

Vinculin (E1E9V) XP[®] Rabbit mAb (HRP Conjugate)

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For Research Use Only. Not for Use in Diagnostic Procedures.

Applications:	Reactivity:	Sensitivity:	MW (kDa):	Source/Isotype:	UniProt ID:	Entrez-Gene Id:
W	H M R Mk	Endogenous	124	Rabbit IgG	#P18206-2	7414

Product Usage Information**Application**

Western Blotting

Dilution

1:1000

Storage

Supplied in 136 mM NaCl, 2.6 mM KCl, 12 mM sodium phosphate (pH 7.4) dibasic, 2 mg/ml BSA, and 50% glycerol. Store at -20°C. Do not aliquot the antibody.

Specificity/Sensitivity

Vinculin (E1E9V) XP[®] Rabbit mAb (HRP Conjugate) recognizes endogenous levels of total vinculin protein. This antibody also reacts with metavinculin, a 145 kDa splice variant of vinculin.

Source / Purification

Monoclonal antibody is produced by immunizing animals with recombinant protein specific to the amino terminus of human vinculin protein.

Description

This Cell Signaling Technology antibody is conjugated to the carbohydrate groups of horseradish peroxidase (HRP) via its amine groups. The HRP conjugated antibody is expected to exhibit the same species cross-reactivity as the unconjugated Vinculin (E1E9V) XP[®] Rabbit mAb #13901.

Background

Vinculin is a cytoskeletal protein that plays an important role in the regulation of focal adhesions and embryonic development (1-4). Three structural vinculin domains include an amino-terminal head, a short, flexible proline-rich region, and a carboxy-terminal tail (1). In the inactive state, the head and tail domains of vinculin interact to form a closed conformation. The open and active form of vinculin translocates to focal adhesions, where it is thought to be involved in anchoring F-actin to the membrane and regulation of cell migration (2). Phospholipid binding to the tail domain and subsequent phosphorylation of vinculin at Ser1033 and Ser1045 by PKC- α and Tyr100 and Tyr1065 by Src kinases weakens the head-tail interaction (5,6). This change in vinculin allows the binding of a number of other proteins, including talin, α -actinin, and paxillin, which disrupts the head-tail interaction and initiates the conformational change from the inactive to active state (2,4). Vinculin deficiencies are associated with a decrease in cell adhesion and an increase in cell motility, suggesting a possible role in metastatic growth (7,8). This is supported by a demonstrated relationship between decreased vinculin expression and increased carcinogenesis and metastasis in colorectal carcinoma (9).

Background References

1. Izard, T. et al. (2004) *Nature* 427, 171-5.
2. Humphries, J.D. et al. (2007) *J Cell Biol* 179, 1043-57.
3. Witt, S. et al. (2004) *J Biol Chem* 279, 31533-43.
4. Xu, W. et al. (1998) *Development* 125, 327-37.
5. Ziegler, W.H. et al. (2002) *J Biol Chem* 277, 7396-404.
6. Zhang, Z. et al. (2004) *Mol Biol Cell* 15, 4234-47.
7. Rodríguez Fernández, J.L. et al. (1993) *J Cell Biol* 122, 1285-94.
8. Samuels, M. et al. (1993) *J Cell Biol* 121, 909-21.
9. Yang, H.J. et al. (2010) *Cancer Invest* 28, 127-34.

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

Cross-Reactivity Key

H: Human **M:** Mouse **R:** Rat **Mk:** Monkey

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