

**DTL Antibody (Center) Blocking peptide**  
**Synthetic peptide**  
**Catalog # BP10973c****Specification**

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**DTL Antibody (Center) Blocking peptide - Product Information**Primary Accession [O9NZJ0](#)**DTL Antibody (Center) Blocking peptide - Additional Information****Gene ID** 51514**Other Names**

Denticleless protein homolog, DDB1- and CUL4-associated factor 2, Lethal(2) denticleless protein homolog, Retinoic acid-regulated nuclear matrix-associated protein, DTL, CDT2, CDW1, DCAF2, L2DTL, RAMP

**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.

**DTL Antibody (Center) Blocking peptide - Protein Information****Name** DTL**Synonyms** CDT2, CDW1, DCAF2, L2DTL, RAMP**Function**

Substrate-specific adapter of a DCX (DDB1-CUL4-X-box) E3 ubiquitin-protein ligase complex required for cell cycle control, DNA damage response and translesion DNA synthesis. The DCX(DTL) complex, also named CRL4(CDT2) complex, mediates the polyubiquitination and subsequent degradation of CDT1, CDKN1A/p21(CIP1), FBH1, KMT5A and SDE2 (PubMed:<a href="http://www.uniprot.org/citations/16861906" target="\_blank">16861906</a>, PubMed:<a href="http://www.uniprot.org/citations/16949367" target="\_blank">16949367</a>, PubMed:<a href="http://www.uniprot.org/citations/16964240" target="\_blank">16964240</a>, PubMed:<a href="http://www.uniprot.org/citations/17085480" target="\_blank">17085480</a>, PubMed:<a href="http://www.uniprot.org/citations/18703516" target="\_blank">18703516</a>, PubMed:<a href="http://www.uniprot.org/citations/18794347" target="\_blank">18794347</a>, PubMed:<a href="http://www.uniprot.org/citations/18794348" target="\_blank">18794348</a>, PubMed:<a href="http://www.uniprot.org/citations/19332548" target="\_blank">19332548</a>, PubMed:<a href="http://www.uniprot.org/citations/20129063" target="\_blank">20129063</a>, PubMed:<a href="http://www.uniprot.org/citations/23478441" target="\_blank">23478441</a>, PubMed:<a

[23478445](http://www.uniprot.org/citations/23478445), PubMed: [23677613](http://www.uniprot.org/citations/23677613), PubMed: [27906959](http://www.uniprot.org/citations/27906959)). CDT1 degradation in response to DNA damage is necessary to ensure proper cell cycle regulation of DNA replication (PubMed: [16861906](http://www.uniprot.org/citations/16861906), PubMed: [16949367](http://www.uniprot.org/citations/16949367), PubMed: [17085480](http://www.uniprot.org/citations/17085480)). CDKN1A/p21(CIP1) degradation during S phase or following UV irradiation is essential to control replication licensing (PubMed: [18794348](http://www.uniprot.org/citations/18794348), PubMed: [19332548](http://www.uniprot.org/citations/19332548)). KMT5A degradation is also important for a proper regulation of mechanisms such as TGF-beta signaling, cell cycle progression, DNA repair and cell migration (PubMed: [23478445](http://www.uniprot.org/citations/23478445)). Most substrates require their interaction with PCNA for their polyubiquitination: substrates interact with PCNA via their PIP-box, and those containing the 'K+4' motif in the PIP box, recruit the DCX(DTL) complex, leading to their degradation. In undamaged proliferating cells, the DCX(DTL) complex also promotes the 'Lys-164' monoubiquitination of PCNA, thereby being involved in PCNA-dependent translesion DNA synthesis (PubMed: [20129063](http://www.uniprot.org/citations/20129063), PubMed: [23478441](http://www.uniprot.org/citations/23478441), PubMed: [23478445](http://www.uniprot.org/citations/23478445), PubMed: [23677613](http://www.uniprot.org/citations/23677613)). The DDB1-CUL4A-DTL E3 ligase complex regulates the circadian clock function by mediating the ubiquitination and degradation of CRY1 (PubMed: [26431207](http://www.uniprot.org/citations/26431207)).

### Cellular Location

Nucleus. Nucleus membrane; Peripheral membrane protein; Nucleoplasmic side. Cytoplasm, cytoskeleton, microtubule organizing center, centrosome. Chromosome Note=Nuclear matrix-associated protein. Translocates from the interphase nucleus to the metaphase cytoplasm during mitosis

### Tissue Location

Expressed in placenta and testis, very low expression seen in skeletal muscle. Detected in all hematopoietic tissues examined, with highest expression in thymus and bone marrow. A low level detected in the spleen and lymph node, and barely detectable level in the peripheral leukocytes. RA treatment down-regulated the expression in NT2 cell.

## DTL Antibody (Center) Blocking peptide - Protocols

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

- [Blocking Peptides](#)

## DTL Antibody (Center) Blocking peptide - Images

## DTL Antibody (Center) Blocking peptide - Background

Substrate-specific adapter of a DCX (DDB1-CUL4-X-box) E3 ubiquitin-protein ligase complex required for cell cycle control, DNA damage response and translesion DNA synthesis. The DCX(DTL) complex, also named CRL4(CDT2) complex, mediates the polyubiquitination and subsequent degradation of CDT1 and CDKN1A/p21(CIP1). CDT1 degradation in response to DNA damage is necessary to ensure proper cell cycle regulation of DNA replication. CDKN1A/p21(CIP1) degradation during S phase or following UV irradiation is essential to control replication licensing. Most substrates require their interaction with PCNA for their polyubiquitination: substrates interact with

PCNA via their PIP-box, and those containing the 'K+4' motif in the PIP box, recruit the DCX(DTL) complex, leading to their degradation. In undamaged proliferating cells, the DCX(DTL) complex also promotes the 'Lys-164' monoubiquitination of PCNA, thereby being involved in PCNA-dependent translesion DNA synthesis.

#### **DTL Antibody (Center) Blocking peptide - References**

Centore, R.C., et al. Mol. Cell 40(1):22-33(2010)Abbas, T., et al. Mol. Cell 40(1):9-21(2010)Song, B., et al. Mol. Cancer 9, 96 (2010) :Li, J., et al. Br. J. Cancer 101(4):691-698(2009)Abbas, T., et al. Genes Dev. 22(18):2496-2506(2008)