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DYRK1B Antibody (C-term)

Purified Rabbit Polyclonal Antibody (Pab)
Catalog # AP7538b

Product Information

Application	WB, IHC-P, E
Primary Accession	Q9Y463
Reactivity	Human, Mouse
Predicted	Rat, Bovine, Canine, Rabbit
Host	Rabbit
Clonality	Polyclonal
Isotype	Rabbit IgG
Calculated MW	69198
Antigen Region	561-589

Additional Information

Gene ID	9149
Other Names	Dual specificity tyrosine-phosphorylation-regulated kinase 1B, Minibrain-related kinase, Mirk protein kinase, DYRK1B, MIRK
Target/Specificity	This DYRK1B antibody is generated from rabbits immunized with a KLH conjugated synthetic peptide between 561-589 amino acids from the C-terminal region of human DYRK1B.
Dilution	WB~~1:1000 IHC-P~~1:100~500 E~~Use at an assay dependent concentration.
Format	Purified polyclonal antibody supplied in PBS with 0.09% (W/V) sodium azide. This antibody is prepared by Saturated Ammonium Sulfate (SAS) precipitation followed by dialysis against PBS.
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	DYRK1B Antibody (C-term) is for research use only and not for use in diagnostic or therapeutic procedures.

Protein Information

Name	DYRK1B
Synonyms	MIRK
Function	Dual-specificity kinase which possesses both serine/threonine and tyrosine kinase activities. Plays an essential role in ribosomal DNA (rDNA)

	double-strand break repair and rDNA copy number maintenance (PubMed:33469661). During DNA damage, mediates transcription silencing in part via phosphorylating and enforcing DSB accumulation of the histone methyltransferase EHMT2 (PubMed:32611815). Enhances the transcriptional activity of TCF1/HNF1A and FOXO1. Inhibits epithelial cell migration. Mediates colon carcinoma cell survival in mitogen-poor environments. Inhibits the SHH and WNT1 pathways, thereby enhancing adipogenesis. In addition, promotes expression of the gluconeogenic enzyme glucose-6-phosphatase catalytic subunit 1 (G6PC1).
Cellular Location	Nucleus. Nucleus, nucleolus. Chromosome. Note=Localizes to sites of double-strand breaks (DSBs) following DNA damage.
Tissue Location	Highest expression in skeletal muscle, testis, heart and brain with little expression in colon or lung. Expressed in a variety of tumor cell lines.

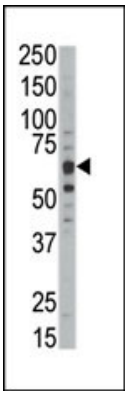
Background

DYRK1B is a member of the DYRK family of protein kinases. DYRK1B contains a bipartite nuclear localization signal and is found mainly in muscle and testis. The protein is proposed to be involved in the regulation of nuclear functions. Three isoforms of DYRK1B have been identified differing in the presence of two alternatively spliced exons within the catalytic domain.

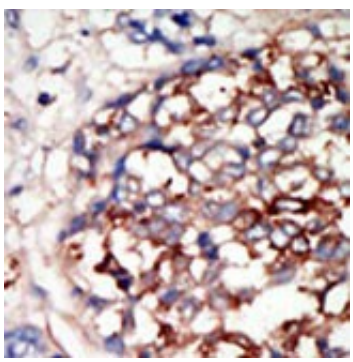
References

Lim, S., et al., J. Biol. Chem. 277(51):49438-49445 (2002).
Lim, S., et al., J. Biol. Chem. 277(28):25040-25046 (2002).
Lee, K., et al., Cancer Res. 60(13):3631-3637 (2000).
Leder, S., et al., Biochem. Biophys. Res. Commun. 254(2):474-479 (1999).

Images



Western blot analysis of anti-DYRKB Pab (Cat. #AP7538b) in mouse kidney tissue lysate. DYRKB (Arrow) was detected using purified Pab. Secondary HRP-anti-rabbit was used for signal visualization with chemiluminescence.



Formalin-fixed and paraffin-embedded human cancer tissue reacted with the primary 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. BC = breast carcinoma; HC = hepatocarcinoma.

Citations

- Negative feedback Inhibition of NFATc1 by DYRK1A regulates bone homeostasis.
- Involvement of GSK-3beta and DYRK1B in differentiation-inducing factor-3-induced phosphorylation of cyclin D1 in HeLa cells.
- The involvement of FoxO in cell survival and chemosensitivity mediated by Mirk/Dyrk1B in ovarian cancer.
- Mirk/Dyrk1B mediates G0/G1 to S phase cell cycle progression and cell survival involving MAPK/ERK signaling in human cancer cells.

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