Purified Anti-Human CD268 Antibody[H353-7H12]

catalog number: **AN006560P**

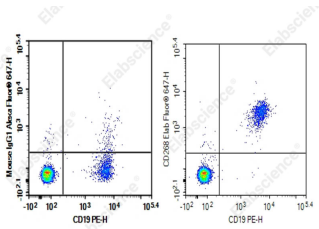
Note: *Centrifuge before opening to ensure complete recovery of vial contents.*

Description

Reactivity	Human
Immunogen	Recombinant Human CD268 protein
Host	Mouse
Isotype	Mouse IgG1, κ
Clone	H353-7H12
Purification	>98%, Protein A/G purified
Conjugation	Unconjugated
Buffer	Phosphate-buffered solution, pH 7.2, containing 0.05% non-protein stabilizer. Dialyze to completely remove the stabilizer prior to labeling.

Applications	Recommended Dilution
FCM	≤ 0.2 μg per million cells in 100 μL volume

Data



Human peripheral blood lymphocytes cell were stained with 0.2 μg Purified Anti-Human CD268 Antibody[7H12] (Right) and 0.2 μg Mouse IgG1, κ Isotype Control (Left), followed by Elab Fluor® 647-conjugated Goat Anti-Mouse IgG Secondary Antibody, then anti-Human CD19 PE-conjugated Monoclonal Antibody.

Preparation & Storage

Storage	Store at 4°C valid for 12 months or -20°C valid for long term storage, avoid freeze / thaw cycles.

Background

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B-cell activating factor (BAFF), also known as BlyS, TALL-1, TNAK, and zTNF4, is a TNF ligand superfamily member and has been designated TNFSF13B. Produced by macrophages, dendritic cells, and T lymphocytes, 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. Whereas TACI and BCMA bind BAFF and another TNF superfamily ligand, APRIL (a proliferation-inducing ligand), BAFF R selectively binds BAFF. The BAFF R extracellular domain lacks the TNF receptor canonical cysteine-rich domain (CRD) and contains only a partial CRD with four cysteine residues. Human and mouse BAFF R share 56% aa sequence identity. 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. In contrast, BCMA- or TACI-deficient mice have no major defect in B-cell development. While the function of BCMA is not defined, TACI has been shown to control B-cell homeostasis and T-cell-independent immune responses.

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