Recombinant Human RRM2B/P53R2 Protein (His Tag)

Catalog Number:PKSH030709

Note: Centrifuge before opening to ensure complete recovery of vial contents.

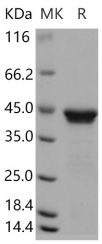
Description

Synonyms	MTDPS8A;MTDPS8B;P53R2
Species	Human
Expression Host	E.coli
Sequence	Met 1-Phe 351
Accession	Q7LG56-1
Calculated Molecular Weight	42.6 kDa
Observed molecular weight	43 kDa
Tag	N-His

Properties

Purity	> 92 % as determined by reducing SDS-PAGE.
Endotoxin	Please contact us for more information.
Storage	Generally, lyophilized proteins are stable for up to 12 months when stored at -20 to -80℃. Reconstituted protein solution can be stored at 4-8℃ for 2-7 days. Aliquots of reconstituted samples are stable at < -20℃ for 3 months.
Shipping	This product is provided as lyophilized powder which is shipped with ice packs.
Formulation	Lyophilized from sterile PBS, 30% glycerol, pH 8.5 Normally 5 % - 8 % trehalose, mannitol and 0.01% Tween80 are added as protectants before lyophilization. Please refer to the specific buffer information in the printed manual.
Reconstitution	Please refer to the printed manual for detailed information.

Data



> 92 % as determined by reducing SDS-PAGE.

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

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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. In addition, 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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