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SLUG Antibody (Center) Blocking Peptide

Synthetic peptide
Catalog # BP2053a

Specification

SLUG Antibody (Center) Blocking Peptide - Product Information

Primary Accession O43623

SLUG Antibody (Center) Blocking Peptide - Additional Information

Gene ID 6591

Other Names
Zinc finger protein SNAI2, Neural crest transcription factor Slug, Protein snail homolog 2, SNAI2, SLUG, SLUGH

Target/Specificity
The synthetic peptide sequence used to generate the antibody AP2053a was selected from the Center region of human SLUG . 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.

SLUG Antibody (Center) Blocking Peptide - Protein Information

Name SNAI2

Synonyms SLUG, SLUGH

Function
Transcriptional repressor that modulates both activator- dependent and basal transcription. Involved in the generation and migration of neural crest cells. Plays a role in mediating RAF1-induced transcriptional repression of the TJ protein, occludin (OCLN) and subsequent oncogenic transformation of epithelial cells (By similarity). Represses BRCA2 expression by binding to its E2-box- containing silencer and recruiting CTBP1 and HDAC1 in breast cells. In epidermal keratinocytes, binds to the E-box in ITGA3 promoter and represses its transcription. Involved in the regulation of ITGB1 and ITGB4 expression and cell adhesion and proliferation in epidermal keratinocytes. Binds to E-box2 domain of BSG and activates its expression during TGFB1-induced epithelial-mesenchymal transition (EMT) in hepatocytes. Represses E-Cadherin/CDH1 transcription

via E-box elements. Involved in osteoblast maturation. Binds to RUNX2 and SOC9 promoters and may act as a positive and negative transcription regulator, respectively, in osteoblasts. Binds to CXCL12 promoter via E-box regions in mesenchymal stem cells and osteoblasts. Plays an essential role in TWIST1-induced EMT and its ability to promote invasion and metastasis.

Cellular Location
Nucleus. Cytoplasm. Note=Observed in discrete foci in interphase nuclei. These nuclear foci do not overlap with the nucleoli, the SP100 and the HP1 heterochromatin or the coiled body, suggesting SNAI2 is associated with active transcription or active splicing regions

Tissue Location
Expressed in most adult human tissues, including spleen, thymus, prostate, testis, ovary, small intestine, colon, heart, brain, placenta, lung, liver, skeletal muscle, kidney and pancreas. Not detected in peripheral blood leukocyte. Expressed in the dermis and in all layers of the epidermis, with high levels of expression in the basal layers (at protein level). Expressed in osteoblasts (at protein level). Expressed in mesenchymal stem cells (at protein level) Expressed in breast tumor cells (at protein level)

SLUG Antibody (Center) Blocking Peptide - Protocols

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

- Blocking Peptides

SLUG Antibody (Center) Blocking Peptide - Images

SLUG Antibody (Center) Blocking Peptide - Background

SLUG is a member of the Snail family of C2H2-type zinc finger transcription factors. The encoded protein acts as a transcriptional repressor that binds to E-box motifs and is also likely to repress E-cadherin transcription in breast carcinoma. This protein is involved in epithelial-mesenchymal transitions and has antiapoptotic activity. Mutations in this gene may be associated with sporadic cases of neural tube defects.

SLUG Antibody (Center) Blocking Peptide - References

Sanchez-Martin, M., et al., Hum. Mol. Genet. 11(25):3231-3236 (2002).Hajra, K.M., et al., Cancer Res. 62(6):1613-1618 (2002).Hemavathy, K., et al., Mol. Cell. Biol. 20(14):5087-5095 (2000).Inukai, T., et al., Mol. Cell 4(3):343-352 (1999).Cohen, M.E., et al., Genomics 51(3):468-471 (1998).