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Mouse Dyrk2 Antibody (C-term) Blocking peptide

Synthetic peptide
Catalog # BP14070b

Specification

Mouse Dyrk2 Antibody (C-term) Blocking peptide - Product Information

Primary Accession Q5U4C9

Mouse Dyrk2 Antibody (C-term) Blocking peptide - Additional Information

Gene ID 69181

Other Names
Dual specificity tyrosine-phosphorylation-regulated kinase 2, Dyrk2 {ECO:0000312|MGI:MGI:1330301}

Target/Specificity
The synthetic peptide sequence used to generate the antibody AP14070b was selected from the C-term region of Mouse Dyrk2. A 10 to 100 fold molar excess to antibody is recommended. Precise conditions should be optimized for a particular assay.

Format
Peptides are lyophilized in a solid powder format. Peptides can be reconstituted in solution using the appropriate buffer as needed.

Storage
Maintain refrigerated at 2-8°C for up to 6 months. For long term storage store at -20°C.

Precautions
This product is for research use only. Not for use in diagnostic or therapeutic procedures.

Mouse Dyrk2 Antibody (C-term) Blocking peptide - Protein Information

Name Dyrk2 {ECO:0000312|MGI:MGI:1330301}

Function
Serine/threonine-protein kinase involved in the regulation of the mitotic cell cycle, cell proliferation, apoptosis, organization of the cytoskeleton and neurite outgrowth. Functions in part via its role in ubiquitin-dependent proteasomal protein degradation. Functions downstream of ATM and phosphorylates p53/TP53 at 'Ser-46', and thereby contributes to the induction of apoptosis in response to DNA damage. Phosphorylates NFATC1, and thereby inhibits its accumulation in the nucleus and its transcription factor activity. Phosphorylates EIF2B5 at 'Ser-544', enabling its subsequent phosphorylation and inhibition by GSK3B. Likewise, phosphorylation of NFATC1, CRMP2/DPYSL2 and CRMP4/DPYSL3 promotes their subsequent phosphorylation by GSK3B. May play a general role in the priming of GSK3 substrates. Inactivates GYS1 by phosphorylation at 'Ser-641', and potentially also a second phosphorylation site, thus regulating glycogen synthesis. Mediates EDVP E3 ligase complex formation and is required for the phosphorylation and subsequent degradation of KATNA1. Phosphorylates TERT at 'Ser-457', promoting TERT

ubiquitination by the EDVP complex. Phosphorylates SIAH2, and thereby increases its ubiquitin ligase activity. Promotes the proteasomal degradation of MYC and JUN, and thereby regulates progress through the mitotic cell cycle and cell proliferation. Promotes proteasomal degradation of GLI2 and GLI3, and thereby plays a role in smoothened and sonic hedgehog signaling. Phosphorylates CRMP2/DPYSL2, CRMP4/DPYSL3, DCX, EIF2B5, EIF4EBP1, GLI2, GLI3, GYS1, JUN, MDM2, MYC, NFATC1, p53/TP53, TAU/MAPT and KATNA1. Can phosphorylate histone H1, histone H3 and histone H2B (in vitro). Can phosphorylate CARHSP1 (in vitro) (By similarity). Plays a role in cytoskeleton organization and neurite outgrowth via its phosphorylation of DCX.

Cellular Location
Cytoplasm. Nucleus {ECO:0000250|UniProtKB:Q92630}. Note=Translocates into the nucleus following DNA damage. {ECO:0000250|UniProtKB:Q92630}

Mouse Dyrk2 Antibody (C-term) Blocking peptide - Protocols

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

- Blocking Peptides

Mouse Dyrk2 Antibody (C-term) Blocking peptide - Images

Mouse Dyrk2 Antibody (C-term) Blocking peptide - Background

Role in the regulation of cellular growth and/or development. Regulates TP53 by phosphorylation on Ser-46 to induce apoptosis in response to DNA damage, functioning downstream of ATM. Inactivates GYS1 by phosphorylation at Ser-641, and potentially also a second phosphorylation site, thus regulating glycogen synthesis. Phosphorylates EIF2B5 at Ser-544, enabling its subsequent phosphorylation and inhibition by GSK3, and may play a more general role in the priming of GSK3 substrates (By similarity).

Mouse Dyrk2 Antibody (C-term) Blocking peptide - References

Guo, X., et al. J. Biol. Chem. 285(17):13223-13232(2010)Kudo, L.C., et al. Cereb. Cortex 17(9):2108-2122(2007)Blackshaw, S., et al. PLoS Biol. 2 (9), E247 (2004) :Clark, A.G., et al. Science 302(5652):1960-1963(2003)Geiger, J.N., et al. Blood 97(4):901-910(2001)