www.innocyto.com

InnoCyto Inc.

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PE Anti-Human IFNg Antibody

Catalog Number:	110608, 110609
Size:	25 tests, 100 tests
Target Name:	IFNg, IFN-g, IFN-gamma, Interferon-γ
Regulatory Status:	RUO

PRODUCT DETAILS

Clone:	4S.B3
Application:	Intracellular Flow Cytometry
Reactivity:	Human
Format:	PE
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 µL 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. PE has an excitation max at 565 nm and an emission max at 575 nm.
Excitation Laser:	Blue Laser (488 nm) Green/Yellow laser (532/561nm)
Isotype Control:	301407
RRID:	AB_3738949

BACKGROUND INFORMATION

Interferon-gamma (IFN-γ) is a cytokine with key immunoregulatory and antimicrobial functions, playing a pivotal role in both innate and adaptive immunity. It is primarily produced by activated T lymphocytes, especially $CD4^+$ Th1 cells and $CD8^+$ cytotoxic T cells, as well as by natural killer (NK) cells. IFN-γ serves as a crucial mediator of macrophage activation, enhances antigen presentation, and orchestrates the immune response against intracellular pathogens such as viruses, bacteria, and certain parasites. It also contributes to inflammation and immune surveillance by modulating the differentiation and function of various immune cells.

Structurally, human IFN-γ is a homodimeric glycoprotein consisting of two identical subunits of approximately 17 kDa each, yielding a biologically active molecule of about 34 kDa. Each subunit comprises an α-helical structure characteristic of the class II cytokine family. The dimer binds to its specific cell surface receptor complex, composed of two IFN-γ receptor 1 (IFNGR1) and two IFN-γ receptor 2 (IFNGR2) chains, forming a tetrameric signaling unit. This receptor engagement activates the Janus kinase (JAK)-signal transducer and activator of transcription (STAT) pathway, particularly JAK1, JAK2, and STAT1, leading to transcription of genes

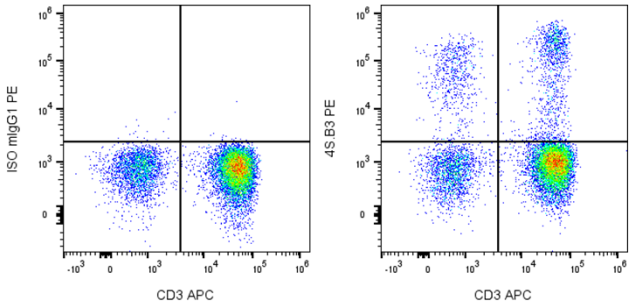
involved in antiviral defense, immune regulation, and cell cycle control.

The major ligands for IFN-γ are its receptors, IFNGR1 and IFNGR2, which are expressed on nearly all nucleated cells. Interaction with this receptor complex induces a broad range of immunomodulatory effects, including upregulation of major histocompatibility complex (MHC) class I and II molecules and enhancement of antigen presentation, which heightens immune recognition and cytotoxic responses.

In disease, dysregulation of IFN-γ signaling can lead to pathological consequences. Deficiency or impaired signaling causes increased susceptibility to intracellular infections such as mycobacterial and viral diseases. On the other hand, excessive IFN-γ activity contributes to chronic inflammation and autoimmunity, as seen in conditions like systemic lupus erythematosus and rheumatoid arthritis. In cancer, IFN-γ plays a dual role; it can promote anti-tumor immunity by enhancing immune cell activation but can also drive immune evasion under chronic exposure.

Therapeutically, recombinant IFN-γ is used in treating chronic granulomatous disease and severe osteopetrosis, where it enhances macrophage function. Ongoing research explores its potential in cancer immunotherapy and as an adjuvant to improve vaccine efficacy. Modulating IFN-γ signaling, either by boosting or suppressing its effects, continues to offer promising avenues for controlling immune-mediated diseases and improving therapeutic outcomes.

PRODUCT DATA



PMA/Ionomycin-stimulated human peripheral blood lymphocytes stained with APC Anti-human CD3 and either PE Anti-Human IFNɣ clone 4S.B2 (right panel) or an isotype control (left panel).