Data Sheet (Cat.No.TMAY-02507)

Anti-p53R2 Antibody (4V66)

Product Details

Ig Type:	Mouse IgG1
Reactivity:	Human
Conjugation:	Unconjugated
Clone:	4V66
Purification:	Protein A

Applications

Verified Activity:	1. Anti-RRM2B mouse monoclonal antibody at 1:500 dilution. -Lane A: MCF7 Whole Cell lysate. -Lysates/proteins at 30 µg per lane. -Secondary -Goat Anti-Mouse IgG H&L (Dylight800) at 1/15000 dilution. -Developed using the Odyssey technique. -Performed under reducing conditions. -Predicted band size:40 kDa. -Observed band size:40 kDa. 2. Anti-RRM2B mouse monoclonal antibody at 1:500 dilution. -Lane A: 293T Whole Cell Lysate. -Lane B: RRM2B knockout 293T Whole Cell lysate. -Lysates/proteins at 30 µg per lane. -Secondary -Goat Anti-Mouse IgG (H+L)/HRP at 1/10000 dilution. -Developed using the ECL technique. -Performed under reducing conditions. -Predicted band size:40 kDa. -Observed band size:39 kDa
Application:	ELISA,ELISA(Det),WB
Recommended	WB: 1:500-1:2000; ELISA: 1:5000-1:10000; ELISA(Det): 1:5000-1:50000

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:	MTDPS8B;ribonucleotide reductase M2 B (TP53 inducible);MTDPS8A;P53R2

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 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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