Brilliant Violet 711[™] anti-human CD152 (CTLA-4)

Catalog # / Size:	2448160 / 100 tests 2448155 / 25 tests	
Clone:	BNI3	
lsotype:	Mouse IgG2a, к	11 July 11
lmmunogen:	Extracellular domain of human CTLA- 4 and constant regions of the human IgG heavy chain (CTLA-4/IgG)	CD131 (close BNU) brilliart Vo
Reactivity:	Human	
Preparation:	The antibody was purified by affinity chromatography and conjugated with Brilliant Violet 711™ under optimal conditions.	CD3 FITC
Formulation:	Phosphate-buffered solution, pH 7.2, containing 0.09% sodium azide and BSA (origin USA)	Human peripher mononuclear ce stimulated with Cocktail (withou 3 hours, surface anti-human CD3 permeabilized, intracellularly s CD152 (CTLA-4)
Workshop Number:	IV N816	
Concentration:	Lot-specific	

Human peripheral blood mononuclear cells were stimulated with Cell Activation Cocktail (without Brefeldin A) for 3 hours, surface stained with anti-human CD3 FITC, fixed, permeabilized, and intracellularly stained with CD152 (CTLA-4) (clone BNI3) Brilliant Violet[™] 711 (left), or mouse IgG2a, κ Brilliant Violet[™] 711 isotype control (right).

Applications:

Applications: Intracellular Flow Cytometry

Recommended Usage: Each lot of this antibody is quality control tested by intracellular immunofluorescent staining with flow cytometric analysis. For flow cytometric staining, the suggested use of this reagent is 5 μ L per million cells in 100 μ L staining volume or 5 μ L per 100 μ L of whole blood. It is recommended that the reagent be titrated for optimal performance for each application.

Brilliant Violet 711[™] excites at 405 nm and emits at 711 nm. The bandpass filter 710/50 nm is recommended for detection, although filter optimization may be required depending on other fluorophores used. Be sure to verify that your cytometer configuration and software setup are appropriate for detecting this channel. Refer to your instrument manual or manufacturer for support. Brilliant Violet 711[™] is a trademark of Sirigen Group Ltd.

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Application
Notes:Based on in-house testing, we do not recommend using clone BNI3 for
immunohistochemistry of paraffin-embedded tissue section.

- Application
 1. Linsley PS, et al. 1992. J. Exp. Med. 176:1595.

 References:
 2. Bonzheim I, et al. 2008. Am. J. Clin. Pathol. 130:613.
- **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. CTLA-4 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, insulindependent 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. sCTLA-4 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. Kuiper HM, et al. 1995. J Immunol. 155:1776.
- **References:**
- 2. Castan J, et al. 1997. Immunology. 90:265.
- 3. Lee CC, et al. 2009. Pediatr Allergy Immunol. 20:624.
- 4. Pistillo MP, et al. 2003. Blood. 101:202.
- 5. Tan PH, et al. 2005. Blood. 106:2936.
- 6. Steiner K, et al. 2001. Clin Exp Immunol. 126:143.