

RCAS1 (D8K2E) Mouse mAb

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Applications: W, IP, IF-IC	Reactivity: H Mk	Sensitivity: Endogenous	MW (kDa): 32	Source/Isotype: Mouse IgG1	UniProt ID: #O00559	Entrez-Gene Id: 9166
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Product Usage Information**Application**

Western Blotting
Immunoprecipitation
Immunofluorescence (Immunocytochemistry)

Dilution

1:1000
1:50
1:50

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

RCAS1 (D8K2E) Mouse mAb recognizes endogenous levels of total RCAS1 protein.

Source / Purification

Monoclonal antibody is produced by immunizing animals with a synthetic peptide corresponding to residues surrounding Gly147 of human RCAS1 protein.

Background

Receptor binding cancer antigen expressed on SiSo cells (RCAS1) is also known as estrogen receptor-binding fragment-associated gene 9 (EBAG9). Originally identified as an estrogen-inducible gene (1), RCAS1 was recently found to play a novel role in the adaptive immune response by negatively regulating the cytolytic activity of cytotoxic T lymphocytes (CTLs) (2). RCAS1 is conserved in phylogeny and is ubiquitously expressed in most human tissues and cells (3,4). There is evidence that tissue expression of RCAS1 is increased in a variety of malignancies, including cancers of the gastrointestinal tract, liver, lung, breast, ovary, endometrium, and cervix. Research studies have shown that levels of RCAS1 tissue expression are negatively correlated with the prognosis of patients harboring the aforementioned malignancies (4). It is also noteworthy that research studies have detected elevated levels of RCAS1 in the sera of cancer patients (4). Initial studies indicated that RCAS1 was secreted from cancer cells and functioned as a ligand for a putative receptor expressed on NK cells, as well as T and B lymphocytes, inducing their apoptosis, which enabled cancer cells to evade immune surveillance (5,6). Subsequent studies have identified RCAS1 as a type III transmembrane Golgi protein with the ability to regulate vesicle formation, secretion, and protein glycosylation (2,7-9). Indeed, it has been shown that RCAS1 overexpression negatively regulates the cytolytic function of CTLs by negatively regulating protein trafficking from the *trans*-Golgi to secretory lysosomes (2). Furthermore, RCAS1 overexpression delays vesicle transport from the ER to Golgi and causes components of the ER quality control and glycosylation machinery to mislocalize. As a consequence, RCAS1 induces the deposition of tumor-associated glycan antigens on the cell surface, which are thought to contribute to tumor pathogenesis through the mediation of adhesion, invasion, and metastasis (8,9).

Background References

1. Watanabe, T. et al. (1998) *Mol Cell Biol* 18, 442-9.
2. Rüder, C. et al. (2009) *J Clin Invest* 119, 2184-203.
3. Tsuchiya, F. et al. (2001) *Biochem Biophys Res Commun* 284, 2-10.
4. Giaginis, C. et al. (2009) *Histol Histopathol* 24, 761-76.
5. Matsushima, T. et al. (2001) *Blood* 98, 313-21.
6. Nakashima, M. et al. (1999) *Nat Med* 5, 938-42.
7. Reimer, T.A. et al. (2005) *BMC Cancer* 5, 47.
8. Wolf, J. et al. (2010) *FASEB J* 24, 4000-19.
9. Engelsberg, A. et al. (2003) *J Biol Chem* 278, 22998-3007.

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 nonfat dry milk, 1X TBS, 0.1% Tween® 20 at 4°C with gentle shaking, overnight.

Applications Key

W: Western Blotting **IP:** Immunoprecipitation **IF-IC:** Immunofluorescence (Immunocytochemistry)

Cross-Reactivity Key

H: Human **Mk:** Monkey

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