

**LIN28A Antibody**  
**Purified Mouse Monoclonal Antibody (Mab)**  
**Catalog # AP52783****Specification**

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**LIN28A Antibody - Product Information**

Application	<b>WB</b>
Primary Accession	<a href="#">O9H9Z2</a>
Reactivity	<b>Human, Mouse</b>
Host	<b>Mouse</b>
Clonality	<b>Monoclonal</b>
Isotype	<b>IgG2a</b>
Calculated MW	<b>26 KDa</b>

**LIN28A Antibody - Additional Information****Gene ID** 79727**Other Names**

CSDD1;FLJ12457;LIN 28;Lin-28A;LIN28;LIN28A;LN28A\_HUMAN;Protein lin-28 homolog A;ZCCHC1; Zinc finger CCHC domain-containing protein 1.

**Dilution**

WB~~1:1000

**Format**

Liquid in PBS containing 50% glycerol, 0.5% BSA and 0.02% sodium azide, pH 7.3.

**Storage**

Store at 4°C short term. Aliquot and store at -20°C long term. Avoid freeze/thaw cycles.

**LIN28A Antibody - Protein Information****Name** LIN28A**Synonyms** CSDD1, LIN28, ZCCHC1**Function**

RNA-binding protein that inhibits processing of pre-let-7 miRNAs and regulates translation of mRNAs that control developmental timing, pluripotency and metabolism (PubMed:<a href="http://www.uniprot.org/citations/21247876" target="\_blank">21247876</a>). Seems to recognize a common structural G-quartet (G4) feature in its miRNA and mRNA targets (Probable). 'Translational enhancer' that drives specific mRNAs to polysomes and increases the efficiency of protein synthesis. Its association with the translational machinery and target mRNAs results in an increased number of initiation events per molecule of mRNA and, indirectly, in mRNA stabilization. Binds IGF2 mRNA, MYOD1 mRNA, ARBP/36B4 ribosomal protein mRNA and its own mRNA. Essential for skeletal muscle differentiation program through the translational up- regulation of IGF2 expression. Suppressor of microRNA (miRNA) biogenesis, including that of let-7, miR107,

miR-143 and miR-200c. Specifically binds the miRNA precursors (pre-miRNAs), recognizing an 5'-GGAG-3' motif found in pre-miRNA terminal loop, and recruits TUT4 and TUT7 uridylyltransferases (PubMed:<a href="http://www.uniprot.org/citations/18951094" target="\_blank">18951094</a>, PubMed:<a href="http://www.uniprot.org/citations/19703396" target="\_blank">19703396</a>, PubMed:<a href="http://www.uniprot.org/citations/22118463" target="\_blank">22118463</a>, PubMed:<a href="http://www.uniprot.org/citations/22898984" target="\_blank">22898984</a>). This results in the terminal uridylation of target pre-miRNAs (PubMed:<a href="http://www.uniprot.org/citations/18951094" target="\_blank">18951094</a>, PubMed:<a href="http://www.uniprot.org/citations/19703396" target="\_blank">19703396</a>, PubMed:<a href="http://www.uniprot.org/citations/22118463" target="\_blank">22118463</a>, PubMed:<a href="http://www.uniprot.org/citations/22898984" target="\_blank">22898984</a>). Uridylated pre-miRNAs fail to be processed by Dicer and undergo degradation. The repression of let-7 expression is required for normal development and contributes to maintain the pluripotent state by preventing let-7-mediated differentiation of embryonic stem cells (PubMed:<a href="http://www.uniprot.org/citations/18951094" target="\_blank">18951094</a>, PubMed:<a href="http://www.uniprot.org/citations/19703396" target="\_blank">19703396</a>, PubMed:<a href="http://www.uniprot.org/citations/22118463" target="\_blank">22118463</a>, PubMed:<a href="http://www.uniprot.org/citations/22898984" target="\_blank">22898984</a>). Localized to the periendoplasmic reticulum area, binds to a large number of spliced mRNAs and inhibits the translation of mRNAs destined for the ER, reducing the synthesis of transmembrane proteins, ER or Golgi lumen proteins, and secretory proteins. Binds to and enhances the translation of mRNAs for several metabolic enzymes, such as PFKF, PDHA1 or SDHA, increasing glycolysis and oxidative phosphorylation. Which, with the let-7 repression may enhance tissue repair in adult tissue (By similarity).

#### Cellular Location

Cytoplasm. Rough endoplasmic reticulum {ECO:0000250|UniProtKB:Q8K3Y3}. Cytoplasm, P-body. Cytoplasm, Stress granule. Nucleus, nucleolus {ECO:0000250|UniProtKB:Q8K3Y3}. Note=Predominantly cytoplasmic (PubMed:22118463). In the cytoplasm, localizes to peri-endoplasmic reticulum regions and detected in the microsomal fraction derived from rough endoplasmic reticulum (RER) following subcellular fractionation May be bound to the cytosolic surface of RER on which ER-associated mRNAs are translated (By similarity). Shuttle from the nucleus to the cytoplasm requires RNA-binding (PubMed:17617744). Nucleolar localization is observed in 10-15% of the nuclei in differentiated myotubes (By similarity). {ECO:0000250|UniProtKB:Q8K3Y3, ECO:0000269|PubMed:17617744, ECO:0000269|PubMed:22118463}

#### Tissue Location

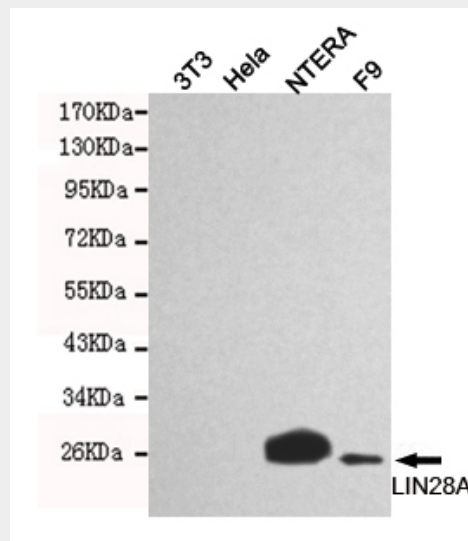
Expressed in embryonic stem cells, placenta and testis. Tends to be up-regulated in HER2-overexpressing breast tumors

#### LIN28A Antibody - Protocols

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

- [Western Blot](#)
- [Blocking Peptides](#)
- [Dot Blot](#)
- [Immunohistochemistry](#)
- [Immunofluorescence](#)
- [Immunoprecipitation](#)
- [Flow Cytometry](#)
- [Cell Culture](#)

#### LIN28A Antibody - Images



Western blot detection of LIN28 in NTERA and F9 cell lysates using LIN28A mouse mAb (1:1000 diluted). Predicted band size: 26KDa. Observed band size: 26KDa.

#### LIN28A Antibody - Background

Acts as a 'translational enhancer', driving specific mRNAs to polysomes and thus increasing the efficiency of protein synthesis. Its association with the translational machinery and target mRNAs results in an increased number of initiation events per molecule of mRNA and, indirectly, in stabilizing the mRNAs. Binds IGF2 mRNA, MYOD1 mRNA, ARBP/36B4 ribosomal protein mRNA and its own mRNA. Essential for skeletal muscle differentiation program through the translational up-regulation of IGF2 expression (By similarity). Acts as a suppressor of microRNA (miRNA) biogenesis by specifically binding the precursor let-7 (pre-let-7), a miRNA precursor. Acts by binding pre-let-7 and recruiting ZCCHC11/TUT4 uridylyltransferase, leading to the terminal uridylation of pre-let-7. Uridylated pre-let-7 miRNAs fail to be processed by Dicer and undergo degradation. Degradation of pre-let-7 in embryonic stem (ES) cells contributes to the maintenance of ES cells. In contrast, LIN28A down-regulation in neural stem cells by miR-125, allows the processing of pre-let-7. Specifically recognizes the 5'-GGAG-3' motif in the terminal loop of pre-let-7. Also recognizes and binds non pre-let-7 pre-miRNAs that contain the 5'-GGAG-3' motif in the terminal loop, leading to their terminal uridylation and subsequent degradation.

#### LIN28A Antibody - References

- Moss E.G., et al. Dev. Biol. 258:432-442(2003).
- Moss E.G., et al. Dev. Biol. 262:361-361(2003).
- Ota T., et al. Nat. Genet. 36:40-45(2004).
- Gregory S.G., et al. Nature 441:315-321(2006).
- Sempere L.F., et al. Genome Biol. 5:R13.1-R13.11(2004).