10320 Camino Santa Fe, Suite G
San Diego, CA 92121
Tel: 858.875.1900 Fax: 858.875.1999

MASTL Antibody

Affinity Purified Rabbit Polyclonal Antibody (Pab)
Catalog # AP7147d

Product Information

Application	WB, IHC-P, FC, E
Primary Accession	Q96GX5
Reactivity	Human
Host	Rabbit
Clonality	Polyclonal
Isotype	Rabbit IgG
Clone Names	RB22931
Calculated MW	97319

Additional Information

Gene ID	84930
Other Names	Serine/threonine-protein kinase greatwall, GW, GWL, hGWL, Microtubule-associated serine/threonine-protein kinase-like, MAST-L, MASTL, GW, GWL, THC2
Target/Specificity	This MASTL antibody is generated from rabbits immunized with human partial MASTL recombinant protein.
Dilution	WB~~1:1000 IHC-P~~1:100~500 FC~~1:10~50 E~~Use at an assay dependent concentration.
Format	Purified polyclonal antibody supplied in PBS with 0.09% (W/V) sodium azide. This antibody is purified through a protein A column, followed by peptide affinity purification.
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	MASTL Antibody is for research use only and not for use in diagnostic or therapeutic procedures.

Protein Information

Name	MASTL
Synonyms	GW, GWL, THC2
Function	Serine/threonine kinase that plays a key role in M phase by acting as a regulator of mitosis entry and maintenance (PubMed:19680222). Acts by

promoting the inactivation of protein phosphatase 2A (PP2A) during M phase: does not directly inhibit PP2A but acts by mediating phosphorylation and subsequent activation of ARPP19 and ENSA at 'Ser-62' and 'Ser-67', respectively (PubMed:38123684). ARPP19 and ENSA are phosphatase inhibitors that specifically inhibit the PPP2R2D (PR55-delta) subunit of PP2A. Inactivation of PP2A during M phase is essential to keep cyclin-B1-CDK1 activity high (PubMed:20818157). Following DNA damage, it is also involved in checkpoint recovery by being inhibited. Phosphorylates histone protein in vitro; however such activity is unsure in vivo. May be involved in megakaryocyte differentiation.

Cellular Location

Cytoplasm, cytoskeleton, microtubule organizing center, centrosome. Nucleus. Cleavage furrow. Note=During interphase is mainly nuclear, upon nuclear envelope breakdown localizes at the cytoplasm and during mitosis at the centrosomes. Upon mitotic exit moves to the cleavage furrow.

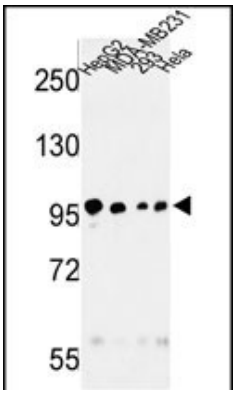
Background

MASTL, microtubule associated serine/threonine kinase-like, contains 1 protein kinase domain which belongs to the Ser/Thr protein kinase family. It may be involved in megakaryocyte differentiation. Defects in MASTL are a cause of nonsyndromic autosomal

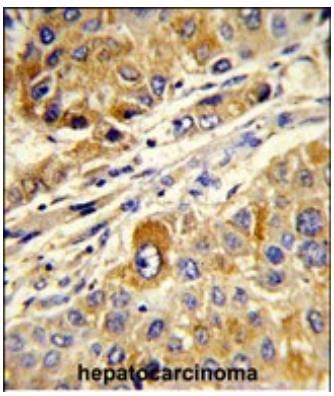
References

Gandhi, M.J., et al., Hum. Hered. 55(1):66-70 (2003).

Images

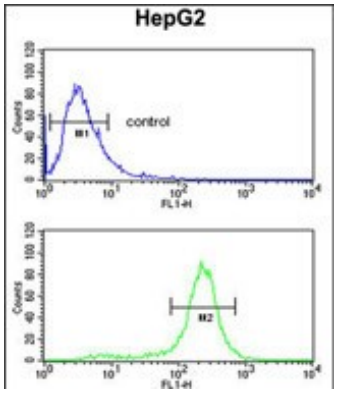


Western blot analysis of MASTL Antibody (Cat. #AP7147d) in HepG2, MDA-MB231, 293, Hela cell line lysates (35ug/lane). MASTL (arrow) was detected using the purified Pab.



Formalin-fixed and paraffin-embedded human hepatocarcinoma reacted with MASTL Antibody, 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.

MASTL Antibody (Cat. #AP7147d) flow cytometric 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.

Citations

- MKI-1, a Novel Small-Molecule Inhibitor of MASTL, Exerts Antitumor and Radiosensitizer Activities Through PP2A Activation in Breast Cancer
- Thrombocytopenia-associated mutations in Ser/Thr kinase MASTL deregulate actin cytoskeleton dynamics in platelets.
- MASTL inhibition promotes mitotic catastrophe through PP2A activation to inhibit cancer growth and radioresistance in breast cancer cells.
- Discovery and Characterization of a Novel MASTL Inhibitor MKI-2 Targeting MASTL-PP2A in Breast Cancer Cells and Oocytes

Please note: All products are 'FOR RESEARCH USE ONLY. NOT FOR USE IN DIAGNOSTIC OR THERAPEUTIC PROCEDURES'.