

## **Mouse Monoclonal Antibody to CTNNA1**

Purified Mouse Monoclonal Antibody Catalog # AO2373a

### **Specification**

### Mouse Monoclonal Antibody to CTNNA1 - Product Information

Application E, WB, IHC
Primary Accession P35221
Reactivity Human
Host Mouse
Clonality Monoclonal
Isotype Mouse IgG1
Calculated MW 100kDa KDa

**Description** 

CTNNA1 (Catenin (Cadherin-Associated Protein), Alpha 1, 102kDa) is a Protein Coding gene. Diseases associated with CTNNA1 include diffuse gastric cancer and acquired thrombocytopenia. Among its related pathways are Signaling by GPCR and Developmental Biology. GO annotations related to this gene include poly(A) RNA binding and actin filament binding. An important paralog of this gene is CTNNA3.;

#### **Immunogen**

Purified recombinant fragment of human CTNNA1 (AA: 371-574) expressed in E. Coli.

#### **Formulation**

Purified antibody in PBS with 0.05% sodium azide

#### **Application Note**

ELISA: 1/10000; WB: 1/500 - 1/2000; IHC: 1/200 - 1/1000;

### Mouse Monoclonal Antibody to CTNNA1 - Additional Information

**Gene ID 1495** 

**Other Names** 

CAP102

## **Storage**

Maintain refrigerated at 2-8°C for up to 6 months. For long term storage store at -20°C in small aliquots to prevent freeze-thaw cycles.

#### **Precautions**

Mouse Monoclonal Antibody to CTNNA1 is for research use only and not for use in diagnostic or therapeutic procedures.

#### Mouse Monoclonal Antibody to CTNNA1 - Protein Information

Name CTNNA1 (HGNC:2509)



#### **Function**

Associates with the cytoplasmic domain of a variety of cadherins. The association of catenins to cadherins produces a complex which is linked to the actin filament network, and which seems to be of primary importance for cadherins cell-adhesion properties. Can associate with both E- and N-cadherins. Originally believed to be a stable component of E-cadherin/catenin adhesion complexes and to mediate the linkage of cadherins to the actin cytoskeleton at adherens junctions. In contrast, cortical actin was found to be much more dynamic than E-cadherin/catenin complexes and CTNNA1 was shown not to bind to F-actin when assembled in the complex suggesting a different linkage between actin and adherens junctions components. The homodimeric form may regulate actin filament assembly and inhibit actin branching by competing with the Arp2/3 complex for binding to actin filaments. Involved in the regulation of WWTR1/TAZ, YAP1 and TGFB1- dependent SMAD2 and SMAD3 nuclear accumulation (By similarity). May play a crucial role in cell differentiation.

#### **Cellular Location**

Cytoplasm, cytoskeleton {ECO:0000250|UniProtKB:P26231}. Cell junction, adherens junction. Cell membrane {ECO:0000250|UniProtKB:P26231}; Peripheral membrane protein; Cytoplasmic side {ECO:0000250|UniProtKB:P26231}. Cell junction Cytoplasm {ECO:0000250|UniProtKB:Q9PVF8}. Nucleus. Note=Found at cell-cell boundaries and probably at cell-matrix boundaries. {ECO:0000250|UniProtKB:P26231}

#### **Tissue Location**

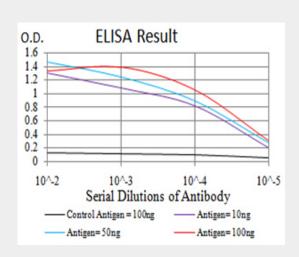
[Isoform 1]: Ubiquitously expressed in normal tissues.

### Mouse Monoclonal Antibody to CTNNA1 - Protocols

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

- Western Blot
- Blocking Peptides
- Dot Blot
- Immunohistochemistry
- Immunofluorescence
- Immunoprecipitation
- Flow Cytomety
- Cell Culture

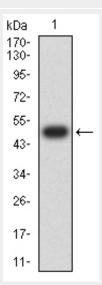
#### Mouse Monoclonal Antibody to CTNNA1 - Images



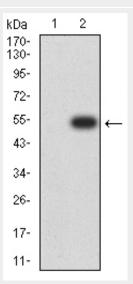
Black line: Control Antigen (100 ng); Purple line: Antigen (10ng); Blue line: Antigen (50 ng); Red



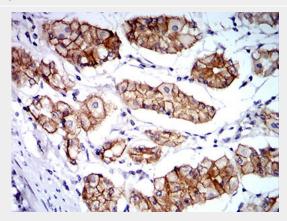
## line:Antigen (100 ng)



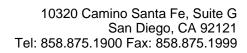
Western blot analysis using CTNNA1 mAb against human CTNNA1 (AA: 371-574) recombinant protein. (Expected MW is 48.8 kDa)



Western blot analysis using CTNNA1 mAb against HEK293 (1) and CTNNA1 (AA: 371-574)-hlgGFc transfected HEK293 (2) cell lysate.



Immunohistochemical analysis of paraffin-embedded stomach tissues using CTNNA1 mouse mAb with DAB staining.





# **Mouse Monoclonal Antibody to CTNNA1 - References**

1.Cell Cycle. 2014;13(15):2334-9.; 2.J Pathol. 2013 Mar;229(4):621-9.;