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ECT2 Antibody (Center) Blocking Peptide

Synthetic peptide
Catalog # BP14755c

Specification

ECT2 Antibody (Center) Blocking Peptide - Product Information

Primary Accession Q9H8V3

ECT2 Antibody (Center) Blocking Peptide - Additional Information

Gene ID 1894

Other Names
Protein ECT2, Epithelial cell-transforming sequence 2 oncogene, ECT2

Format
Peptides are lyophilized in a solid powder format. Peptides can be reconstituted in solution using the appropriate buffer as needed.

Storage
Maintain refrigerated at 2-8°C for up to 6 months. For long term storage store at -20°C.

Precautions
This product is for research use only. Not for use in diagnostic or therapeutic procedures.

ECT2 Antibody (Center) Blocking Peptide - Protein Information

Name ECT2 (HGNC:3155)

Function
Guanine nucleotide exchange factor (GEF) that catalyzes the exchange of GDP for GTP. Promotes guanine nucleotide exchange on the Rho family members of small GTPases, like RHOA, RHOC, RAC1 and CDC42. Required for signal transduction pathways involved in the regulation of cytokinesis. Component of the centralspindlin complex that serves as a microtubule-dependent and Rho-mediated signaling required for the myosin contractile ring formation during the cell cycle cytokinesis. Regulates the translocation of RHOA from the central spindle to the equatorial region. Plays a role in the control of mitotic spindle assembly; regulates the activation of CDC42 in metaphase for the process of spindle fibers attachment to kinetochores before chromosome congression. Involved in the regulation of epithelial cell polarity; participates in the formation of epithelial tight junctions in a polarity complex PARD3-PARD6-protein kinase PRKCQ-dependent manner. Plays a role in the regulation of neurite outgrowth. Inhibits phenobarbital (PB)-induced NR1I3 nuclear translocation. Stimulates the activity of RAC1 through its association with the oncogenic PARD6A- PRKCI complex in cancer cells, thereby acting to coordinately drive tumor cell proliferation and invasion. Also stimulates genotoxic stress-induced RHOB activity in breast cancer cells leading to their cell death.

Cellular Location
Nucleus. Cytoplasm. Cytoplasm, cytoskeleton, spindle. Cleavage furrow. Midbody. Cell junction.

Cell junction, tight junction. Cytoplasm, cytoskeleton, microtubule organizing center, centrosome. Note=Sequestered within the nucleus during interphase (PubMed:10579713). Dispersed throughout the cytoplasm upon breakdown of the nuclear envelope during mitosis (PubMed:10579713). Colocalizes with the centralspindlin complex to the mitotic spindles during anaphase/metaphase, the cleavage furrow during telophase and at the midbody at the end of cytokinesis (PubMed:10579713). Colocalized with RhoA at the midbody (PubMed:10579713). Its subcellular localization to tight junction is increased by calcium (PubMed:15254234).

Tissue Location
Expressed in lung epithelial cells (at protein level). Expressed in squamous cell carcinoma, primary non-small cell lung cancer tumors and lung adenocarcinoma

ECT2 Antibody (Center) Blocking Peptide - Protocols

Provided below are standard protocols that you may find useful for product applications.

- Blocking Peptides

ECT2 Antibody (Center) Blocking Peptide - Images

ECT2 Antibody (Center) Blocking Peptide - Background

The protein encoded by this gene is a transforming proteinthat is related to Rho-specific exchange factors and yeast cellcycle regulators. The expression of this gene is elevated with theonset of DNA synthesis and remains elevated during G2 and M phases.In situ hybridization analysis showed that expression is at a highlevel in cells undergoing mitosis in regenerating liver. Thus, thisprotein is expressed in a cell cycle-dependent manner during liverregeneration, and is thought to have an important role in theregulation of cytokinesis.

ECT2 Antibody (Center) Blocking Peptide - References

Justilien, V., et al. Oncogene 28(41):3597-3607(2009)Burkard, M.E., et al. PLoS Biol. 7 (5), E1000111 (2009) :Wolfe, B.A., et al. PLoS Biol. 7 (5), E1000110 (2009) :Hirata, D., et al. Clin. Cancer Res. 15(1):256-266(2009)Seguin, L., et al. Mol. Cell. Biol. 29(2):570-581(2009)