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ASCL2 Antibody (N-term) Blocking peptide

Synthetic peptide
Catalog # BP10976a

Specification

ASCL2 Antibody (N-term) Blocking peptide - Product Information

Primary Accession Q99929

ASCL2 Antibody (N-term) Blocking peptide - Additional Information

Gene ID 430

Other Names
Achaete-scute homolog 2, ASH-2, hASH2, Class A basic helix-loop-helix protein 45, bHLHa45, Mash2, ASCL2, BHLHA45, HASH2

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.

ASCL2 Antibody (N-term) Blocking peptide - Protein Information

Name ASCL2

Synonyms BHLHA45, HASH2

Function
Transcription factor. Binds to E-box motifs 5'-CANNTG-3' in the regulatory elements of target genes, probably as a heterodimer with another basic helix-loop-helix (bHLH) protein such as the transcription factor TCF3. May bind both open and closed chromatin, acting as a pioneer transcription factor to allow other factors to bind and activate lineage-specific genes. Required during post-implantation development for the generation of some differentiated trophoblast cell types. Transcriptional activity of ASCL2 may be antagonised in a subset of trophoblast cells by bHLH transcription factor HAND1, perhaps by competing for dimerization with other bHLH proteins. Involved in differentiation and function of follicular T-helper (Tfh) cells, thereby playing a role in germinal center responses; probably modulates expression of genes involved in Tfh cell function, such as BCL6. May also act as a suppressor of Th1-, Th2- and Th17-cell differentiation. Induces the formation of stem cells in intestinal crypts in vitro, synergistically activating transcription of target genes, such as SOX9, together with TCF4/beta-catenin. May form a bistable transcriptional switch, controlling expression of its own gene together with Wnt/R- spondin signaling, and thereby maintaining stem cell characteristics (By similarity). Modulates expression of target genes, including perhaps down-regulating EGR1/Krox24 and chemokine CXCL10/Mob-1 and up- regulating

CXCR4 and CDKN1C/p57kip2, in Schwann cells. May play a role in reducing proliferation of Schwann cells, perhaps acting via modulation of expression of CDKN1C (By similarity). May be dispensable for blastocyst formation and later embryonic function (By similarity). May be involved in the determination of neuronal precursors (By similarity).

Cellular Location
Nucleus {ECO:0000255|PROSITE-ProRule:PRU00981, ECO:0000269|PubMed:11440538}

Tissue Location
Expressed in the placenta at a stage between the first and second trimesters and when it matures, at about 32-36 weeks (PubMed:12099555). Expressed in the extravillous trophoblasts, the intermediate trophoblasts, and at lower levels in the cytotrophoblasts and stroma of chorionic villi of the developing placenta (PubMed:9175731, PubMed:12099555). Expressed in follicular T-helper (Tfh) cells (PubMed:24463518).

ASCL2 Antibody (N-term) Blocking peptide - Protocols

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

- Blocking Peptides

ASCL2 Antibody (N-term) Blocking peptide - Images

ASCL2 Antibody (N-term) Blocking peptide - Background

This gene is a member of the basic helix-loop-helix (BHLH)family of transcription factors. It activates transcription bybinding to the E box (5'-CANNTG-3'). Dimerization with other BHLHproteins is required for efficient DNA binding. Involved in thedetermination of the neuronal precursors in the peripheral nervoussystem and the central nervous system.

ASCL2 Antibody (N-term) Blocking peptide - References

Stange, D.E., et al. Gut 59(9):1236-1244(2010)Eeles, R.A., et al. Nat. Genet. 41(10):1116-1121(2009)Barrett, J.C., et al. Nat. Genet. 41(6):703-707(2009)Shahib, M.N., et al. J Reprod Med 51(11):892-896(2006)Jubb, A.M., et al. Oncogene 25(24):3445-3457(2006)