Animal-Free APRIL/TNFSF13 Protein, Human (His)

Cat. No.:	HY-P700016AF
Synonyms:	TNFSF13; CD256; TALL-2; TALL2; TNLG7B; TRDL-1; UNQ383/PRO715; ZTNF2
Species:	Human
Source:	E. coli
Accession:	O75888 (A105-L250)
Gene ID:	8741
Molecular Weight:	Approximately 17.29 kDa

PROPERTIES

AA Sequence	M A V L T Q K Q K K Q H S V L H L V P I N A T S K D D S D V T E V M W Q P A L R R G R G L Q A Q G Y G V R I Q D A G V Y L L Y S Q V L F Q D V T F T M G Q V V S R E G Q G R Q E T L F R C I R S M P S H P D R A Y N S C Y S A G V F H L H Q G D I L S V I I P R A R A K L N L S P H G T F L G F V K L
Biological Activity	Measured by its ability to induce cell death in Jurkat cells. The ED_{50} for this effect is 2.6-4.0 μg/mL
Appearance	Lyophilized powder.
Formulation	Lyophilized from a solution containing 0.1% sarkosyl in 1X PBS, pH8.0.
Endotoxin Level	<0.1 EU per 1 μg of the protein by the LAL method.
Reconsititution	It is not recommended to reconstitute to a concentration less than 100 μg/mL in ddH_2O.
Storage & Stability	Stored at -20°C for 2 years. After reconstitution, it is stable at 4°C for 1 week or -20°C for longer (with carrier protein). It is recommended to freeze aliquots at -20°C or -80°C for extended storage.
Shipping	Room temperature in continental US; may vary elsewhere.

DESCRIPTION

Background	APRIL/TNFSF13 Protein is a cytokine and an independent secretory ligand belongs to TNF family. It binds to TNFRSF13B/TACI and to TNFRSF17/BCMA. APRIL/TNFSF13 Protein plays a role in the regulation of tumor cell growth, may involve in monocyte/macrophage-mediated immunological processes[1]. APRIL is produced by myeloid cells and their precursors in the bone marrow. APRIL is retained by surrounding tissues and via HSPG (heparan sulfate proteoglycans) is not retained. It accumulates in large amounts in the bone marrow, leading to more rapid cell maturation and peripheral burst. As for infection response, tonsil mucosa neutrophils present within the infected tissue were the main source of APRIL, whereas keratinocytes were the primary source of APRIL in tissues showing

no symptoms of infection[2].

APRIL acts function by binding BCMA (B cell maturation antigen) and TACI (transmembrane activator and CAML-interactor) and competes with TALL-I (also called BLyS or BAFF) for receptor binding. Soluble BCMA and TACI specifically prevent binding of APRIL and block APRIL-stimulated proliferation of primary B cells, and soluble BCMA is a dominant-negative molecule capable of inhibiting antibody production in vivo. Thus, APRIL stimulates in vitro the proliferation of primary lymphocytes, in addition to lymphoma cell lines, and promotes in vivo the accumulation of B cells in the spleen. Therefore, APRIL-TALL-I and BCMA-TACI form a two ligands-two receptors pathway involved in stimulation of B and T cell function. Moreover, APRIL is also a stimulator of tumor cell growth although TNRF death ligand-1 (TRDL-1), which induces tumor cell apoptosis[1].

It is a type II membrane protein with a cytoplasmic domain, a hydrophobic transmembrane region, and an extracellular domain[2]. Mouse and human APRIL proteins are 82% identical in the COOH-terminal part of the extracellular domain, which contains the presumed receptor-binding domain. And the protein sequences of human and mouse are different with similarity of 80.91%. The APRIL protein is most often studied in the context of lymphoid malignancies[1].

REFERENCES

[1]. Yu G, et al. APRIL and TALL-I and receptors BCMA and TACI: system for regulating humoral immunity. Nat Immunol. 2000 Sep;1(3):252-6.

[2]. Nowacka KH, et al. Role of the APRIL molecule in solid tumors. Cytokine Growth Factor Rev. 2021 Oct;61:38-44.

[3]. Ding W, et al. APRIL knockdown suppresses migration and invasion of human colon carcinoma cells. Clin Biochem. 2009 Nov;42(16-17):1694-8.

[4]. Deshayes F, et al. Abnormal production of the TNF-homologue APRIL increases the proliferation of human malignant glioblastoma cell lines via a specific receptor. Oncogene. 2004 Apr 15;23(17):3005-12.

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