Data Sheet (Cat.No.TMAY-03042)

TargetMol

Anti-Fas/CD95 Antibody (1L304)

Product Details

Ig Type:	Rabbit IgG
Reactivity:	Mouse
Conjugation:	Unconjugated
Clone:	1L304
Purification:	Protein A

Applications

Verified Activity:	1. Immunofluorescence staining of mouse FAS in mouse splenocytes. Cells were fixed with 4% PFA, blocked with 10% serum, and incubated with rabbit anti-mouse FAS monoclonal antibody (1:60) at 37°C 1 hour. Then cells were stained with the Alexa Fluor® 594-conjugated Goat Anti-rabbit IgG secondary antibody (red). 2. Flow cytometric analysis of Mouse FAS(CD95) expression on BABL/c splenocytes. Cells were stained with purified anti-Mouse FAS(CD95), then a FITC-conjugated second step antibody. The fluorescence histograms were derived from gated events with the forward and side light-scatter characteristics of intact cells.
Application:	FCM,ICC/IF
Recommended	ICC-IF: 1:20-1:100; FCM: 1:25-1:100

Properties

Stability & Storage:	Store at 2°C-8°C for 1 month. Store at -20°C or -80°C for 12 months. Avoid repeated freeze-thaw cycles. Preservative-Free.
Shipping:	Shipping with blue ice.

Antigen Details

Immunogen:	Recombinant Protein: Mouse CD95 / APO-1 / TNFRSF6 / FAS Protein (TMPY-01426)
Antigen Species:	Mouse
Synonyms:	Fas cell surface death receptor
Biology Area:	Tumor Suppressors

Research Background

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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