Purified Anti-Human CD95/Fas Antibody

catalog number: **E-AB-F13790P**

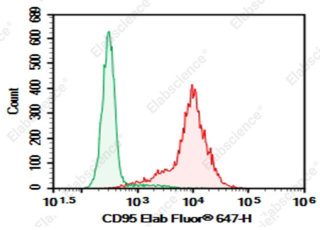
Note: *Centrifuge before opening to ensure complete recovery of vial contents.*

Description

Reactivity	Human
Immunogen	Recombinant Human CD95 protein
Host	Mouse
Isotype	Mouse IgG2a, κ
Clone	APO-1-1
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 were stained with 0.2 μg Purified Anti-Human CD95/Fas Antibody[APO-1-1] (Right) and 0.2 μg Mouse IgG2a, κ Isotype Control (Left), followed by Elab Fluor® 647-conjugated Goat Anti-Mouse IgG Secondary Antibody.

Preparation & Storage

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

Background

Toll-free: 1-888-852-8623
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CD95 (APO-1/Fas) is an important inducer of the extrinsic apoptosis signaling pathway and therapy induced apoptosis of many tumor cells has been linked to the activity of CD95. is a prototype death receptor characterized by the presence of an 80 amino acid death domain in its cytoplasmic tail. This domain is essential for the recruitment of a number of signaling components upon activation by either agonistic anti-CD95 antibodies or cognate CD95 ligand that initiate apoptosis. The complex of proteins that forms upon triggering of CD95 is called the death-inducting signaling complex (DISC). The DISC consists of an adaptor protein and initiator caspases and is essential for induction of apoptosis. CD95 is also crucial for the negative selection of B cells within the germinal center (GC). Impairment of CD95-mediated apoptosis results in defective affinity maturation and the persistence of autoreactive B-cell clones. Changes in the expression of CD95 and/or its ligand CD95L are frequently found in human cancer. The downregulation or mutation of CD95 has been proposed as a mechanism by which cancer cells avoid destruction by the immune system through reduced apoptosis sensitivity. Thus, CD95 has therefore been viewed as a tumor suppressor. CD95 has been reported to be involved in the activation of NF-kappaB, MAPK3/ERK1, MAPK8/JNK, and the alternate pathways for CTL-mediated cytotoxicity. Accordingly, this protein is implicated in the pathogenesis of various malignancies and diseases of the immune system. The CD95/CD95L system was implicated in the etiology of inflammatory bowel disease (IBD) based, primarily, on the finding that CD95 is highly expressed in the intestinal epithelial cells and that epithelial apoptosis is increased in IBD.

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