

1P-706-T100

## Monoclonal Antibody to IFN-gamma Phycoerythrin (PE) conjugated (100 tests)

<b>Clone:</b>	4S.B3
<b>Isotype:</b>	Mouse IgG1
<b>Specificity:</b>	The mouse monoclonal antibody 4S.B3 recognizes IFN-gamma, a 16-25 kDa cytokine produced by activated Th1 cells and NK cells. Binds both glycosylated and non-glycosylated protein.
<b>Regulatory Status:</b>	RUO
<b>Immunogen:</b>	Interferon gamma derived from human leukocytes
<b>Species Reactivity:</b>	Human, Non-Human Primates
<b>Preparation:</b>	The purified antibody is conjugated with R-Phycoerythrin (PE) under optimum conditions. The conjugate is purified by size-exclusion chromatography and adjusted for direct use. No reconstitution is necessary.
<b>Storage Buffer:</b>	The reagent is provided in stabilizing phosphate buffered saline (PBS) solution containing 15mM sodium azide.
<b>Storage / Stability:</b>	Store in the dark at 2-8°C. Do not freeze. Avoid prolonged exposure to light. Do not use after expiration date stamped on vial label.
<b>Usage:</b>	The reagent is designed for Flow Cytometry analysis of human blood cells using 10 µl reagent / 100 µl of whole blood or 10 <sup>6</sup> cells in a suspension. The content of a vial (1 ml) is sufficient for 100 tests.
<b>Expiration:</b>	See vial label
<b>Lot Number:</b>	See vial label
<b>Background:</b>	<p>The Interferon gamma (IFN-gamma; 16-25 kDa) is an important regulator of the immune response, produced in activated Th1 cells and NK cells, particularly in response to IL-2, TNF-alpha and IL-12; its production is suppressed by IL-4, IL-10, and TGF-beta. The producing of IFN-gamma is activated by specific antigens or mitogens through the T cell antigen receptor. IFN-gamma polypeptide forms: 40-60 kDa forms are observable under non-denaturing conditions as dimers and trimers; 20 kDa and 25 kDa forms exist due to variable glycosylation. IFN-gamma belongs to the type II interferons, also called immune IFN.</p> <p>IFN-gamma shows antiviral activity and has important immunoregulatory functions. It is a potent activator of macrophages and had antiproliferative effects on transformed cells. IFN-gamma plays an important role in regulating B cell differentiation by simultaneously stimulating class switch recombination to the IgG3 and IgG2a isotypes while repressing class switch recombination to the IgE and IgG1 isotypes. It also appears to promote antigen presentation by B cells through its effects on MHC. Binding of IFN-gamma to its receptor increases the expression of class I MHC on all somatic cells. It also enhances the expression of class II MHC on antigen-presenting cells. IFN-gamma is the major means by which T cells activate macrophages, increasing their ability to kill bacteria, parasites, and tumours. The activation of macrophages by IFN-gamma is essential for the elimination of bacteria that replicate within the phagosomes of macrophages (f.e. Mycobacteria and Listeria monocytogenes). IFN-gamma can potentiate the high antiviral and antitumor effects of the type I interferons (IFN-alpha, IFN-beta). IFN-gamma may also activate neutrophils and NK cells.</p>

**For laboratory research only, not for drug, diagnostic or other use.**

**Antibodies****References:**

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\*Brattig NW, Lepping B, Timmann C, Büttner DW, Marfo Y, Hamelmann C, Horstmann RD: Onchocerca volvulus-exposed persons fail to produce interferon-gamma in response to O. volvulus antigen but mount proliferative responses with interleukin-5 and IL-13 production that decrease with increasing microfilarial density. *J Infect Dis.* 2002 Apr 15;185(8):1148-54.

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\*David A. Kosub, Ginger Lehrman, Jeffrey M. Milush, Dejiang Zhou, Elizabeth Chacko, Amanda Leone, Shari Gordon, Guido Silvestri, James G. Else, Philip Keiser, Mamta K. Jain, Donald L. Sodora: Gamma/Delta T-Cell Functional Responses Differ after Pathogenic Human Immunodeficiency Virus and Nonpathogenic Simian Immunodeficiency Virus Infections. *J Virol.* 2008 February; 82(3): 1155-1165.

\*Erik L. Brincks, Tamara A. Kucaba, Kevin L. Legge, Thomas S. Griffith: Influenza-induced expression of functional TNF-related apoptosis-inducing ligand (TRAIL) on human PBMC. *Hum Immunol.* 2008 October; 69(10): 634-646.

\*Coles AJ, Wing M, Smith S, Coraddu F, Greer S, Taylor C, Weetman A, Hale G, Chatterjee VK, Waldmann H, Compston A: Pulsed monoclonal antibody treatment and autoimmune thyroid disease in multiple sclerosis. *Lancet.* 1999 Nov 13;354(9191):1691-5.

\*And many other.

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