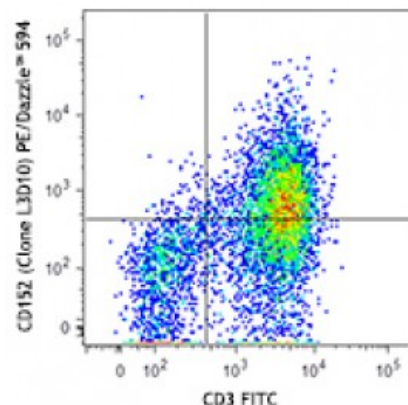


**PE/Dazzle™ 594 anti-human CD152 (CTLA-4)**

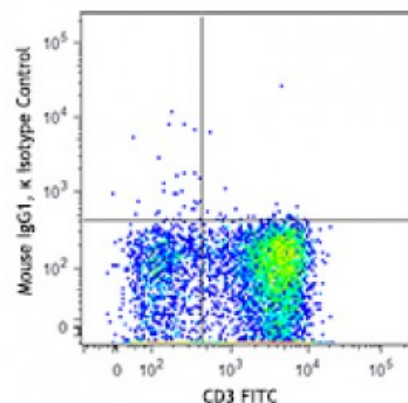
<b>Catalog # / Size:</b>	2349605 / 25 tests 2349610 / 100 tests
<b>Clone:</b>	L3D10
<b>Isotype:</b>	Mouse IgG1, κ
<b>Immunogen:</b>	Extracellular domain of human CTLA-4 and human IgG1 Fc fusion protein
<b>Reactivity:</b>	Human
<b>Preparation:</b>	The antibody was purified by affinity chromatography and conjugated with PE/Dazzle™ 594 under optimal conditions. The solution is free of unconjugated PE/Dazzle™ 594 and unconjugated antibody.
<b>Formulation:</b>	Phosphate-buffered solution, pH 7.2, containing 0.09% sodium azide and 0.2% (w/v) BSA (origin USA).
<b>Concentration:</b>	Lot-specific



PHA-stimulated (three days) human peripheral blood lymphocytes were stained with CD3 FITC and CD152 (clone L3D10) PE/Dazzle™ 594 (top) or mouse IgG1, κ PE/Dazzle™ 594 isotype control (bottom).

**Applications:**

<b>Applications:</b>	Flow Cytometry
<b>Recommended Usage:</b>	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 microL per million cells or 5 microL per 100 microL of whole blood. It is recommended that the reagent be titrated for optimal performance for each application.



\* PE/Dazzle™ 594 has a maximum excitation of 566 nm and a maximum emission of 610 nm.

<b>Application Notes:</b>	<b>ELISA Detection:</b> 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.
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**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 & 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<sup>1,2</sup>.

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

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**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