

iF488 Anti-Mouse CD8a Antibody

Catalog Number:	201005, 201006
Size:	25 tests, 100 tests
Target Name:	CD8, CD8 alpha, T8, Lyt2, Ly-2
Regulatory Status:	RUO

PRODUCT DETAILS

Clone:	53-6.7
Application:	Flow Cytometry
Reactivity:	Mouse
Format:	iF488
Isotype:	Rat IgG2a
Antibody Type:	Monoclonal
Formulation:	Phosphate-buffered solution, pH 7.2, containing 0.09% sodium azide and 0.2% (w/v) BSA
Protein Concentration:	Supplied at a lot-specific concentration.
Storage and Handling:	The antibody solution should be stored undiluted between 2°C and 8°C, and protected from prolonged exposure to light. Do not freeze.
Recommended Usage:	For flow cytometric staining, it is recommended to use less than 0.1 µg of this reagent per 0.5-1.0 million cells in a 100 µL volume. Optimal reagent performance should be determined by titration for each specific application. iF488 has an excitation max at 491 nm and an emission max at 516 nm.
Excitation Laser:	Blue Laser (488 nm)
Isotype Control:	300202
RRID:	AB_3739019
Storage&Handling:	The antibody solution should be stored undiluted between 2°C and 8°C, and protected from prolonged exposure to light. Do not freeze.

BACKGROUND INFORMATION

CD8 is a cell surface glycoprotein that plays a critical role in cellular immunity. It is most commonly expressed on cytotoxic T lymphocytes (CD8⁺ T cells), although lower levels can also be found on subsets of natural killer (NK) cells and dendritic cells.

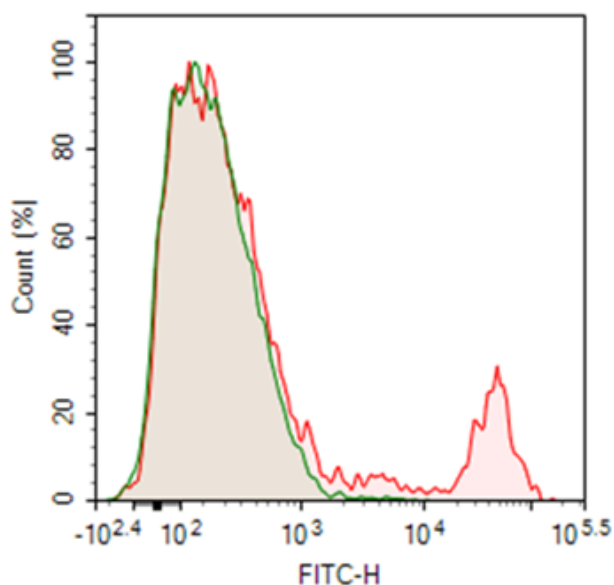
Structurally, CD8 exists either as a homodimer of two CD8α chains (CD8αα) or, more commonly on T cells, as a heterodimer composed of CD8α and CD8β chains (CD8αβ). Each chain contains an extracellular immunoglobulin-like domain, a transmembrane region, and a short cytoplasmic tail that associates with intracellular signaling molecules. Functionally, CD8 acts as a co-receptor for the T cell receptor (TCR) during antigen recognition. Its primary ligand is major histocompatibility complex class I (MHC I), which

is expressed on nearly all nucleated cells. During immune surveillance, the TCR recognizes peptide antigens presented by MHC I, while CD8 binds to a conserved region of the MHC I molecule. This interaction stabilizes the TCR-peptide-MHC complex and brings the Src-family kinase Lck into proximity with the TCR signaling machinery, thereby enhancing signal transduction and lowering the threshold for T cell activation. Once activated, CD8⁺ T cells differentiate into cytotoxic effector cells capable of directly killing infected or malignant cells. They mediate target cell death primarily through the release of perforin and granzymes, which induce apoptosis, as well as through engagement of death receptor pathways such as Fas-Fas ligand interactions. CD8⁺ T cells also secrete cytokines, including interferon- γ (IFN- γ) and tumor necrosis factor- α (TNF- α), which further shape immune responses and inhibit pathogen replication.

CD8⁺ T cells are essential for protection against viral infections and for immune surveillance against cancer. However, their dysregulation contributes to disease. Insufficient CD8⁺ T cell responses can result in chronic viral infections or tumor immune evasion, while excessive or misdirected activity can cause tissue damage and contribute to autoimmune diseases, such as type 1 diabetes and multiple sclerosis. In chronic infections and cancer, persistent antigen exposure can drive CD8⁺ T cell exhaustion, characterized by reduced effector function and sustained expression of inhibitory receptors.

In therapeutics, CD8⁺ T cells are central to modern immunotherapy. Cancer treatments such as immune checkpoint inhibitors aim to reinvigorate exhausted CD8⁺ T cells, while adoptive cell therapies, including CAR-T and TCR-engineered T cells, harness or enhance CD8⁺ cytotoxic activity. CD8⁺ T cell responses are also a key goal of antiviral vaccines, underscoring their importance in both disease control and therapeutic intervention.

PRODUCT DATA



Mouse splenocytes were stained with iF488 Anti-Mouse CD8a clone 53-6.7 (color-filled histogram) or an isotype control (gray histogram).

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