

**SNAI1 Antibody (N-term) Blocking Peptide**  
**Synthetic peptide**  
**Catalog # BP2054d****Specification**

---

**SNAI1 Antibody (N-term) Blocking Peptide - Product Information**Primary Accession [O95863](#)**SNAI1 Antibody (N-term) Blocking Peptide - Additional Information****Gene ID** 6615**Other Names**

Zinc finger protein SNAI1, Protein snail homolog 1, Protein sna, SNAI1, SNAH

**Target/Specificity**

The synthetic peptide sequence used to generate the antibody [AP2054d](/products/AP2054d) was selected from the N-term region of human SNAI1. A 10 to 100 fold molar excess to antibody is recommended. Precise conditions should be optimized for a particular assay.

**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.

**SNAI1 Antibody (N-term) Blocking Peptide - Protein Information****Name** SNAI1**Synonyms** SNAH**Function**

Involved in induction of the epithelial to mesenchymal transition (EMT), formation and maintenance of embryonic mesoderm, growth arrest, survival and cell migration. Binds to 3 E-boxes of the E-cadherin/CDH1 gene promoter and to the promoters of CLDN7 and KRT8 and, in association with histone demethylase KDM1A which it recruits to the promoters, causes a decrease in dimethylated H3K4 levels and represses transcription (PubMed: [20389281](http://www.uniprot.org/citations/20389281), PubMed: [20562920](http://www.uniprot.org/citations/20562920)). The N-terminal SNAG domain competes with histone H3 for the same binding site on the histone demethylase complex formed by KDM1A and RCOR1, and thereby inhibits demethylation of histone H3 at 'Lys-4' (in vitro) (PubMed: [20389281](http://www.uniprot.org/citations/20389281))

target="\_blank">20389281</a>, PubMed:<a href="http://www.uniprot.org/citations/21300290" target="\_blank">21300290</a>, PubMed:<a href="http://www.uniprot.org/citations/23721412" target="\_blank">23721412</a>). During EMT, involved with LOXL2 in negatively regulating pericentromeric heterochromatin transcription (By similarity). SNAI1 recruits LOXL2 to pericentromeric regions to oxidize histone H3 and repress transcription which leads to release of heterochromatin component CBX5/HP1A, enabling chromatin reorganization and acquisition of mesenchymal traits (By similarity). Associates with EGR1 and SP1 to mediate tetradecanoyl phorbol acetate (TPA)-induced up-regulation of CDKN2B, possibly by binding to the CDKN2B promoter region 5'-TCACA-3. In addition, may also activate the CDKN2B promoter by itself.

#### **Cellular Location**

Nucleus. Cytoplasm. Note=Once phosphorylated (probably on Ser-107, Ser-111, Ser-115 and Ser-119) it is exported from the nucleus to the cytoplasm where subsequent phosphorylation of the destruction motif and ubiquitination involving BTRC occurs

#### **Tissue Location**

Expressed in a variety of tissues with the highest expression in kidney. Expressed in mesenchymal and epithelial cell lines.

### **SNAI1 Antibody (N-term) Blocking Peptide - Protocols**

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

- [Blocking Peptides](#)

### **SNAI1 Antibody (N-term) Blocking Peptide - Images**

### **SNAI1 Antibody (N-term) Blocking Peptide - Background**

The Drosophila embryonic protein snail is a zinc finger transcriptional repressor which downregulates the expression of ectodermal genes within the mesoderm. The nuclear protein is structurally similar to the Drosophila snail protein, and is also thought to be critical for mesoderm formation in the developing embryo.

### **SNAI1 Antibody (N-term) Blocking Peptide - References**

Strausberg, R.L., et al., Proc. Natl. Acad. Sci. U.S.A. 99(26):16899-16903 (2002).Deloukas, P., et al., Nature 414(6866):865-871 (2001).Batlle, E., et al., Nat. Cell Biol. 2(2):84-89 (2000).Paznekas, W.A., et al., Genomics 62(1):42-49 (1999).Twigg, S.R., et al., Hum. Genet. 105(4):320-326 (1999).