BCMA/TNFRSF17 Trimer Protein, Human (HEK293, His-Avi)

Cat. No.:	HY-P78389
Synonyms:	CD269; TNFRSF17; BCMA; BCM; TNFRSF13A
Species:	Human
Source:	HEK293
Accession:	Q02223 (M1-A54)
Gene ID:	608
Molecular Weight:	30-48 kDa

PROPERTIES

Biological Activity	1. Immobilized Human BCMA Trimer at 0.5μg/ml (100μl/Well) on the plate. Dose response curve for Human BAFF, hFc Tag with the EC_{50} of 17ng/ml determined by ELISA. 2. Immobilized Human BCMA Trimer at 0.5μg/ml (100μl/Well) on the plate. Dose response curve for Anti-BCMA Antibody, hFc Tag with the EC_{50} of 4.0ng/ml determined by ELISA.
Appearance	Lyophilized powder.
Formulation	Lyophilized from a 0.22 μm filtered solution of PBS, pH 7.4. Normally 5% trehalose is added as protectant before lyophilization.
Endotoxin Level	<1 EU/μg, determined by 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	BCMA is expressed preferentially by mature B lymphocytes, with minimal expression in hematopoietic stem cells or nonhematopoietic tissue[1]. BCMA is almost exclusively expressed on plasmablasts and PCs[2]. The amino acid sequence of human BCMA protein has low homology for mouse BCMA protein. BCMA is a 184 amino acid and 20.2-kDa type III transmembrane glycoprotein, with the extracellular N terminus containing a conserved motif of 6 cysteines. BCMA has two agonist ligands: a proliferation-inducing ligand (APRIL) and B cell activating factor (BAFF). Upon binding of the ligands to BCMA, activates B cells (NF-κβ), rat sarcoma/mitogen-activated protein kinase (RAS/MAPK), and phosphoinositide-3-kinase-protein kinase B/Akt (PI3K-PKB/Akt) signaling pathway. These pathways result in proliferation stimulation by modulating cell cycle checkpoints, increasing survival by upregulating anti-apoptotic proteins, and production of cell adhesion molecules, angiogenesis factors, and immunosuppressive molecules[2]. BCMA can be used as a promising antigen to target using a variety of immuno-therapy treatments including CART cells, for

MM patients[3]. BCMA markedly reduces plasma IgA, IgG, and IgM levels and splenic Ig heavy chain mRNA levels in mouse[4]. In BCMA−/− mice, the long-term survival of PCs is impaired, but lack of BCMA has no effect in short-lived PCs, B cell development, or early humoral immune response, and the splenic architecture and germinal centers appear intact in these BCMA-deficient mice[5]. BCMA overexpression significantly promotes in vivo growth of xenografted MM cells in murine models[6].

REFERENCES

[1]. Nobari ST, et al. B-cell maturation antigen targeting strategies in multiple myeloma treatment, advantages and disadvantages. J Transl Med. 2022 Feb 10;20(1):82.

[2]. Yu B, et al. BCMA-targeted immunotherapy for multiple myeloma. J Hematol Oncol. 2020 Sep 17;13(1):125.

[3]. Perez-Amill L, et al. Preclinical development of a humanized chimeric antigen receptor against B cell maturation antigen for multiple myeloma. Haematologica. 2021 Jan 1;106(1):173-184.

[4]. Sanchez E, et al. Soluble B-Cell Maturation Antigen Mediates Tumor-Induced Immune Deficiency in Multiple Myeloma. Clin Cancer Res. 2016 Jul 1;22(13):3383-97.

[5]. O'Connor BP, et al. BCMA is essential for the survival of long-lived bone marrow plasma cells. J Exp Med. 2004 Jan 5;199(1):91-8.

[6]. Tai YT, et al. APRIL and BCMA promote human multiple myeloma growth and immunosuppression in the bone marrow microenvironment. Blood. 2016 Jun 23;127(25):3225-36.

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