

iF647Anti-Human CD127 (IL-7R α) Antibody

Catalog Number:	104909, 104910
Size:	25 tests, 100 tests
Target Name:	CD127, IL-7 receptor α chain, IL-7R α
Regulatory Status:	RUO

PRODUCT DETAILS

Clone:	127AM1
Application:	Flow Cytometry
Reactivity:	Human
Format:	iF647
Isotype:	Mouse IgG1
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&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 5 μ L of this reagent per 0.5-1.0 million cells in a 100 μ L volume. Optimal reagent performance should be determined by titration for each specific application. iF647 has an excitation max at 656 nm and an emission max at 670 nm.
Excitation Laser:	Red Laser (633 nm)
Isotype Control:	301413
RRID:	AB_3738671

BACKGROUND INFORMATION

CD127 is the α chain of the interleukin-7 receptor (IL-7R α), a critical cytokine receptor involved in lymphocyte development, survival, and homeostasis. CD127 is expressed on multiple immune cell types, including naïve and memory CD4⁺ and CD8⁺ T cells, innate lymphoid cells (ILCs), and early B cell progenitors, while it is characteristically low or absent on regulatory T cells (Tregs). Because of this expression pattern, CD127 is widely used as a phenotypic marker to distinguish functional T cell subsets.

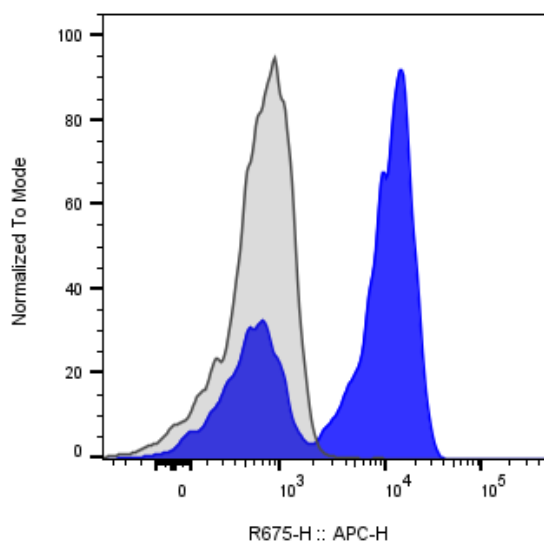
Structurally, CD127 is a type I transmembrane glycoprotein belonging to the class I cytokine receptor family. It consists of an extracellular domain containing conserved cytokine receptor motifs, a single transmembrane region, and a cytoplasmic tail that lacks intrinsic kinase activity. CD127 pairs with the common gamma chain (γ c, CD132) to form the functional IL-7 receptor complex. Upon ligand binding, associated Janus kinases (JAK1 on CD127 and JAK3 on γ c) are activated, leading to downstream signaling through STAT5 and other pathways that regulate cell survival and proliferation.

The primary ligand for CD127 is interleukin-7 (IL-7), a stromal cell-derived cytokine essential for T cell development in the thymus and for the maintenance of peripheral T cell pools. IL-7 signaling promotes lymphocyte survival by upregulating anti-apoptotic molecules such as BCL-2 and supports metabolic fitness and long-term immune competence. Tight regulation of CD127 expression ensures appropriate responsiveness to IL-7 and prevents excessive or dysregulated lymphocyte expansion.

CD127 is implicated in several diseases. Reduced CD127 expression on T cells is observed in chronic infections, cancer, and autoimmune diseases, often reflecting persistent immune activation or exhaustion. Genetic polymorphisms in the IL7R gene have been linked to susceptibility to autoimmune disorders, most notably multiple sclerosis. In contrast, aberrant IL-7/IL-7R signaling has been implicated in certain leukemias and lymphomas, where it may contribute to malignant cell survival and proliferation.

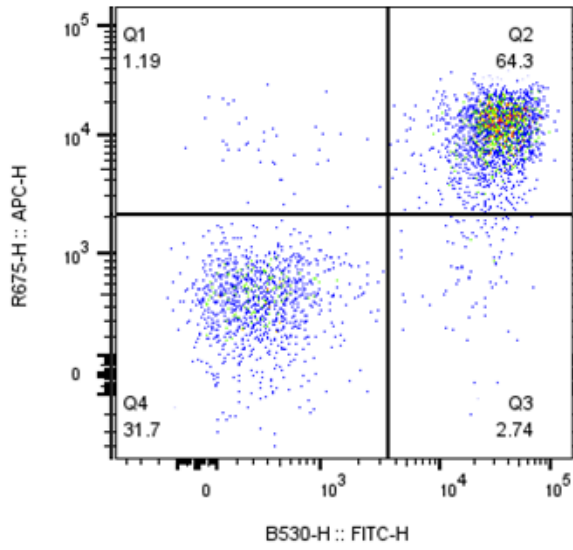
Therapeutically, CD127 and the IL-7 pathway are active areas of clinical interest. Recombinant IL-7 has been explored to enhance immune reconstitution in settings such as lymphopenia, HIV infection, and cancer immunotherapy, where boosting T cell numbers and function is desirable. Conversely, therapeutic strategies targeting CD127 signaling are being investigated for autoimmune and inflammatory diseases, aiming to dampen pathogenic T cell responses. In research and cell therapy, CD127 is also routinely used as a marker to identify memory T cells and to exclude regulatory T cells during immune profiling and cell product manufacturing.

PRODUCT DATA



Human peripheral blood lymphocytes stained with either iF647 Anti-human CD127 clone 127AM1 (blue histogram) or an isotype control (gray histogram).

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Human peripheral blood lymphocytes co-stained with iF647 Anti-human CD127 clone 127AM1 and FITC anti-human CD3 clone OKT3.