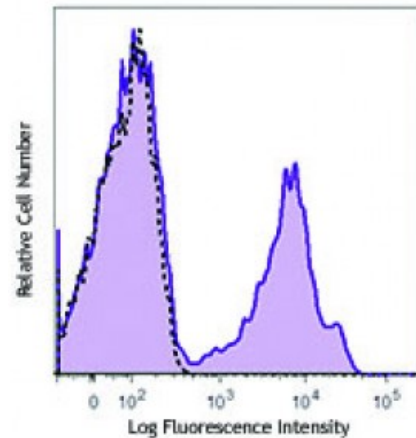


Biotin anti-human CD161

Catalog # / Size: 2299660 / 100 µg
Clone: HP-3G10
Isotype: Mouse IgG1, κ
Immunogen: Human NK cells
Reactivity: Human
Preparation: The antibody was purified by affinity chromatography and conjugated with biotin under optimal conditions. The solution is free of unconjugated biotin.
Formulation: Phosphate-buffered solution, pH 7.2, containing 0.09% sodium azide.
Concentration: 0.5



Human peripheral blood lymphocytes were stained with biotinylated CD161 (clone HP-3G10) (filled histogram) or biotinylated mouse IgG1, κ isotype control (open histogram), followed by Sav-PE.

Applications:

Applications: Other

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 ≤0.5 microg per million cells in 100 microL volume. It is recommended that the reagent be titrated for optimal performance for each application.

Application Notes: Additional reported applications (for the relevant formats) include: inhibition of cytokine production and Western blotting under nonreducing conditions.

Application References:

1. Gumá M, et al. 2004. *Blood* 104:3664.
2. Exley M, et al. 1998. *J. Exp. Med.* 188:867.
3. Marquez C, et al. 1998. *Blood* 91:2760.

Description: CD161 is a type II transmembrane glycoprotein, also known as NKR-P1A, that is expressed as a 40-44 kD homodimer. It is a member of the C-type lectin superfamily. CD161 is expressed on a majority of NK cells, NKT cells, and subsets of peripheral T cells and CD3⁺ thymocytes. It has been reported that Th17 cells are a subpopulation of CD4⁺CD161⁺CCR6⁺ cells. While the biological function of CD161 is not clear, it has been suggested to serve either as a stimulatory receptor or to inhibit NK cell-mediated cytotoxicity and cytokine production. LLT-1 (lectin-like transcript-1, also named as osteoclast inhibitory lectin or CLEC2D) is the ligand of CD161.

Antigen References:

1. Takahashi T, et al. 2006. *J. Immunol.* 176:211.
2. Cosmi L, et al. 2008. *J. Exp. Med.* 205:1903.
3. Aldemir H, et al. 2005. *J. Immunol.* 175:7791.
4. Rosen DB, et al. 2008. <