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SIGLEC7 (D-siglec) Antibody (C-term H422) Blocking peptide

Synthetic peptide
Catalog # BP1633c

Specification

SIGLEC7 (D-siglec) Antibody (C-term H422) Blocking peptide - Product Information

Primary Accession	Q9Y286
Other Accession	Q9NZQ1

SIGLEC7 (D-siglec) Antibody (C-term H422) Blocking peptide - Additional Information

Gene ID 27036

Other Names
Sialic acid-binding Ig-like lectin 7, Siglec-7, Adhesion inhibitory receptor molecule 1, AIRM-1, CDw328, D-siglec, QA79 membrane protein, p75, CD328, SIGLEC7, AIRM1

Target/Specificity
The synthetic peptide sequence used to generate the antibody AP1633c was selected from the C-term region of human D-siglec . 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.

SIGLEC7 (D-siglec) Antibody (C-term H422) Blocking peptide - Protein Information

Name SIGLEC7

Synonyms AIRM1

Function
Putative adhesion molecule that mediates sialic-acid dependent binding to cells. Preferentially binds to alpha-2,3- and alpha-2,6-linked sialic acid. Also binds disialogangliosides (disialogalactosyl globoside, disialyl lactotetraosylceramide and disialyl GalNAc lactotetraoslylceramide). The sialic acid recognition site may be masked by cis interactions with sialic acids on the same cell surface. In the immune response, may act as an inhibitory receptor upon ligand induced tyrosine phosphorylation by recruiting cytoplasmic phosphatase(s) via their SH2 domain(s) that block signal transduction through dephosphorylation of signaling molecules. Mediates inhibition of natural killer cells cytotoxicity. May play a role in hemopoiesis. Inhibits differentiation of CD34+

cell precursors towards myelomonocytic cell lineage and proliferation of leukemic myeloid cells (in vitro).

Cellular Location
Membrane; Single-pass type I membrane protein.

Tissue Location
Predominantly expressed by resting and activated natural killer cells and at lower levels by granulocytes and monocytes High expression found in placenta, liver, lung, spleen, and peripheral blood leukocytes

SIGLEC7 (D-siglec) Antibody (C-term H422) Blocking peptide - Protocols

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

- Blocking Peptides

SIGLEC7 (D-siglec) Antibody (C-term H422) Blocking peptide - Images

SIGLEC7 (D-siglec) Antibody (C-term H422) Blocking peptide - Background

SIGLECs are cell surface proteins of the Ig superfamily. Most SIGLECs have 1 or more cytoplasmic immune receptor tyrosine-based inhibitory motifs, or ITIMs. A large subgroup of SIGLECs share high homology with SIGLEC3 (CD33) and are localized to 19q13.4. The cDNA for the SLG gene encodes 2 variants, SLG-long (SLGL) and SLG-short (SLGS). The 595-amino acid SLGL protein contains a signal peptide and 2 V-set N-terminal Ig-like domains. The 477-amino acid SLGS protein has a weak signal sequence and, like most SIGLEC3-like SIGLECs, has only 1 V-set N-terminal Ig-like domain. Both variants contain 2 C2-set N-terminal Ig-like domains, a transmembrane domain, and a cytoplasmic tail with a putative ITIM and a putative SLAM-like tyrosine-based motif. The conserved arginine residue thought to be essential for sialic acid binding in other SIGLECs is replaced by a glutamine in SLGS and by a cysteine in SLGL. RT-PCR analysis detected high expression of both variants in spleen and small intestine, and SLGS was highly expressed in adrenal gland and SLGL was highly expressed in bone marrow.

SIGLEC7 (D-siglec) Antibody (C-term H422) Blocking peptide - References

Nicoll, G., et al., Eur. J. Immunol. 33(6):1642-1648 (2003).Alphey, M.S., et al., J. Biol. Chem. 278(5):3372-3377 (2003).Angata, T., et al., Glycobiology 10(4):431-438 (2000).Falco, M., et al., J. Exp. Med. 190(6):793-802 (1999).Nicoll, G., et al., J. Biol. Chem. 274(48):34089-34095 (1999).