

Alexa Fluor® 647 anti-human CD223 (LAG-3)

Catalog # / Size: 2446020 / 100 tests
2446015 / 25 tests

Clone: 7H2C65

Isotype: Mouse IgG1, κ

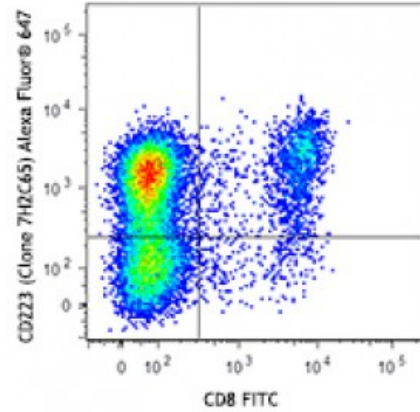
Immunogen: Human LAG-3 transfected cells.

Reactivity: Human

Preparation: The antibody was purified by affinity chromatography and conjugated with Alexa Fluor® 647 under optimal conditions.

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

Concentration: Lot-specific



CD3/CD28/IL-2 stimulated (three days) peripheral blood monocular cells (PBMCs) were stained with CD8 FITC and CD223 (clone 7H2C65) Alexa Fluor® 647 (top) or mouse IgG1, κ Alexa Fluor® 647 isotype control (bottom).

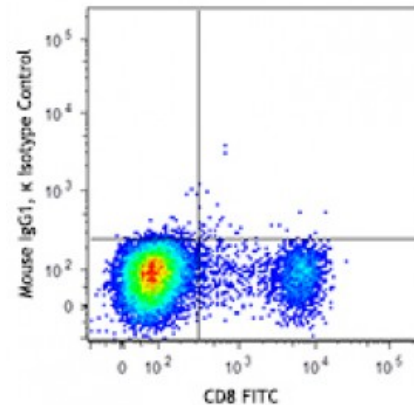
Applications:

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 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.

* Alexa Fluor® 647 has a maximum emission of 668 nm when it is excited at 633 nm / 635 nm.

Application Notes: The staining of clone 7H2C65 cannot be blocked by clone 11C3C65, which is another anti-human CD223 (LAG-3) antibody.



Description: CD223, also known as LAG-3, is a 70 kD type I transmembrane glycoprotein that is involved in T-cell signaling. Similar to CD4, CD223 binds MHC class II, but with a higher affinity. CD223 negatively regulates T-cell activation. It is expressed by activated T-cells and natural killer cells (NKs), as well as regulatory T-cells. It is transiently expressed on the surface of activated T-cells in acute conditions but high expression is maintained under tolerizing conditions. CD223 deficiency results in reduced tumor growth. CD223 and PD-1 can act in synergy and reverse exhausted phenotypes, improve tumor rejection, and control viral load.

- Antigen**
- References:**
1. Castelli C, *et al.* 2014. *Oncoimmunology* 3(11):e967146.
 2. Poirier N, *et al.* 2011. *Clin. Exp. Immunol.* 164:265.
 3. Juno JA, *et al.* 2015. *Retrovirology* 12:17.
 4. Casati C, *et al*