



CAP1 (M-30): sc-100917

BACKGROUND

Cyclase-associated proteins (CAPs) are a family of evolutionary conserved proteins that participate in signal transduction and function to regulate events associated with the Actin cytoskeleton. CAP1 and CAP2 (adenylate cyclase-associated protein 1 and 2, respectively) are two members of the CAP family, both of which localize to the cell membrane and contain one C-CAP/cofactor C-like domain. CAP1 is involved in the regulation of Actin filaments and is thought to mediate processes such as establishment of cell polarity and mRNA localization, while CAP2 has a bifunctional regulatory role and can interact directly with Actin. Although CAP1 is expressed throughout the body, CAP2 is predominately expressed in skin, brain, heart and skeletal muscle. Overexpression of CAP2 is associated with hepatocellular carcinoma, suggesting a possible role for CAP2 in carcinogenesis.

REFERENCES

- Matviiv, H., et al. 1992. Identification of a human cDNA encoding a protein that is structurally and functionally related to the yeast adenyl cyclase-associated CAP proteins. *Mol. Cell. Biol.* 12: 5033-5040.
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- Hubberstey, A., et al. 1996. Mammalian CAP interacts with CAP, CAP2, and Actin. *J. Cell. Biochem.* 61: 459-466.
- Moriyama, K. and Yahara, I. 2002. Human CAP1 is a key factor in the recycling of Cofilin and Actin for rapid Actin turnover. *J. Cell Sci.* 115: 1591-1601.
- Dodatko, T., et al. 2004. Crystal structure of the Actin binding domain of the cyclase-associated protein. *Biochemistry* 43: 10628-10641.
- Bertling, E., et al. 2004. Cyclase-associated protein 1 (CAP1) promotes Cofilin-induced Actin dynamics in mammalian nonmuscle cells. *Mol. Biol. Cell* 15: 2324-2334.
- Shibata, R., et al. 2006. Overexpression of cyclase-associated protein 2 in multistage hepatocarcinogenesis. *Clin. Cancer Res.* 12: 5363-5368.
- Peche, V., et al. 2007. CAP2, cyclase-associated protein 2, is a dual compartment protein. *Cell. Mol. Life Sci.* 64: 2702-2715.

CHROMOSOMAL LOCATION

Genetic locus: CAP1 (human) mapping to 1p34.2.

SOURCE

CAP1 (M-30) is a mouse monoclonal antibody raised against recombinant CAP1 of human origin.

PRODUCT

Each vial contains 100 µg IgG₁ kappa light chain in 1.0 ml of PBS with < 0.1% sodium azide and 0.1% gelatin.

RESEARCH USE

For research use only, not for use in diagnostic procedures.

APPLICATIONS

CAP1 (M-30) is recommended for detection of CAP1 of human origin by Western Blotting (starting dilution 1:200, dilution range 1:100-1:1000), immunoprecipitation [1-2 µg per 100-500 µg of total protein (1 ml of cell lysate)] and solid phase ELISA (starting dilution 1:30, dilution range 1:30-1:3000).

Suitable for use as control antibody for CAP1 siRNA (h): sc-88068, CAP1 shRNA Plasmid (h): sc-88068-SH and CAP1 shRNA (h) Lentiviral Particles: sc-88068-V.

Molecular Weight of CAP1: 52 kDa.

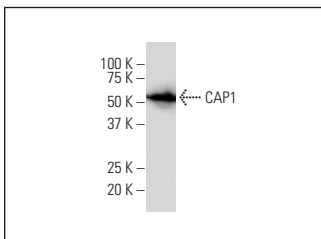
Positive Controls: HeLa whole cell lysate: sc-2200.

RECOMMENDED SUPPORT REAGENTS

To ensure optimal results, the following support reagents are recommended:

1) Western Blotting: use m-IgGκ BP-HRP: sc-516102 or m-IgGκ BP-HRP (Cruz Marker): sc-516102-CM (dilution range: 1:1000-1:10000), Cruz Marker™ Molecular Weight Standards: sc-2035, UltraCruz® Blocking Reagent: sc-516214 and Western Blotting Luminol Reagent: sc-2048. 2) Immunoprecipitation: use Protein A/G PLUS-Agarose: sc-2003 (0.5 ml agarose/2.0 ml).

DATA



CAP1 (M-30): sc-100917. Western blot analysis of CAP1 expression in HeLa whole cell lysate.

SELECT PRODUCT CITATIONS

- Izzi, B., et al. 2012. Methylation defect in imprinted genes detected in patients with an Albright's hereditary osteodystrophy like phenotype and platelet G_s hypofunction. *PLoS ONE* 7: e38579.
- Shin, D., et al. 2024. PCSK9 stimulates Syk, PKCδ, and NFκB, leading to atherosclerosis progression independently of LDL receptor. *Nat. Commun.* 15: 2789.

STORAGE

Store at 4° C, **DO NOT FREEZE**. Stable for one year from the date of shipment. Non-hazardous. No MSDS required.

PROTOCOLS

See our web site at www.scbt.com for detailed protocols and support products.