

Anti-p53R2 Antibody (1K473)

Product Details

Ig Type:	Rabbit IgG
Reactivity:	Human
Conjugation:	Unconjugated
Clone:	1K473
Purification:	Protein A

Applications

Verified Activity:	1. Immunochemical staining of human RRM2B in human tonsil with rabbit monoclonal antibody (1:200, formalin-fixed paraffin embedded sections).
	2. Immunochemical staining of human RRM2B in human squamous cancer with rabbit monoclonal antibody (1:200, formalin-fixed paraffin embedded sections).
	3. Immunochemical staining of human RRM2B in human skeletal muscle with rabbit monoclonal antibody (1:200, formalin-fixed paraffin embedded sections).
	4. Immunofluorescence staining of RRM2B in Hela cells. Cells were fixed with 4% PFA, permeabilized with 0.1% Triton X-100 in PBS, blocked with 10% serum, and incubated with rabbit anti-Y RRM2B monoclonal antibody (dilution ratio 1:60) at 4°C overnight. Then cells were stained with the Alexa Fluor®488-conjugated Goat Anti-rabbit IgG secondary antibody (green). Positive staining was localized to Cytoplasm and Nucleus.
Application:	ICC/IF,IHC-P
Recommended	IHC-P: 1:100-1:500; 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 RRM2B / P53R2 protein (TMPY-02527)
Antigen Species:	Human
Synonyms:	MTDPS8A;P53R2;MTDPS8B;ribonucleotide reductase M2 B (TP53 inducible)

Research Background

Ribonucleoside reductase subunit M2B, also known as RRM2B or p53R2, is an enzyme belonging to the iron-dependent ribonucleotide reductase (RNR) enzyme family which is essential for DNA synthesis. Ribonucleotide reductase (RNR) is an enzyme that catalyzes the formation of deoxyribonucleotides from ribonucleotides and plays a critical role in regulating the total rate of DNA synthesis so that DNA to cell mass is maintained at a constant ratio during cell division and DNA repair. RRM2B is a phosphorylated protein. It is hypothesized that RRM2B activity can be regulated at the posttranslational level in response to DNA damage. RRM2B has previously been shown to be essential for the maintenance of mtDNA copy number and its candidacy for tumor suppression has been evaluated in several mutational analyses of different cancer types. However, the contribution of RRM2B to the DNA damage

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response has been questioned because its transcriptional induction upon DNA damage is not rapid enough for prompt DNA repair. Instead, ATM-mediated phosphorylation has been suggested to regulate the DNA repair activity of RRM2B posttranslationally. Besides, a defect in RRM2B can induce a mild muscle disease of adult onset through disturbance of mitochondrial homeostasis but that this defect does not appear to be oncogenic.

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