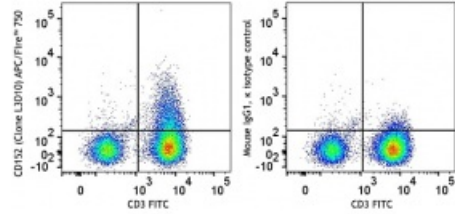


APC/Fire™ 750 anti-human CD152 (CTLA-4)

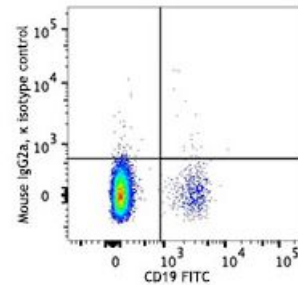
Catalog # / 2349650 / 100 tests
Size: 2349645 / 25 tests
Clone: L3D10
Isotype: Mouse IgG1, κ
Immunogen: Extracellular domain of human CTLA-4 and human IgG1 Fc fusion protein
Reactivity: Human
Preparation: The antibody was purified by affinity chromatography and conjugated with APC/Fire™ 750 under optimal conditions.
Formulation: Phosphate-buffered solution, pH 7.2, containing 0.09% sodium azide and 0.2% (w/v) BSA (origin USA).
Workshop Number: 700 under optimal conditions.
Concentration: Lot-specific



Cell Activation Cocktail (w/o Brefeldin A) (Cat. No. 423301) stimulated (4 hrs) human peripheral blood lymphocytes were stained with CD3 FITC and CD152 (CTLA-4, clone L3D10) APC/Fire™ 750 (left) or Mouse IgG1 APC/Fire™ 750, κ isotype control (right).

Applications:

Applications: Flow Cytometry
Recommended Usage: Each lot of this antibody is quality control tested by immunofluorescent staining with flow cytometric analysis. For flow cytometric staining, the suggested use of this reagent is 5 µl per million cells in 100 µl staining volume or 5 µl per 100 µl of whole blood.



* APC/Fire™ 750 has a maximum excitation of 650 nm and a maximum emission of 787 nm.

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-coated Plates (Cat. No. 437407) 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}.

This clone has been tested in-house and determined to not be suitable for applications in immunohistochemistry of frozen or paraffin-embedded tissue sections (IHC-F or IHC-P).

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** 1. Barclay N, *et al.* The Leukocyte Antigen FactsBook. Academic Press Inc. San Diego.
- References:** 2. Kuiper H, *et al.* 1995. *J. Immunol.* 155:1776.
3. Lindsten T, *et al.* 1993. *J. Immunol.* 151:3489.
4. Morton P, *et al.* 1996. *J. Immunol.* 156:1047.