

Th1/Th2/Th17 Cytokines ELISA Kit | Th1/Th2/Th17 elisa kit

Multiplex Human Cytokine ELISA Kit (Th1/Th2/Th17 Cytokines)

CATALOG #
AAA14622

PRODUCT SYNONYMS

Th1/Th2/Th17 Cytokines; Multiplex Human Cytokine ELISA Kit (Th1/Th2/Th17 Cytokines); Th1/Th2/Th17 Cytokines (IFN-gamma; IL-2; IL-4; IL-10; IL-13; IL-17; IL-22 and TNF-alpha); Th1/Th2/Th17 elisa kit

PRODUCT SPECIFICATIONS

Samples	Cell culture supernatant and other biological samples
Assay Type	Quantitative Sandwich
Preparation and Storage	SUBSTRATE B (20% ACETONE) Handling Procedures and Equipment - Wear proper personal protective equipments. Keep away from heat, sparks and flame. Do not ingest. Do not breathe gas/ fumes/ vapor/ spray. Do not get in eyes, on skin or on clothing. Storage Requirements - 2-10 degree C STOP SOLUTION (2N SULFURIC ACID) Handling Procedures and Equipment - Wear proper personal protective equipments. Avoid generating mist. Do not ingest. Do not breathe gas/ fumes/ vapor/ spray. Do not get in eyes, on skin or on clothing. Storage Requirements - 2-10 degree C

**Related Product Information for
Th1/Th2/Th17 elisa kit**

Background: T helper cells (Th cells) are a diversified group of CD4+ T cells that play an essential role in immune-response through cytokine secretion and cell to cell contact (adhesion molecule expression). The importance of Th cells is manifested by the acquired immunodeficiency in HIV patients whose CD4+ T cells are selectively infected and destroyed by HIV virus. The function of Th cells is multifaceted, including stimulation of CD8+ cytotoxic cells, promoting B cell proliferation, regulating antibody subclass switching, and activation of macrophages and other cells in innate immune system. T helper cells are originated from Naïve CD4+ T cells, which are generated in bone marrow and differentiate into effector Th cells in peripheral lymphoid organs and blood upon contact with cognate antigen that matches with the complementarity-determining region on T cell receptor. Th cells were classified into T helper 1 cells (Th1) and T helper 2 cells (Th2) in the mid-1980s based on their unique cytokine secretion patterns and their roles in cellular and humoral immunity. Th1 cells secrete IFN-gamma as their signature cytokine, and are also the primary producer of IL-2. Th2 cells fail to make IFN-gamma, but produce IL-4, IL-5, and IL-13 as their signature cytokines. Th1 cells are needed in host defense against intracellular bacteria and virus, while Th2 cells are essential for humoral responses against extracellular parasites. Cytokines play important roles in the development and maintenance of T helper cell lineage. In the presence of IL-12, Naïve CD4+ cells tend to differentiate into Th1, whereas IL-4 stimulates Th2 development. Interestingly, cytokines produced by one Th subset tends to inhibit the development of other Th subset(s). For example, IL-10 secreted by Th2 can counteract the effect of IL-12 through suppressing Th1 and stimulating Th2 differentiation. In 2005, Harrington et al described a new subset of IL-17 secreting CD4+ T cells as T helper 17 (Th17) cells, which is involved with autoimmunity and clearance of extracellular bacteria and fungi. It was observed that Th17 cells can be induced from Naïve CD4+ T cells by IL-23 in the absence of IFN- γ and IL-4. More recently, T follicular helper cells (Tfh), regulatory T cells (Treg), T helper 9 cells (Th9) and T helper 22 cells (Th22) were proposed. IL-2 is one of the key cytokines that drive adaptive immunity. It is secreted by Naïve CD4+ cells immediately after T cell receptor is activated through binding with antigens presented on MHC by antigen-presenting cells. IL-2 stimulates Th1 differentiation, clone expansion and acts as autocrine to stimulate its own production. It also promotes the survival of antigen specific memory T cell, and stimulates the maturation of regulatory T-cells. The cytokine also influences the activity of B cells, CD8+ T cells, gamma delta T cells, NK cells and LAK cells. IL-10 is expressed by a variety of cell types and plays an important role in inflammatory and immune responses. It down-regulates the expression of Th1 cytokines. IL-10 also suppresses the production of pro-inflammatory cytokines by monocytes and neutrophils, down-regulates the expression of activating and co-stimulatory molecules on monocytes and dendritic cells, and stimulates the growth of B cells and mast cells. Produced by Th1, CD8+ T cells and natural killer (NK) cells, IFN-gamma is known to stimulate Naïve CD4+ T cells to differentiate into Th1 cells. It is both an inhibitor of viral replication and a regulator of numerous immunological functions. IFN-gamma has been reported to induce its own expression. IFN-gamma has documented antiviral, antiprotozoal and immunomodulatory activities. The immunomodulatory effects of IFN-gamma are extensive and diverse. In monocyte/ macrophages, IFN-gamma increases expression of class II MHC antigens; increases the production of IL-1, platelet-activating factor, H₂O₂, and pterin, protects monocytes against LAK cell-mediated lysis; and down-regulates IL-8 mRNA expression. IFN-gamma selectively enhances IgG2a secretion by LPS-stimulated B cell activation. IFN-gamma has been shown to up-regulate ICAM-1, but not E-selectin or VCAM-1 expression on endothelial cells. IFN-gamma has also been reported to implicate in the development of a cholinergic phenotype in embryonic septal neurons. IL-4 was initially characterized as a B cell stimulatory factor (BSF-1) for its crucial role in Th2 cell differentiation, B cell expansion and differentiation into plasma cells. It was subsequently revealed that IL-4 is a pleiotropic cytokine with multiple immune response modulating functions on diverse cell types. IL-4 induces antibody class switching to IgE, up-regulates MHC class II production and plays a role in airway allergy. IL-4 stimulates protective immunity against parasites. It is also reported that IL-4 is implicated with metastasis. IL-4 is produced by naïve CD4+ T cells, Th2 cells, fetal thymocytes, CD8+ T cells, mast cells and basophils. IL-4 exhibits approximately 25% amino acid sequence homology to IL-13 that shares a number of biological functions with IL-4. Human IL-4 gene, composed of four exons and three introns, have been localized to a chromosome that contains tandem-arranged genes for IL-3, IL-5, IL-9, IL-13 and GM-CSF. IL-13 is secreted mainly by activated T helper 2 cells. IL-13 binds to IL-4R α in addition to IL-13R α 1 and IL-13R α 2. The function of IL-13 partially overlaps with IL-4. Both IL-4 and IL-13 link to signal transducer and activator of transcription 6 (STAT6) through the receptor activation. IL-13 can induce B-cell proliferation and IgE class switching. However, IL-13 is less competent in this function comparing with IL-4. Unlike IL-4, IL-13 does not play an important role in hematopoietic cell shift, such as naïve T helper differentiation to T helper 2. IL-13 is also associated with many features of the allergic airway diseases, including mucus hypersecretion, goblet metaplasia, chemokine induction and recruitment of effector cells. All of them contribute to airway obstruction. Polymorphisms in IL-13 gene have been found to link to increased eosinophil count, serum total IgE and high risk of asthma. It is reported that IL-13 signaling is significant in inducing physiological changes to non-immune cells in parasitized organs. IL-17A is the proto-type of a newly discovered pro-inflammatory cytokine family which consists of IL-17A, IL-17B, IL-17C, IL-17D, IL-17E (IL-25) and IL-17F. All the IL-17s have similar structure with four highly conserved cysteine residues and same 3 dimensional structures, which distinguish this family from other cytokines. IL-17 is produced by a group of CD4+ T help cells termed Th17 cells. In addition to previous discovered Th1 and Th2 cells, the Th17 cells consist in the third group of T helper cells. IL-23, a growth and stabilization factor was the first cytokine that was found to involve with the development of Th 17 cells. Later on, other cytokines including TGF- β , IL-6, and IL-21, were also found to contribute to the procedure. The transcription factors implicated in the procedure were identified as STAT3, ROR γ t, and ROR α . IL-17, like IFN-gamma, is a potent mediator of delayed-type reaction. IL-17 induces the secretion of many other pro-inflammatory cytokines and chemokines such as IL-6, IL-8, GM-CSF, G-CSF, TGF- β , TNF- α , GRO- α , MCP-1, and stimulates the expression of NF- κ B, mitogen activated protein kinase, and prostaglandins from many cell types. By stimulating cytokine production and recruiting monocytes

For Research Use Only. Not for use in diagnostic or therapeutic procedures. Small volumes of product may become entrapped in the seal of the vial during shipment and storage; if necessary, briefly centrifuge the vial on a tabletop centrifuge to dislodge any liquid in the cap.

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Online datasheet: <https://www.aaabiotech.com/elisa-kits/th1-th2-th17-cytokines/14622>