

FITC Anti-Human TNF- α Antibody

Catalog Number:	113505, 113506
Size:	25 tests, 100 tests
Target Name:	TNF- α , TNF-alpha, Tumor necrosis factor- α , Macrophage cytotoxic factor (MCF)
Regulatory Status:	RUO

PRODUCT DETAILS

Clone:	Infliximab
Application:	Flow Cytometry
Reactivity:	Human
Format:	FITC
Isotype:	Human 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. FITC has an excitation max at 493 nm and an emission max at 525 nm.
Excitation Laser:	Blue Laser (488 nm)
Isotype Control:	301205

BACKGROUND INFORMATION

TNF α (tumor necrosis factor alpha) is a potent pro-inflammatory cytokine that plays a central role in immune regulation, host defense, and inflammation. It is primarily produced by activated macrophages, T cells, and other immune cells in response to infection, injury, or immune stimulation. TNF α mediates a wide range of biological effects, including induction of fever, activation of endothelial cells, promotion of leukocyte recruitment, and regulation of cell survival, apoptosis, and necrosis.

Structurally, TNF α is initially synthesized as a type II transmembrane protein (membrane-bound TNF α) that forms stable homotrimers. It can be cleaved by the metalloprotease TACE (ADAM17) to release a soluble trimeric form. Both membrane-bound and soluble TNF α are biologically active, although they may have distinct functional roles. The trimeric structure is essential for binding and activating its receptors.

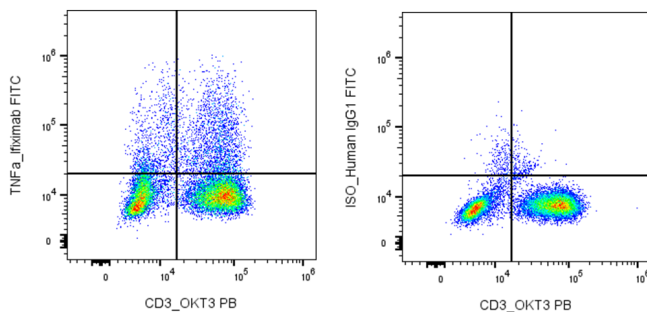
The primary receptors for TNF α are TNFR1 (p55, CD120a) and TNFR2 (p75, CD120b). TNFR1 is widely expressed and contains a death domain that can trigger apoptosis or activate NF- κ B signaling pathways, leading to inflammation and cell survival. TNFR2 is more restricted in expression and is mainly involved in immune regulation and tissue repair. The interaction between TNF α and its

receptors initiates complex signaling cascades that determine cellular outcomes.

In disease, TNF α is a key driver of chronic inflammatory and autoimmune conditions, including rheumatoid arthritis, inflammatory bowel disease, psoriasis, and ankylosing spondylitis. Excessive or dysregulated TNF α production contributes to tissue damage and disease progression. TNF α is also involved in cancer, infection, and sepsis, where it can have both protective and pathological effects.

Therapeutically, TNF α is one of the most successfully targeted cytokines in modern medicine. Anti-TNF biologics, such as monoclonal antibodies and receptor fusion proteins, have revolutionized the treatment of inflammatory diseases by neutralizing TNF α activity. These therapies reduce inflammation and improve clinical outcomes, although they may increase susceptibility to infections due to immune suppression.

PRODUCT DATA



Human peripheral lymphocytes stimulated with PMA/Ionomycin were stained with PB Anti-Human CD3 clone OKT3, fixed, permeabilized and then stained with FITC Anti-Human TNF α biosimilar Infliximab (left) or an isotype control (right).

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