

## anti-human CD4 no azide

Monoclonal antibody IT4 to human CD4

Cat-No: **21459040**

100 µg in 100 µl

**Clone:** IT4

**Specificity** The antibody IT4 reacts with CD4 antigen which is expressed on a subset of T lymphocytes ("helper" T-cells) and also on monocytes, tissue macrophages and granulocytes.

**Isotype subclass:** Mouse IgG1k

**Form:** Purified by protein affinity chromatography.

**Expiration date:** The reagent is stable until the expiry date stated on the vial label

**Physical state:** Liquid

**Buffer/Additives/Preservative:** PBS (sterile), (pH 7.2)

**Storage conditions:** Aliquot and store at -20°C. Avoid freeze/thaw cycles. Should be handled under aseptic conditions.

**Application:** Functional application

**References:**

\*Millan J, Cerny J, Horejsi V, Alonso MA.: Tissue Antigens. 1999 Jan;53(1):33-40.

**Background:** CD4 is a single chain transmembrane glycoprotein and belongs to immunoglobulin supergene family. In extracellular region there are 4 immunoglobulin-like domains (1 Ig-like V-type and 3 Ig-like C2-type). Transmembrane region forms 25 aa, cytoplasmic tail consists of 38 aa. Domains 1,2 and 4 are stabilized by disulfide bonds. The intracellular domain of CD4 is associated with p56Lck, a Src-like protein tyrosine kinase. It was described that CD4 segregates into specific detergent-resistant T-cell membrane microdomains. Extracellular ligands: MHC class II molecules (binds to CDR2-like region in CD4 domain 1); HIV envelope protein gp120 (binds to CDR2-like region in CD4 domain 1); IL-16 (binds to CD4 domain 3), Human seminal plasma glycoprotein gp17 (binds to CD4 domain 1), L-selectin - Intracellular ligands: p56Lck CD4 is a co-receptor involved in immune response (co-receptor activity in binding to MHC class II molecules) and HIV infection (human immunodeficiency virus; CD4 is primary receptor for HIV-1 surface glycoprotein gp120). CD4 regulates T-cell activation, T/B-cell adhesion, T-cell differentiation, T-cell selection and signal transduction. Defects in antigen presentation (MHC class II) cause dysfunction of CD4+ T-cells and their almost complete absence in patients blood, tissue and organs (SCID immunodeficiency).

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