

## CDH5

**Reactivity:** Human Mouse

**Tested applications:** WB IHC

**Recommended Dilution:** WB 1:500 - 1:1000 IHC 1:50 - 1:100

**Calculated MW:** 88kDa

**Observed MW:** Refer to Figures

**Immunogen:**

Recombinant protein of human CDH5

**Storage Buffer:**

Store at -20. Avoid freeze / thaw cycles. Buffer: PBS with 0.02% sodium azide, 50% glycerol, pH7.3.

**Synonym:**

CDH5; Cadherin-5; 7B4 antigen; VE-Cadherin; Vascular endothelial cadherin ;CD144;

**Catalog #:** A0734

**Antibody Type:**

Polyclonal Antibody

**Species:** Rabbit

**Gene ID:** 1003

**Isotype:** IgG

**Swiss Prot:** P33151

**Purity:** Affinity purification

For research use only.

**Background:**

, R-, B- and E-cadherins, as well as about ten other members that are found in adherens junctions, a cellular structure near the apical surface of polarized epithelial cells. The cytoplasmic domain of classical cadherins interacts with  $\beta$ -catenin,  $\gamma$ -catenin (also called plakoglobin), and p120 catenin.  $\beta$ -catenin and  $\gamma$ -catenin associate with  $\alpha$ -catenin, which links the cadherin-catenin complex to the actin cytoskeleton (1,2). While  $\beta$ - and  $\gamma$ -catenin play structural roles in the junctional complex, p120 regulates cadherin adhesive activity and trafficking (1-4). E-cadherin is considered an active suppressor of invasion and growth of many epithelial cancers (1-3). Recent studies indicate that cancer cells have up-regulated N-cadherin in addition to loss of E-cadherin. This change in cadherin expression is called the "cadherin switch". N-cadherin cooperates with the FGF receptor, leading to overexpression of MMP-9 and cellular invasion (3). In endothelial cells, VE-cadherin signaling, expression, and localization correlate with vascular permeability and tumor angiogenesis (5,6). Expression of P-cadherin, which is normally present in epithelial cells, is also altered in ovarian and other human cancers (7,8).

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