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**Purified anti-human CD161**

<b>Catalog # / Size:</b>	2299510 / 100 µg
<b>Clone:</b>	HP-3G10
<b>Isotype:</b>	Mouse IgG1, κ
<b>Immunogen:</b>	Human NK cells
<b>Reactivity:</b>	Human
<b>Preparation:</b>	The antibody was purified by affinity chromatography.
<b>Formulation:</b>	Phosphate-buffered solution, pH 7.2, containing 0.09% sodium azide.
<b>Concentration:</b>	0.5

**Applications:**

<b>Applications:</b>	Other
<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 ≤2.0 microg per million cells in 100 microL volume. It is recommended that the reagent be titrated for optimal performance for each application.
<b>Application Notes:</b>	Additional reported applications (for the relevant formats) include: inhibition of cytokine production and Western blotting under nonreducing conditions.
<b>Application References:</b>	1. Gumá M, et al. 2004. <i>Blood</i> 104:3664. 2. Exley M, et al. 1998. <i>J. Exp. Med.</i> 188:867. 3. Marquez C, et al. 1998. <i>Blood</i> 91:2760.

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**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<sup>+</sup> thymocytes. It has been reported that Th17 cells are a subpopulation of CD4<sup>+</sup>CD161<sup>+</sup>CCR6<sup>+</sup> 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. <