, VEGF is known to promote vascular leakage, and apparently does so by inducing a β-arrestin-dependent endocytosis of VE-Cadherin (9). Part of this effect may be mediated by VE-Cadherin itself which is reported to increase the membrane half-life of VEGF R2 (10). VE-Cadherin acts homotypically at sites of zonula adherens. On each expressing cell, it is proposed that VE-Cadherin first forms a trimer, which then dimerizes with a trimeric counterpart *in-trans*. Alternatively, two *cis*-dimers could act *in-trans* to generate homotypic binding (11). In addition to cell adhesion, VE-Cadherin also is reported to mediate TGF-β receptor assembly. When clustered, VE-Cadherin enhances TβRII/TβRI assembly into an active receptor complex on endothelial cells (12). VE-Cadherin is expressed on endothelial cells, trophoblast cells, endothelial progenitor cells and embryonic hematopoietic cells (5, 8, 13, 14).

References:

1. Patel, S.D. *et al.* (2007) *Curr. Opin. Struct. Biol.* **13**:690.
2. Vestweber, D. (2008) *Arterioscler. Thromb. Vasc. Biol.* **28**:223.
3. Vincent, P.A. *et al.* (2004) *Am. J. Physiol. Cell. Physiol.* **286**:C987.
4. Cavallaro, U. *et al.* (2006) *Exp. Cell Res.* **312**:659.
5. Breier, G. *et al.* (1996) *Blood* **87**:630.
6. Huber, P. *et al.* (1996) *Genomics* **32**:21.
7. Pokutta, S. and W.I. Weis (2007) *Annu. Rev. Cell Dev. Biol.* **23**:237.
8. Crosby, C.V. *et al.* (2005) *Blood* **105**:2771.
9. Gavard, J. and J.S. Gutkind (2006) *Nat. Cell Biol.* **8**:1223.
10. Calera, M.R. *et al.* (2004) *Exp. Cell Res.* **300**:248.
11. Hewat, E.A. *et al.* (2007) *J. Mol. Biol.* **365**:744.
12. Rudini, N. *et al.* (2008) *EMBO J.* **27**:993.
13. Kogata, N. *et al.* (2006) *Circ. Res.* **98**:897.
14. Ema, M. *et al.* (2006) *Blood* **108**:4018.