SONY

Product Data Sheet

PE anti-human CD152 (CTLA-4)

Catalog # / Size: 2349530 / 100 tests
2349525 / 25 tests

Clone: L3D10

Isotype: Mouse IgG1, κ

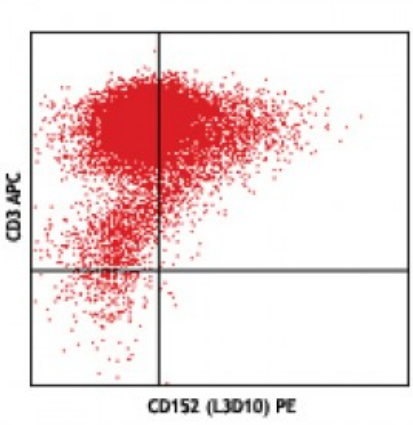
Immunogen: Extracellular domain of human CTLA-4 and human IgG1 Fc fusion protein

Reactivity: Human

Preparation: The antibody was purified by affinity chromatography and conjugated with PE under optimal conditions. The solution is free of unconjugated PE and unconjugated antibody.

Formulation: Phosphate-buffered solution, pH 7.2, containing 0.09% sodium azide and 0.2% (w/v) BSA (origin USA).

Concentration: Lot-specific



PHA-stimulated human peripheral blood mononuclear cells (day-3) stained with L3D10 PE and CD3 APC

Applications:

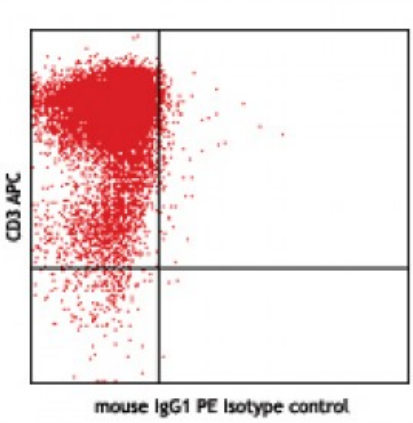
Applications: Flow Cytometry

Recommended Usage: Each lot of this antibody is quality control tested by immunofluorescent staining with flow cytometric analysis. **Test size products are transitioning from 20 microL to 5 microL per test**. Please check your vial or your CoA to find the suggested use of this reagent per million cells in 100 microL staining volume or per 100 microL of whole blood. It is recommended that the reagent be titrated for optimal performance for each application.

Application Notes: **ELISA Detection:** The biotinylated L3D10 antibody is useful as the detection antibody in a sandwich ELISA assay, when used in conjunction with the purified A3.6B10.G1 antibody (Cat. No. 525401) as the capture antibody and recombinant human CTLA-4 (Cat. No. 591909) as the standard.

Flow Cytometry: The fluorochrome-labeled L3D10 antibody is useful for immunofluorescent staining and flow cytometric analysis to identify CTLA-4-producing cells within mixed cell populations.

Note: For testing human soluble CTLA-4 in serum, plasma or cell culture supernatant, LEGEND MAX™ Human Soluble CTLA-4 ELISA Kit with Pre-



PHA-stimulated human peripheral blood mononuclear cells (day-3) stained with CD3 APC and mouse IgG1, κ PE isotype control

coated Plates (Cat. No. 437407 & 437408) are specially developed and recommended.

Additional reported applications (for the relevant formats) include: Blocking of CTLA-4/B7-1 interaction and blocking of CTLA-4-mediated inhibitory function to promote T cell expansion[1,2].

Application References:

1. May K, *et al.* 2005. *Blood* 105:1114. (Block)
2. Lute K, *et al.* 2005. *Blood* 106:3127. (Block)

Description: CD152, also known as Cytotoxic T-Lymphocyte Antigen 4 (CTLA-4), is a 33 kD member of the immunoglobulin superfamily. It is transiently expressed on activated T cells. CTLA4 is expressed on the surface of helper T cells and transmits an inhibitory signal to T cells. Regulatory T cells express high levels of CTLA-4. CTLA-4 (CD152) is similar to CD28 in amino acid sequence, structure, and genomic organization. Whereas CD28 delivers a costimulatory signal in T cell activation, CTLA-4 negatively regulates cell-mediated immune responses through interaction with CD80 (B7-1) and CD86 (B7-2) present on antigen presenting cells (APC). CTLA-4 is thought to play a role in the induction and maintenance of immunological tolerance as well as the development of protective immunity and thymocyte regulation.

Mutations in the CTLA-4 gene have been associated with various autoimmune diseases, such as systemic lupus erythematosus, insulin-dependent diabetes mellitus, and other autoimmune diseases. A transcript of the CTLA-4 gene that may represent a native soluble form of CTLA-4 (sCTLA-4) showed that eleven of twenty patients with autoimmune thyroid disease (ATD) had a high concentration of sCTLA-4, whereas only 1 of 30 apparently healthy volunteers contained measurable levels. sCTLA-4 immunoreactivity was inhibited by its binding to B7.1, suggesting that sCTLA-4 is a functional receptor. sCTLA4 also plays a role in the initial immune response to infection of immune cells by HIV, along with the CD-1 pathway and others.

Antigen References:

1. Barclay N, *et al.* The Leukocyte Antigen FactsBook. Academic Press Inc. San Diego.
2. Kuiper H, *et al.* 1995. *J. Immunol.* 155:1776.
3. Lindsten T, *et al.* 1993. *J. Immunol.* 151:3489.
4. Mort