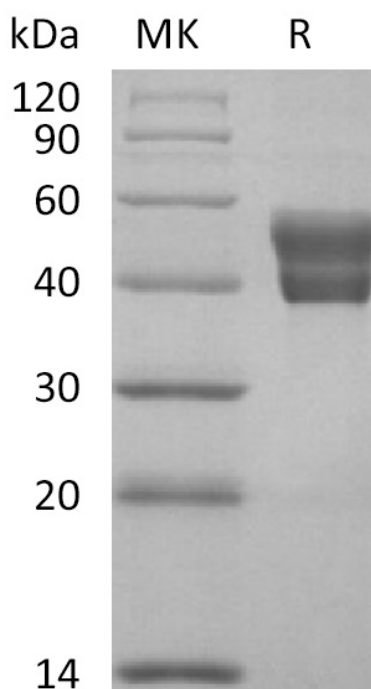


Summary

Name	BAFFR/TNFRSF13C
Purity	Greater than 95% as determined by reducing SDS-PAGE
Endotoxin level	<1 EU/μg as determined by LAL test.
Construction	Recombinant Human Tumor Necrosis Factor Receptor Superfamily Member 13C is produced by our Mammalian expression system and the target gene encoding Ser7-Ala71 is expressed with a human IgG1 Fc tag at the C-terminus.
Accession #	Q96RJ3
Host	Human Cells
Species	Human
Predicted Molecular Mass	33.4 KDa
Formulation	Lyophilized from a 0.2 μm filtered solution of PBS, pH 7.4.
Shipping	The product is shipped at ambient temperature. Upon receipt, store it immediately at the temperature listed below.
Stability&Storage	Store at ≤-70°C, stable for 6 months after receipt. Store at ≤-70°C, stable for 3 months under sterile conditions after opening. Please minimize freeze-thaw cycles.
Reconstitution	Always centrifuge tubes before opening. Do not mix by vortex or pipetting. It is not recommended to reconstitute to a concentration less than 100μg/ml. Dissolve the lyophilized protein in distilled water. Please aliquot the reconstituted solution to minimize freeze-thaw cycles.

SDS-PAGE image

Product Name: Recombinant Human BAFFR (C-Fc)
Catalog #: PHH2181



Alternative Names

BAFF R; BAFFR; BR3; CD268; TNFRSF13C

Background

Tumor necrosis factor receptor superfamily, member 13C (TNFRSF13C) also known as B-cell-activating factor receptor (BAFFR) and CD268 antigen, is a member of the tumor necrosis factor receptor superfamily. BAFF promotes the survival of B cells and is essential for B cell maturation. BAFF binds to three TNF receptor superfamily members: B-cell maturation antigen (BCMA/TNFRSF17), transmembrane activator and calcium-modulator and cyclophilin ligand interactor (TACI/TNFRSF13B) and BAFF receptor (BAFF R/BR3/TNFRSF13C). These receptors are type III transmembrane proteins that lack a signal peptide. BAFF R is highly expressed in spleen, lymph node and resting B cells. It is also expressed at lower levels in activated B cell, in resting CD4+ T cells, in thymus and peripheral blood leukocytes. BAFF knockout mice lack mature B cells. Similarly, A/WySnJ mice that are defective in BAFF-R intracellular signaling also lack mature B cells, suggesting that BAFF R is the critical receptor for BAFF during B lymphopoiesis. It has been proposed that abnormally high levels of BAFFR/TNFRSF13C (CD268) may contribute to the pathogenesis of autoimmune diseases by enhancing the survival of autoreactive B cells.

Note

For Research Use Only , Not for Diagnostic Use.