

DUSP16/MKP7 (D5F4) Rabbit mAb

Orders: 877-616-CELL (2355)
orders@cellsignal.com

Support: 877-678-TECH (8324)

Web: info@cellsignal.com
cellsignal.com

3 Trask Lane | Danvers | Massachusetts | 01923 | USA

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Applications:	Reactivity:	Sensitivity:	MW (kDa):	Source/Isotype:	UniProt ID:	Entrez-Gene Id:
W	H M R Mk	Endogenous	79	Rabbit IgG	#Q9BY84	80824

Product Usage Information**Application**

Western Blotting

Dilution

1:1000

Storage

Supplied in 10 mM sodium HEPES (pH 7.5), 150 mM NaCl, 100 µg/ml BSA, 50% glycerol and less than 0.02% sodium azide. Store at -20°C. Do not aliquot the antibody.

Specificity/Sensitivity

DUSP16/MKP7 (D5F4) Rabbit mAb recognizes endogenous levels of total DUSP16 protein.

Species predicted to react based on 100% sequence homology

Horse

Source / Purification

Monoclonal antibody is produced by immunizing animals with a synthetic peptide corresponding to residues surrounding Lys431 of human DUSP16 protein.

Background

MAP kinases are inactivated by dual-specificity protein phosphatases (DUSPs) that differ in their substrate specificity, tissue distribution, inducibility by extracellular stimuli, and cellular localization. DUSPs, also known as MAPK phosphatases (MKPs), specifically dephosphorylate both threonine and tyrosine residues in MAPK P-loops and have been shown to play important roles in regulating the function of the MAPK family (1,2). At least 13 members of the family (DUSP1-10, DUSP14, DUSP16, and DUSP22) display unique substrate specificities for various MAP kinases (3). MAPK phosphatases typically contain an amino-terminal rhodanese-fold responsible for DUSP docking to MAPK family members and a carboxy-terminal catalytic domain (4). These phosphatases can play important roles in development, immune system function, stress responses, and metabolic homeostasis (5). In addition, research studies have implicated DUSPs in the development of cancer and the response of cancer cells to chemotherapy (6).

DUSP16/MKP7 is a negative regulator of the JNK/SAPK family of stress-activated MAP kinases. It inhibits JNK-mediated signaling events by dephosphorylating threonine and tyrosine residues within the activation loop of JNK proteins, effectively preventing further activation of downstream effectors (7,8). DUSP16/MKP7 expression has been shown to be upregulated after oxidative stress, presumably as a means of suppressing JNK activity in order to return the cells to a homeostatic state (9). DUSP16 is normally turned over at a high-rate in most cells, but the stability of the protein can be enhanced by Erk1/2-mediated phosphorylation on Ser446, indicating that activation of mitogenic signaling pathways can suppress stress-response pathways via stabilization of a JNK phosphatase (10,11). Despite demonstrating a substrate preference towards JNK proteins, DUSP16/MKP7 has been shown to interact with other MAPK family members (Erk1/2, p38 MAPKs) as well as scaffolding proteins that may coordinate its activity and specificity (12,13).

DUSP16 is epigenetically silenced in Burkitt's lymphoma by increased methylation of the 5' regulatory regions of the gene (14). Methylation of the *DUSP16* gene and expression of DUSP16 protein inversely correlate with increased basal levels of JNK activity, suggesting DUSP16/MKP7 may play a critical role in maintaining JNK signaling in an "off" state in normal cells (14). More recently, DUSP16/MKP7 has been shown to play a crucial role in T helper (Th) cell differentiation into Th1 and Th2 cells, mediated by JNK signaling pathways (15). DUSP16/MKP7 expression is preferentially high in Th2 cells and low in Th1 cells during differentiation, resulting in either low (Th2) or high (Th1) JNK activity. This suggests that DUSP16 expression may be a regulator of Th cell balance (15).

Background References

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Species Reactivity

Species reactivity is determined by testing in at least one approved application (e.g., western blot).

Western Blot Buffer

IMPORTANT: For western blots, incubate membrane with diluted primary antibody in 5% w/v BSA, 1X TBS, 0.1% Tween® 20 at 4°C with gentle shaking, overnight.

Applications Key

W: Western Blotting

Cross-Reactivity Key

H: Human **M:** Mouse **R:** Rat **Mk:** Monkey

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