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Mouse Monoclonal Antibody to MIB1
Purified Mouse Monoclonal Antibody
Catalog # AO2429a

Specification

Mouse Monoclonal Antibody to MIB1 - Product Information

Application	**WB, FC, ICC, E**
Primary Accession	Q86YT6
Reactivity	**Human, Monkey**
Host	**Mouse**
Clonality	**Monoclonal**
Isotype	**Mouse IgG1**
Calculated MW	**110kDa KDa**

Description
This gene encodes a protein containing multiple ankyrin repeats and RING finger domains that functions as an E3 ubiquitin ligase. The encoded protein positively regulates Notch signaling by ubiquitinating the Notch receptors, thereby facilitating their endocytosis. This protein may also promote the ubiquitination and degradation of death-associated protein kinase 1 (DAPK1). ;

Immunogen
Purified recombinant fragment of human MIB1 (AA: 6-221) expressed in E. Coli.

Formulation
Purified antibody in PBS with 0.05% sodium azide

Application Note
ELISA: 1/10000; WB: 1/500 - 1/2000; ICC: 1/200 - 1/1000; FCM: 1/200 - 1/400

Mouse Monoclonal Antibody to MIB1 - Additional Information

Gene ID 57534

Other Names
MIB; DIP1; ZZZ6; DIP-1; LVNC7; ZZANK2

Dilution
WB~~1:1000
FC~~1:10~50
ICC~~N/A
E~~N/A

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 MIB1 is for research use only and not for use in diagnostic or therapeutic procedures.

Mouse Monoclonal Antibody to MIB1 - Protein Information

Name MIB1

Synonyms DIP1, KIAA1323, ZZANK2

Function
E3 ubiquitin-protein ligase that mediates ubiquitination of Delta receptors, which act as ligands of Notch proteins. Positively regulates the Delta-mediated Notch signaling by ubiquitinating the intracellular domain of Delta, leading to endocytosis of Delta receptors. Probably mediates ubiquitination and subsequent proteasomal degradation of DAPK1, thereby antagonizing anti-apoptotic effects of DAPK1 to promote TNF-induced apoptosis (By similarity). Involved in ubiquitination of centriolar satellite CEP131, CEP290 and PCM1 proteins and hence inhibits primary cilium formation in proliferating cells. Mediates 'Lys-63'-linked polyubiquitination of TBK1, which probably participates in kinase activation.

Cellular Location
Cytoplasm. Cytoplasm, cytoskeleton, microtubule organizing center, centrosome, centriolar satellite. Cell membrane. Note=Localizes to the plasma membrane (By similarity) According to PubMed:15048887, it is mitochondrial, however such localization remains unclear. Displaced from centriolar satellites in response to cellular stress, such as ultraviolet light (UV) radiation or heat shock.

Tissue Location
Widely expressed at low level. Expressed at higher level in spinal cord, ovary, whole brain, and all specific brain regions examined.

Mouse Monoclonal Antibody to MIB1 - 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 MIB1 - Images

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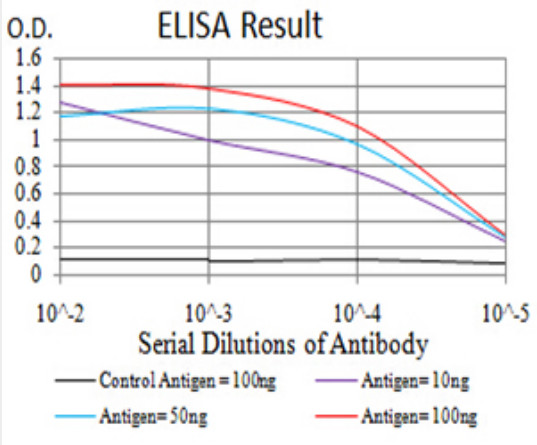
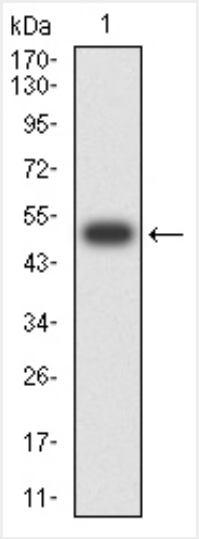
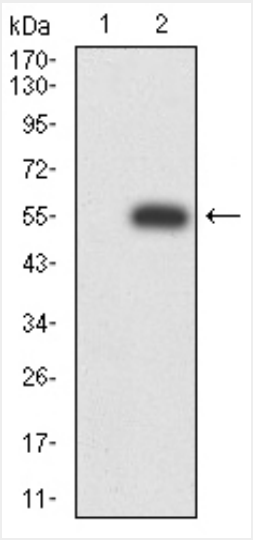


Black line: Control Antigen (100 ng);Purple line: Antigen (10ng); Blue line: Antigen (50 ng); Red line:Antigen (100 ng)



Western blot analysis using MIB1 mAb against human MIB1 (AA: 6-221) recombinant protein. (Expected MW is 50.1 kDa)



Western blot analysis using MIB1 mAb against HEK293 (1) and MIB1 (AA: 6-221)-hIgGFc transfected HEK293 (2) cell lysate.

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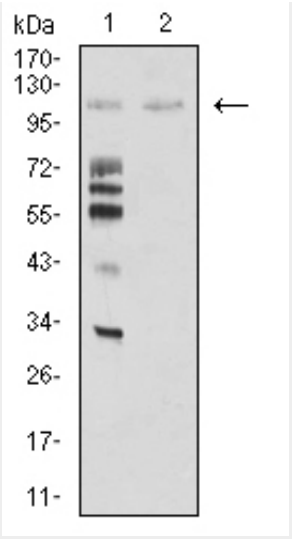
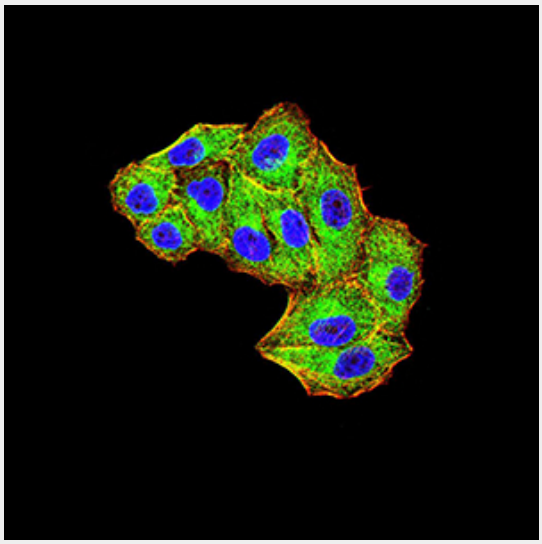
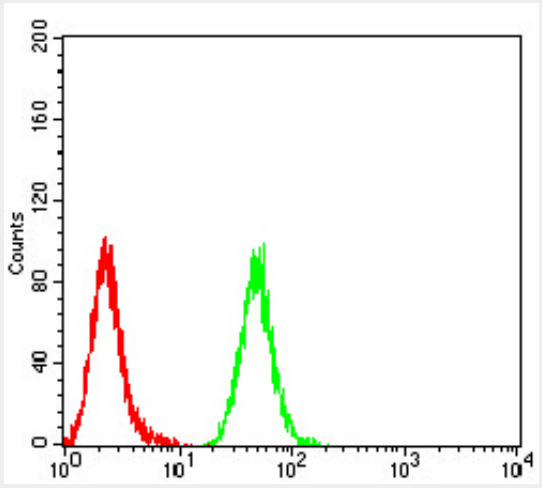


Western blot analysis using MIB1 mouse mAb against Hela (1) and COS7 (2) cell lysate.



Immunofluorescence analysis of Hela cells using MIB1 mouse mAb (green). Blue: DRAQ5 fluorescent DNA dye. Red: Actin filaments have been labeled with Alexa Fluor- 555 phalloidin. Secondary antibody from Fisher



Flow cytometric analysis of Hela cells using MIB1 mouse mAb (green) and negative control (red).

Mouse Monoclonal Antibody to MIB1 - References

1.J Cell Sci. 2015 May 1;128(9):1674-82. ; 2.Cell Res. 2012 Mar;22(3):603-6. ;