

Anti-MOB4A/MOB1B Antibody (6O289)

Product Details

Ig Type:	Mouse IgG2a
Reactivity:	Human
Conjugation:	Unconjugated
Clone:	6O289
Purification:	Protein A

Applications

Verified Activity:	Immunofluorescence staining of Human MOBKL1A in Hela cells. Cells were fixed with 4% PFA, permeabilized with 0.3% Triton X-100 in PBS, blocked with 10% serum, and incubated with mouse anti-Human MOBKL1A monoclonal antibody (1:60) at 4°C overnight. Then cells were stained with the Alexa Fluor® 488-conjugated Goat Anti-mouse IgG secondary antibody(green) and counterstained with DAPI(blue). Positive staining was localized to cytoplasm.
Application:	ICC/IF
Recommended	ICC-IF: 1:20-1:100

Properties

Stability & Storage:	Store at 2°C-8°C for 1 month. Store at -20°C or -80°C for 12 months. Avoid repeated freeze-thaw cycles. Preservative-Free.
Shipping:	Shipping with blue ice.

Antigen Details

Immunogen:	Recombinant Protein: Human MOB1B / MOBKL1A Protein (TMPY-03442)
Antigen Species:	Human
Synonyms:	MATS2;MOB kinase activator 1B;MOBKL1A;MOB4A

Research Background

MST1 and MST2 are the mammalian Ste2-related protein kinases most closely related to Drosophila Hippo, a major regulator of cell proliferation and survival during development. Overexpression of MST1 or MST2 in mammalian cells is proapoptotic. MST1 and MST2 activity increase during mitosis, especially in nocodazole-arrested mitotic cells, where these kinases exhibit an increase in both abundance and activation. MST1 and MST2 also can be activated nonphysiologically by okadaic acid or H₂O₂. The MOB1B and MOBKL1B polypeptides, homologs of the Drosophila MATS polypeptide, are identified as preferred MST1/MST2 substrates in vitro and are phosphorylated in cells in an MST1/MST2-dependent manner in mitosis and response to okadaic acid or H₂O₂. MST1/MST2-catalyzed MOB1B/MOBKL1B phosphorylation alters the ability of MOB1B/MOBKL1B to bind and regulate downstream targets such as the NDR-family protein kinases. Thus, MOB1B/MOBKL1B phosphorylation in cells promotes MOB1B/MOBKL1B binding to the LATS1 kinase and enables H₂O₂-stimulated LATS1 activation loop phosphorylation. Most importantly, the replacement of endogenous MOB1B/MOBKL1B by a non-phosphorylatable mutant is sufficient to accelerate cell proliferation substantially by speeding progression through G1/S as well as mitotic exit.

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