

INTS6 Blocking Peptide (N-Term)
Synthetic peptide
Catalog # BP21956a**Specification**

INTS6 Blocking Peptide (N-Term) - Product InformationPrimary Accession
Other Accession[Q9UL03](#)
[Q5JSJ4](#), [Q8BND4](#), [Q2TAF4](#), [Q5U4W6](#), [Q7SYD9](#),
[Q6PCM2](#)**INTS6 Blocking Peptide (N-Term) - Additional Information****Gene ID** 26512**Other Names**

Integrator complex subunit 6, Int6, DBI-1, Protein DDX26, Protein deleted in cancer 1, DICE1, INTS6, DBI1, DDX26, DDX26A

Target/Specificity

The synthetic peptide sequence is selected from aa 155-167 of HUMAN INTS6

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.

INTS6 Blocking Peptide (N-Term) - Protein Information**Name** INTS6 {ECO:0000303|PubMed:33243860, ECO:0000312|HGNC:HGNC:14879}**Function**

Component of the integrator complex, a multiprotein complex that terminates RNA polymerase II (Pol II) transcription in the promoter-proximal region of genes (PubMed:33243860, PubMed:34004147). The integrator complex provides a quality checkpoint during transcription elongation by driving premature transcription termination of transcripts that are unfavorably configured for transcriptional elongation: the complex terminates transcription by (1) catalyzing dephosphorylation of the C-terminal domain (CTD) of Pol II subunit POLR2A/RPB1 and SUPT5H/SPT5, (2) degrading the exiting nascent RNA transcript via endonuclease activity and (3) promoting the release of Pol II from bound DNA (PubMed:33243860, PubMed:34004147, PubMed:34004147, PubMed:34004147, PubMed:34004147).

[38570683](http://www.uniprot.org/citations/38570683)). The integrator complex is also involved in terminating the synthesis of non-coding Pol II transcripts, such as enhancer RNAs (eRNAs), small nuclear RNAs (snRNAs), telomerase RNAs and long non-coding RNAs (lncRNAs) (PubMed: [16239144](http://www.uniprot.org/citations/16239144)). Within the integrator complex, INTS6 acts as a molecular adapter that promotes assembly of protein phosphatase 2A (PP2A) subunits to the integrator core complex, promoting recruitment of PP2A to transcription pause-release checkpoint (PubMed: [33243860](http://www.uniprot.org/citations/33243860), PubMed: [34004147](http://www.uniprot.org/citations/34004147)). Mediates recruitment of cytoplasmic dynein to the nuclear envelope, probably as component of the integrator complex (PubMed: [23904267](http://www.uniprot.org/citations/23904267)). May have a tumor suppressor role; an ectopic expression suppressing tumor cell growth (PubMed: [15254679](http://www.uniprot.org/citations/15254679), PubMed: [16239144](http://www.uniprot.org/citations/16239144)).

Cellular Location

Nucleus. Chromosome Note=Associates with chromatin and transcription pause-release checkpoint.

Tissue Location

Widely expressed. Expressed in heart, brain, placenta, lung, liver, skeletal muscle, kidney and pancreas

INTS6 Blocking Peptide (N-Term) - Protocols

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

- [Blocking Peptides](#)

INTS6 Blocking Peptide (N-Term) - Images

INTS6 Blocking Peptide (N-Term) - Background

Component of the Integrator complex, a complex involved in the small nuclear RNAs (snRNA) U1 and U2 transcription and in their 3'-box-dependent processing. The Integrator complex is associated with the C-terminal domain (CTD) of RNA polymerase II largest subunit (POLR2A) and is recruited to the U1 and U2 snRNAs genes. May have a tumor suppressor role; an ectopic expression suppressing tumor cell growth.

INTS6 Blocking Peptide (N-Term) - References

Wieland I., et al. Oncogene 18:4530-4537(1999).
Bechtel S., et al. BMC Genomics 8:399-399(2007).
Dunham A., et al. Nature 428:522-528(2004).
Hoff H.B. III, et al. Submitted (APR-1999) to the EMBL/GenBank/DDBJ databases.
Wieland I., et al. Oncol. Res. 12:491-500(2001).