APC Anti-Human CD158b/j Antibody[DX27]

Catalog Number: E-AB-F1381E

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

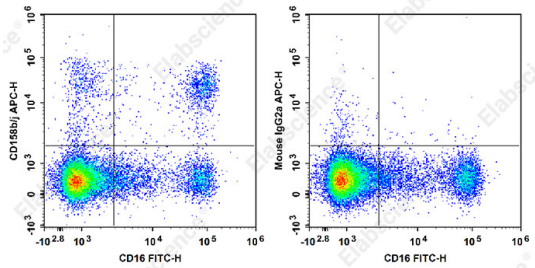
Description

Reactivity	Human
Host	Mouse
Isotype	Mouse IgG2a, κ
Clone No.	DX27
Isotype Control	APC Mouse IgG2a, κ Isotype Control[C1.18.4] [Product E-AB-F09802E]
Conjugation	APC
Conjugation Information	APC is designed to be excited by the Red (627-640 nm) laser and detected using an optical filter centered near 660 nm (e.g., a 660/20 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 normal human peripheral blood cells with FITC Anti-Human CD16 Antibody and APC Anti-Human CD158b/j Antibody[DX27] (left) or APC Mouse IgG2a, κ Isotype Control (right). Cells in the lymphocytes gate 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

Uniprot ID	P43627;P43628
Gene ID	3803;3804

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Background

CD158b is expressed on natural killer cells and a subset of T cells. It is a member of the immunoglobulin superfamily containing two immunoglobulin C2-type domains. Both variants and alternative isoforms of CD158b have been reported. The interaction of CD158b with specific HLA-C antigens on a target cell (HLA-Cw1, HLA-Cw3, HLA-Cw7 alleles, for example) inhibits cytotoxicity and prevents target cell lysis and death. The interactions between KIR and MHC class I are thought to be important in NK cell and T cell regulation following antigen stimulation. The absence of ligands for KIRs may lower the threshold for activation through activating receptors and increase inflammation and susceptibility to autoimmune disease.

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