Revision 1

Store at -20C

#5296

E-Cadherin (32A8) Mouse mAb

Cell Signaling TECHNOLOGY®

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Applications:	Reactivity:	Sensitivity:	MW (kDa):	Source/Isotype:	UniProt ID:	Entrez-Gene Id:
W, IP	H	Endogenous	135	Mouse IgG1	#P12830	999

Product Usage Information

Application	Dilution
Western Blotting	1:1000
Immunoprecipitation	1:200

Storage

Supplied in 10 mM sodium HEPES (pH 7.5), 150 mM NaCl, 100 µg/ml BSA, 50% glycerol and less than 0.02% sodium azide. Store at –20°C. Do not aliquot the antibody.

Specificity/Sensitivity

E-Cadherin (32A8) Mouse mAb detects endogenous levels of total E-cadherin protein.

Source / Purification

Monoclonal antibody is produced by immunizing animals with human cancer cell lines. E-Cadherin (32A8) Mouse mAb recognizes an epitope in the extracellular region of E-cadherin.

Background

Cadherins are a superfamily of transmembrane glycoproteins that contain cadherin repeats of approximately 100 residues in their extracellular domain. Cadherins mediate calcium-dependent cell-cell adhesion and play critical roles in normal tissue development (1). The classic cadherin subfamily includes N-, P-, 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 β-catenin, γ-catenin (also called plakoglobin), and p120 catenin. β-catenin and γ-catenin associate with α-catenin, which links the cadherin-catenin complex to the actin cytoskeleton (1,2). While β- and γ-catenin play structural roles in the junctional complex, p120 regulates cadherin adhesive activity and trafficking (1-4). Investigators consider E-cadherin an active suppressor of invasion and growth of many epithelial cancers (1-3). Research studies indicate that cancer cells have upregulated 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). Research studies have shown that in endothelial cells, VE-cadherin signaling, expression, and localization correlate with vascular permeability and tumor angiogenesis (5,6). Investigators have also demonstrated that expression of P-cadherin, which is normally present in epithelial cells, is also altered in ovarian and other human cancers (7,8).

Background References

1. Wheelock, M.J. and Johnson, K.R. (2003) *Annu Rev Cell Dev Biol* 19, 207-35.
2. Christofori, G. (2003) *EMBO J* 22, 2318-23.
3. Hazan, R.B. et al. (2004) *Ann N Y Acad Sci* 1014, 155-63.
4. Bryant, D.M. and Stow, J.L. (2004) *Trends Cell Biol* 14, 427-34.
5. Rabascio, C. et al. (2004) *Cancer Res* 64, 4373-7.
6. Yamaoka-Tojo, M. et al. (2006) *Arterioscler Thromb Vasc Biol* 26, 1991-7.
7. Patel, I.S. et al. (2003) *Int J Cancer* 106, 172-7.
8. Sanders, D.S. et al. (2000) *J Pathol* 190, 526-30.

Species Reactivity

Species reactivity is determined by testing in at least one approved application (e.g., western blot).

Western Blot Buffer

IMPORTANT: For western blots, incubate membrane with diluted primary antibody in 5% w/v BSA, 1X TBS, 0.1% Tween® 20 at 4°C with gentle shaking, overnight.

Applications Key

W: Western Blotting **IP:** Immunoprecipitation

Cross-Reactivity Key

H: Human

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