Product Data Sheet

PE anti-human CD223 (LAG-3)

Catalog # / Size: 2446525 / 25 tests

2446530 / 100 tests

Clone: 11C3C65

Isotype: Mouse IgG1, κ

Immunogen: Human LAG-3 transfected cells.

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

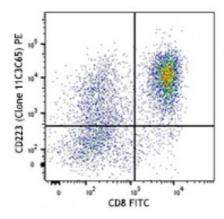
unconjugated antibody.

Formulation: Phosphate-buffered solution, pH 7.2,

containing 0.09% sodium azide and

0.2% (w/v) BSA (origin USA).

Concentration: 0.5



CD3/CD28/IL-2 stimulated (three days) peripheral blood mononuclear cells were stained with CD8 FITC and CD223 (clone 11C3C65) PE (top) or mouse lgG1, κ PE 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

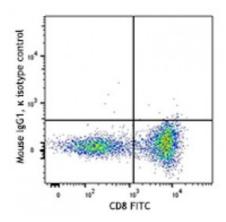
each application.

Application Notes:

The staining of clone 11C3C65 cannot be blocked by clone 7H2C65, 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