FITC Anti-Rat β-2-Microglobulin Antibody[4C9]

Catalog Number: AN01428C

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

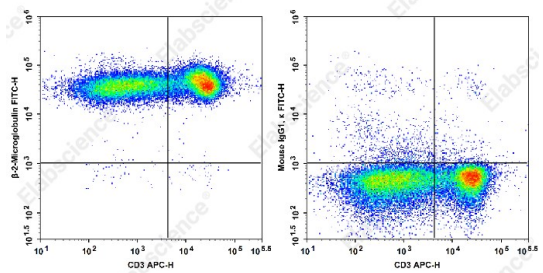
Description

Reactivity	Rat
Host	Mouse
Isotype	Mouse IgG1, κ
Clone No.	4C9
Isotype Control	FITC Mouse IgG1, κ Isotype Control[MOPC-21] [Product E-AB-F09792C]
Conjugation	FITC
Conjugation Information	FITC is designed to be excited by the Blue laser (488 nm) and detected using an optical filter centered near 530 nm (e.g., a 525/40 nm bandpass filter).
Storage Buffer	Phosphate buffered solution, pH 7.2, containing 0.09% stabilizer.

Applications — Recommended usage

FCM	Each lot of this antibody is quality control tested by flow cytometric analysis. **The amount of the reagent is suggested to be used 5 μL of antibody per test (million cells in 100 μL staining volume or per 100 μL of whole blood).** Please check your vial before the experiment. Since applications vary, the appropriate dilutions must be determined for individual use.

Data



Staining of Rat splenocytes cells with APC Anti-Rat CD3 Antibody and FITC Anti-Rat β-2-Microglobulin Antibody[4C9] (left) or FITC Mouse IgG1, κ Isotype Control (right). Total viable cells were used for analysis.

Preparation & Storage

Storage	Keep as concentrated solution. This product can be stored at 2-8°C for 24 months. Please protected from prolonged exposure to light and do not freeze.
Shipping	Ice bag

Antigen Information

Alternate Names	B2m;β2M
Uniprot ID	P07151
Gene ID	24223

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Background

4C9 was generated by immunizing BALB/c mice with soluble FcRn. Splenocytes were fused with HL-1 murine myeloma cells. 4C9 is able to bind to β-2-microglobulin as well as FcRn heterodimers in ELISA tests, can immunoprecipitate soluble FcRn heterodimers, and can also stain lipid-linked rat β-2-microglobulin (β2m-DAF), truncated FcRn heavy chain paired with β2m-DAF, and lipid-linked FcRn heavy chain paired with unaltered β-2-microglobulin.4C9 blocks binding of IgG to FcRn in vitro and FcRn blockade has been shown to have a positive effect on IgG-mediated autoimmune diseases. In support of this, 4C9 induces a transient and dose-dependent increase in the elimination of exogenously administered antimethotrexate IgG in rats.

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