Data Sheet (Cat.No.TMPY-02527)

TargetMol

p53R2 Protein, Human, Recombinant (His)

General Information

Synonyms:	P53R2;MTDPS8A;MTDPS8B;ribonucleotide reductase M2 B (TP53 inducible)
Protein Construction:	A DNA sequence encoding the human RRM2B (Q7LG56-1) (Met 1-Phe 351) was expressed, with a polyhistide tag at the N-terminus. Predicted N terminal: Met
Species:	Human
Expression Host:	E. coli
Accession:	Q7LG56-1
Molecular Weight:	42.6 kDa (predicted); 43 kDa (reducing conditions)

QC Testing

Biological Activity:	Activity testing is in progress. It is theoretically active, but we cannot guarantee it. If you require protein activity, we recommend choosing the eukaryotic expression version first.
Purity:	> 92 % as determined by SDS-PAGE
Endotoxin:	Please contact us for more information.
Formulation:	Supplied as sterile PBS, 30% glycerol, pH 8.5.

Preparation and Storage

Reconstitution:

A Certificate of Analysis (CoA) containing reconstitution instructions is included with the products. Please refer to the CoA for detailed information.

Stability & Storage:

It is recommended to store the product under sterile conditions at -20°C to -80°C. Samples are stable for up to 12 months. Please avoid multiple freeze-thaw cycles and store products in aliquots.

Actual storage temperature shall be subject to the COA.

Shipping:

Proteins are shipped with blue ice.

Protein 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.

Reference

Bourdon A, et al. (2007) Mutation of RRM2B, encoding p53-controlled ribonucleotide reductase (p53R2), causes severe mitochondrial DNA depletion. Nature Genetics. 39: 776-80.

Tyynismaa H, et al. (2009) A Heterozygous Truncating Mutation in RRM2B Causes Autosomal-Dominant Progressive External Ophthalmoplegia with Multiple mtDNA Deletions. AJHG. 85 (2) : 290-5.

Shaibani A, et al. (2009) Mitochondrial neurogastrointestinal encephalopathy due to mutations in RRM2B. Arch Neurol.66 (8): 1028-32.