Recombinant Human APRIL/TNFSF13

Catalog Number: 884-AP

DESCRIPTION

Source Mouse myeloma cell line, NS0-derived

MHHHHHHHHHH	GGGSGGGSGGGS	IEGR	Human APRIL (Lys110-Leu250) Accession # Q8NFH7

N-terminus C-terminus

N-terminal Sequence Analysis Met

Predicted Molecular Mass 18.6 kDa

SPECIFICATIONS

SDS-PAGE	21-25 kDa, reducing conditions
Activity	Measured in a cell proliferation assay using anti-IgM stimulated mouse B cells. The ED_{50} for this effect is typically 25-150 ng/mL in the presence of goat anti-mouse IgM.
Endotoxin Level	<1.0 EU per 1 µg of the protein by the LAL method.
Purity	>95%, by SDS-PAGE under reducing conditions and visualized by silver stain.
Formulation	Lyophilized from a 0.2 µm filtered solution in PBS and NaCl with BSA as a carrier protein. See Certificate of Analysis for details.

PREPARATION AND STORAGE

Reconstitution	Reconstitute at 10 µg/mL in sterile PBS containing at least 0.1% human or bovine serum albumin.
Shipping	The product is shipped at ambient temperature. Upon receipt, store it immediately at the temperature recommended below.
Stability & Storage	**Use a manual defrost freezer and avoid repeated freeze-thaw cycles.** ● 12 months from date of receipt, -20 to -70 °C as supplied. ● 1 month, 2 to 8 °C under sterile conditions after reconstitution. ● 3 months, -20 to -70 °C under sterile conditions after reconstitution.

BACKGROUND

APRIL (a proliferation-inducing ligand), also known as TNFSF13, TALL2, TRDL1, and CD256, is a member of the TNF ligand superfamily (1). It is synthesized as a 32 kDa proprotein which is cleaved by furin in the Golgi to release the active 17 kDa soluble molecule (2-4). Secreted human APRIL, which consists almost entirely of a single TNF homology domain, shares 85% amino acid sequence identity with mouse and rat APRIL (2, 3). Both APRIL and its close relative BAFF bind and signal through the TNF superfamily receptors TACI and BCMA, while BAFF additionally functions through BAFF R (2, 5, 6). APRIL binds to heparan sulfate proteoglycans (HSPGs) independently of its binding to TACI and BCMA (6, 7). The interaction with HSPGs induces APRIL oligomerization, and this augments TACI-, or BMCA-mediated effects (7, 8). HSPGs are also critical for the tumor growth-promoting effects attributed to APRIL (6). APRIL can form bioactive heterotrimers with BAFF, and these circulate in the serum of patients with rheumatic immune disorders (10). TWE-PRIL is a bioactive hybrid protein produced by gene splicing. It consists of the intracellular domain, transmembrane segment, and stalk region of TWEAK fused to the TNF homology domain of APRIL (11). TWE-PRIL is expressed in monocytes and activated T cells and, in contrast to APRIL, is presented on the cell surface (11). APRIL enhances the proliferation and survival of plasma cells and also promotes T cell-dependent humoral responses (2, 12, 13). In the context of autoimmune disorders, however, APRIL can inhibit pathologic humoral responses as well as disease progression (14). Its expression by $CD4^+$ T cells inhibits the production of Th2 cytokines and allergic inflammation (15). APRIL levels are elevated in the serum during coronary artery disease (16), and it is also elevated in many cancers primarily due to expression by tumor-infiltrating neutrophils (4, 7, 17).

References:

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