

PREX1 (D8D2K) Rabbit mAb

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Applications: IHC-P	Reactivity: H	Sensitivity: Endogenous	MW (kDa): 190, 110	Source/Isotype: Rabbit IgG	UniProt ID: #Q8TCU6	Entrez-Gene Id: 57580
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Product Usage Information**Application**

Immunohistochemistry (Paraffin)

Dilution

1:150

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

PREX1 (D8D2K) Rabbit mAb recognizes endogenous levels of total PREX1 protein by immunohistochemistry. This antibody is not recommended for western blot due to weaker signal in comparison to other antibodies available against PREX1.

Source / Purification

Monoclonal antibody is produced by immunizing animals with a synthetic peptide corresponding to residues surrounding His770 of human PREX1 protein.

Background

Phosphoinositide-3,4,5-triphosphate (PtdIns(3,4,5)P₃)-dependent Rac exchanger 1 (PREX1) is a Rac-specific GTP-exchange factor (GEF) regulated by heterotrimeric G-protein β/γ subunits and the lipid second messenger PtdIns(3,4,5)P₃ (1-4). PREX1 contains two DEP (Dishevelled, Egl-10, and Pleckstrin homology) domains that coordinate heterotrimeric G-protein signaling. It also contains a Dbl-homology domain, which exhibits Rac-GEF activity, and PH and PDZ domains for interacting with upstream and downstream signaling components (1). Originally shown to modulate cellular migration of neutrophils by Rac2 activation (5-8), it is clear that PREX1 plays a broader role in modulating cell migration. PREX1 promotes metastasis of prostate cancer and melanoma cells, affects endothelial junction integrity, and is required for platelet generation and function (9-14). Research studies suggest that PREX1 plays an essential role in mediating ErbB-dependent signaling events in breast cancer by coordinating Rac activation in response to paracrine signals within the tumor microenvironment. Activation of PREX1 downstream of ErbB3 and EGFR chemokine receptors (CXCR4) promotes Rac activation, increased migration, proliferation, tumorigenesis, and metastasis in breast cancer cells (15,16). Consistent with this observation, deletion of PREX1 expression in mice results in resistance to melanoma metastasis (11). Expression of PREX1 in human tumors transplanted into mice inversely correlates with increased tumor progression and poor survival (15). Additional research studies suggest that PREX1 Rac-GEF activity is enhanced by phosphorylation in response to growth factors or hormones, and may require coincident dephosphorylation of two PH domain serine residues. The upstream kinases and precise regulatory mechanism remains elusive (15,17).

Background References

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Species Reactivity

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

Applications Key**IHC-P:** Immunohistochemistry (Paraffin)**Cross-Reactivity Key****H:** Human**Trademarks and Patents**

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