

DDX17 Antibody (N-term) Blocking Peptide
Synthetic peptide
Catalog # BP9322a**Specification**

DDX17 Antibody (N-term) Blocking Peptide - Product InformationPrimary Accession [O92841](#)**DDX17 Antibody (N-term) Blocking Peptide - Additional Information**

Gene ID 10521

Other Names

Probable ATP-dependent RNA helicase DDX17, DEAD box protein 17, DEAD box protein p72, RNA-dependent helicase p72, DDX17

Format

Peptides are lyophilized in a solid powder format. Peptides can be reconstituted in solution using the appropriate buffer as needed.

Storage

Maintain refrigerated at 2-8°C for up to 6 months. For long term storage store at -20°C.

Precautions

This product is for research use only. Not for use in diagnostic or therapeutic procedures.

DDX17 Antibody (N-term) Blocking Peptide - Protein Information

Name DDX17

Function

As an RNA helicase, unwinds RNA and alters RNA structures through ATP binding and hydrolysis. Involved in multiple cellular processes, including pre-mRNA splicing, alternative splicing, ribosomal RNA processing and miRNA processing, as well as transcription regulation. Regulates the alternative splicing of exons exhibiting specific features (PubMed:12138182, PubMed:22266867, PubMed:23022728, PubMed:24910439). For instance, promotes the inclusion of AC-rich alternative exons in CD44 transcripts (PubMed:12138182). This function requires the RNA helicase activity (PubMed:12138182, PubMed:22266867, PubMed:23022728, PubMed:24910439). Affects NFAT5 and histone macro- H2A.1/MACROH2A1 alternative splicing in a CDK9-dependent manner (PubMed:22266867, PubMed:22266867, PubMed:22266867).

[26209609](http://www.uniprot.org/citations/26209609)). In NFAT5, promotes the introduction of alternative exon 4, which contains 2 stop codons and may target NFAT5 exon 4-containing transcripts to nonsense-mediated mRNA decay, leading to the down-regulation of NFAT5 protein (PubMed:[22266867](http://www.uniprot.org/citations/22266867)). Affects splicing of mediators of steroid hormone signaling pathway, including kinases that phosphorylates ESR1, such as CDK2, MAPK1 and GSK3B, and transcriptional regulators, such as CREBBP, MED1, NCOR1 and NCOR2. By affecting GSK3B splicing, participates in ESR1 and AR stabilization (PubMed:[24275493](http://www.uniprot.org/citations/24275493)). In myoblasts and epithelial cells, cooperates with HNRNPH1 to control the splicing of specific subsets of exons (PubMed:[24910439](http://www.uniprot.org/citations/24910439)). In addition to binding mature mRNAs, also interacts with certain pri-microRNAs, including MIR663/miR-663a, MIR99B/miR-99b, and MIR6087/miR-6087 (PubMed:[25126784](http://www.uniprot.org/citations/25126784)). Binds pri-microRNAs on the 3' segment flanking the stem loop via the 5'- [ACG]CAUC[ACU]-3' consensus sequence (PubMed:[24581491](http://www.uniprot.org/citations/24581491)). Required for the production of subsets of microRNAs, including MIR21 and MIR125B1 (PubMed:[24581491](http://www.uniprot.org/citations/24581491), PubMed:[27478153](http://www.uniprot.org/citations/27478153)). May be involved not only in microRNA primary transcript processing, but also stabilization (By similarity). Participates in MYC down-regulation at high cell density through the production of MYC-targeting microRNAs (PubMed:[24581491](http://www.uniprot.org/citations/24581491)). Along with DDX5, may be involved in the processing of the 32S intermediate into the mature 28S ribosomal RNA (PubMed:[17485482](http://www.uniprot.org/citations/17485482)). Promoter-specific transcription regulator, functioning as a coactivator or corepressor depending on the context of the promoter and the transcriptional complex in which it exists (PubMed:[15298701](http://www.uniprot.org/citations/15298701)). Enhances NFAT5 transcriptional activity (PubMed:[22266867](http://www.uniprot.org/citations/22266867)). Synergizes with TP53 in the activation of the MDM2 promoter; this activity requires acetylation on lysine residues (PubMed:[17226766](http://www.uniprot.org/citations/17226766), PubMed:[19995069](http://www.uniprot.org/citations/19995069), PubMed:[20663877](http://www.uniprot.org/citations/20663877)). May also coactivate MDM2 transcription through a TP53-independent pathway (PubMed:[17226766](http://www.uniprot.org/citations/17226766)). Coactivates MMP7 transcription (PubMed:[17226766](http://www.uniprot.org/citations/17226766)). Along with CTNNB1, coactivates MYC, JUN, FOSL1 and cyclin D1/CCND1 transcription (PubMed:[17699760](http://www.uniprot.org/citations/17699760)). Alone or in combination with DDX5 and/or SRA1 non-coding RNA, plays a critical role in promoting the assembly of proteins required for the formation of the transcription initiation complex and chromatin remodeling leading to coactivation of MYOD1-dependent transcription. This helicase-independent activity is required for skeletal muscle cells to properly differentiate into myotubes (PubMed:[17011493](http://www.uniprot.org/citations/17011493), PubMed:[24910439](http://www.uniprot.org/citations/24910439)). During epithelial-to-mesenchymal transition, coregulates SMAD-dependent transcriptional activity, directly controlling key effectors of differentiation, including miRNAs which in turn directly repress its expression (PubMed:[24910439](http://www.uniprot.org/citations/24910439)). Plays a role in estrogen and testosterone signaling pathway at several levels. Mediates the use of alternative promoters in estrogen-responsive genes and regulates transcription and splicing of a large number of steroid hormone target genes (PubMed:[19995069](http://www.uniprot.org/citations/19995069), PubMed:[20406972](http://www.uniprot.org/citations/20406972), PubMed:[20663877](http://www.uniprot.org/citations/20663877), PubMed:[24275493](http://www.uniprot.org/citations/24275493)). Contrary to splicing regulation activity, transcriptional

coregulation of the estrogen receptor ESR1 is helicase-independent (PubMed:19718048, PubMed:24275493). Plays a role in innate immunity. Specifically restricts bunyavirus infection, including Rift Valley fever virus (RVFV) or La Crosse virus (LACV), but not vesicular stomatitis virus (VSV), in an interferon- and DROSHA- independent manner (PubMed:25126784). Binds to RVFV RNA, likely via structured viral RNA elements (PubMed:25126784). Promotes mRNA degradation mediated by the antiviral zinc-finger protein ZC3HAV1, in an ATPase-dependent manner (PubMed:18334637).

Cellular Location

Nucleus. Nucleus, nucleolus. Cytoplasm, cytosol. Note=In the course of bunyavirus infection, relocalizes from the nucleus to the cytosol where it binds viral RNA to antagonize replication.

Tissue Location

Widely expressed (PubMed:8871553). Low expression, if any, in normal colonic epithelial cells (at protein level). Levels tend to increase during colon cancer progression, from very low in benign hyperplastic polyps to very high in tubular and villous adenomas (PubMed:17699760).

DDX17 Antibody (N-term) Blocking Peptide - Protocols

Provided below are standard protocols that you may find useful for product applications.

- [Blocking Peptides](#)

DDX17 Antibody (N-term) Blocking Peptide - Images

DDX17 Antibody (N-term) Blocking Peptide - Background

DDX17 box proteins, characterized by the conserved motif Asp-Glu-Ala-Asp (DEAD), are putative RNA helicases. They are implicated in a number of cellular processes involving alteration of RNA secondary structure, such as translation initiation, nuclear and mitochondrial splicing, and ribosome and spliceosome assembly. Based on their distribution patterns, some members of this family are believed to be involved in embryogenesis, spermatogenesis, and cellular growth and division. This protein encodes a DEAD box protein, which is an ATPase activated by a variety of RNA species, but not by dsDNA. This protein, and that encoded by DDX5 gene, are more closely related to each other than to any other member of the DEAD box family.

DDX17 Antibody (N-term) Blocking Peptide - References

Wortham,N.C., et.al., Oncogene 28 (46), 4053-4064 (2009)Tonevitsky,E.A., et.al., Bull. Exp. Biol. Med. 147 (6), 733-736 (2009)Wong,H.Y., et.al., Biochim. Biophys. Acta 1794 (2), 193-198 (2009)