

HOMER1 Antibody (N-term)

Purified Rabbit Polyclonal Antibody (Pab) Catalog # AP7302a

Specification

HOMER1 Antibody (N-term) - Product Information

Application WB, IHC-P, FC,E

Primary Accession <u>Q86YM7</u>

Other Accession <u>O9Z214</u>, <u>O9Z2Y3</u>, <u>O2KI56</u>

Reactivity Human

Predicted Bovine, Mouse, Rat

Host Rabbit
Clonality Polyclonal
Isotype Rabbit IgG
Calculated MW 40277
Antigen Region 96-122

HOMER1 Antibody (N-term) - Additional Information

Gene ID 9456

Other Names

Homer protein homolog 1, Homer-1, HOMER1, SYN47

Target/Specificity

This HOMER1 antibody is generated from rabbits immunized with a KLH conjugated synthetic peptide between 96-122 amino acids from the N-terminal region of human HOMER1.

Dilution

WB~~1:1000 IHC-P~~1:50~100 FC~~1:10~50

Format

Purified polyclonal antibody supplied in PBS with 0.09% (W/V) sodium azide. This antibody is prepared by Saturated Ammonium Sulfate (SAS) precipitation followed by dialysis against PBS.

Storage

Maintain refrigerated at 2-8°C for up to 2 weeks. For long term storage store at -20°C in small aliquots to prevent freeze-thaw cycles.

Precautions

HOMER1 Antibody (N-term) is for research use only and not for use in diagnostic or therapeutic procedures.

HOMER1 Antibody (N-term) - Protein Information

Name HOMER1 (HGNC:17512)



Function Postsynaptic density scaffolding protein. Binds and cross- links cytoplasmic regions of GRM1, GRM5, ITPR1, DNM3, RYR1, RYR2, SHANK1 and SHANK3. By physically linking GRM1 and GRM5 with ER- associated ITPR1 receptors, it aids the coupling of surface receptors to intracellular calcium release. May also couple GRM1 to PI3 kinase through its interaction with AGAP2. Isoform 1 regulates the trafficking and surface expression of GRM5. Isoform 3 acts as a natural dominant negative, in dynamic competition with constitutively expressed isoform 1 to regulate synaptic metabotropic glutamate function. Isoform 3, may be involved in the structural changes that occur at synapses during long-lasting neuronal plasticity and development. Forms a high-order complex with SHANK1, which in turn is necessary for the structural and functional integrity of dendritic spines (By similarity). Negatively regulates T cell activation by inhibiting the calcineurin-NFAT pathway. Acts by competing with calcineurin/PPP3CA for NFAT protein binding, hence preventing NFAT activation by PPP3CA (PubMed:18218901).

Cellular Location

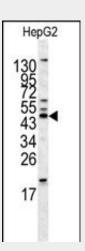
Cytoplasm. Postsynaptic density. Synapse. Cell projection, dendritic spine {ECO:0000250|UniProtKB:Q9Z214}. Note=Isoform 1 inhibits surface expression of GRM5 causing it to be retained in the endoplasmic reticulum.

HOMER1 Antibody (N-term) - Protocols

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

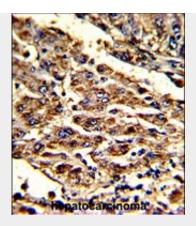
- Western Blot
- Blocking Peptides
- Dot Blot
- <u>Immunohistochemistry</u>
- <u>Immunofluorescence</u>
- Immunoprecipitation
- Flow Cytomety
- Cell Culture

HOMER1 Antibody (N-term) - Images

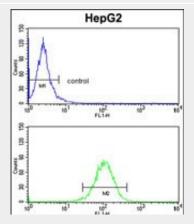


Western blot analysis of HOMER1 antibody (N-term) (Cat.#AP7302a) in HepG2 cell line lysates (35ug/lane). HOMER1 (arrow) was detected using the purified Pab.





Formalin-fixed and paraffin-embedded human hepatocarcinoma with HOMER1 Antibody (N-term), which was peroxidase-conjugated to the secondary antibody, followed by DAB staining. This data demonstrates the use of this antibody for immunohistochemistry; clinical relevance has not been evaluated.



HOMER1 Antibody (N-term) (Cat.#AP7302a) flow cytometry analysis of HepG2 cells (bottom histogram) compared to a negative control cell (top histogram). FITC-conjugated goat-anti-rabbit secondary antibodies were used for the analysis.

HOMER1 Antibody (N-term) - Background

HOMER1 is a member of the homer family of dendritic proteins. Members of this family regulate group 1 metabotrophic glutamate receptor function.

HOMER1 Antibody (N-term) - References

Sanna, S., Jackson, A.U. Nat. Genet. 40 (2), 198-203 (2008) Dahl, J.P., Kampman, K.M. Psychiatr. Genet. 15 (4), 277-283 (2005) Tu, J.C., Xiao, B. Neuron 23 (3), 583-592 (1999)