abcam

ab328589

APC Anti-CD94 antibody [HP-3D9]

Key facts

Isotype	IgG1
Host species	Mouse
Conjugation	APC
Excitation/Emission	Ex: 650nm, Em: 660nm
Storage buffer	pH: 7.4 Preservative: 0.1% Sodium azide Constituents: PBS, 0.2% BSA
Form	Liquid
Clonality	Monoclonal
Immunogen	Cultured human NK cells. Database link Q13241
Clone number	HP-3D9
Purification technique	Size-exclusion chromatography
Specificity	The mouse monoclonal antibody HP-3D9 recognizes an extracellular epitope of CD94, a 70 kDa type II transmembrane glycoprotein expressed on NK cells, NK-T cells, and subsets of CD8+ T cells and gamma/delta T cells.
Light chain type	kappa

Reactivity data

Flow Cyt

Tested

Species	Human

Dilution info	-
Notes	Flow cytometry: The reagent is designed for analysis of human blood cells using 10 µl reagent / 100 µl of whole blood or 10^6 cells in a suspension. The content of a vial (1 ml) is sufficient for 100 tests.

Target data

See full target information KLRD1

Function

Immune receptor involved in self-nonself discrimination. In complex with KLRC1 or KLRC2 on cytotoxic and regulatory lymphocyte subsets, recognizes non-classical major histocompatibility (MHC) class Ib molecule HLA-E loaded with self-peptides derived from the signal sequence of classical MHC class Ia and non-classical MHC class Ib molecules (PubMed:10023772, PubMed:18064301, PubMed:18083576, PubMed:37264229, PubMed:9486650, PubMed:9754572). Enables cytotoxic cells to monitor the expression of MHC class I molecules in healthy cells and to tolerate self (PubMed:12387742, PubMed:18064301, PubMed:9430220). Primarily functions as a ligand binding subunit as it lacks the capacity to signal. KLRD1-KLRC1 acts as an immune inhibitory receptor. Key inhibitory receptor on natural killer (NK) cells that regulates their activation and effector functions (PubMed:30860984, PubMed:9430220, PubMed:9485206, PubMed:9486650). Dominantly counteracts T cell receptor signaling on a subset of memory/effector CD8-positive T cells as part of an antigen-driven response to avoid autoimmunity (PubMed:12387742). On intraepithelial CD8-positive gamma-delta regulatory T cells triggers TGFB1 secretion, which in turn limits the cytotoxic programming of intraepithelial CD8-positive alpha-beta T cells, distinguishing harmless from pathogenic antigens (PubMed:18064301). In HLA-E-rich tumor microenvironment, acts as an immune inhibitory checkpoint and may contribute to progressive loss of effector functions of NK cells and tumor-specific T cells, a state known as cell exhaustion (PubMed:30503213, PubMed:30860984). Upon HLA-E-peptide binding, transmits intracellular signals through KLRC1 immunoreceptor tyrosine-based inhibition motifs (ITIMs) by recruiting INPP5D/SHIP-1 and INPPL1/SHIP-2 tyrosine phosphatases to ITIMs, and ultimately opposing signals transmitted by activating receptors through dephosphorylation of proximal signaling molecules (PubMed:12165520, PubMed:9485206). KLRD1-KLRC2 acts as an immune activating receptor (PubMed:15940674, PubMed:9655483). On cytotoxic lymphocyte subsets recognizes HLA-E loaded with signal sequence-derived peptides from non-classical MHC class Ib HLA-G molecules, likely playing a role in the generation and effector functions of adaptive NK cells and in maternal-fetal tolerance during pregnancy (PubMed:30134159, PubMed:9754572). Regulates the effector functions of terminally differentiated cytotoxic lymphocyte subsets, and in particular may play a role in adaptive NK cell response to viral infection (PubMed:20952657, PubMed:21825173). Upon HLA-E-peptide binding, transmits intracellular signals via the adapter protein TYROBP/DAP12, triggering the phosphorylation of proximal signaling molecules and cell activation (PubMed:15940674, PubMed:9655483). (Microbial infection) Viruses like human cytomegalovirus have evolved an escape mechanism whereby virus-induced down-regulation of host MHC class I molecules is coupled to the binding of viral peptides to HLA-E, restoring HLA-E expression and inducing HLA-E-dependent NK cell immune tolerance to infected cells. Recognizes HLA-E in complex with human cytomegalovirus UL40-derived peptide (VMAPRTLIL) and inhibits NK cell cytotoxicity. (Microbial infection) May recognize

HLA-E in complex with HIV-1 gag/Capsid protein p24-derived peptide (AISPRTLNA) on infected cells and may inhibit NK cell cytotoxicity, a mechanism that allows HIV-1 to escape immune recognition. (Microbial infection) Upon SARS-CoV-2 infection, may contribute to functional exhaustion of cytotoxic NK cells and CD8-positive T cells (PubMed:32859121). On NK cells, may recognize HLA-E in complex with SARS-CoV-2 S/Spike protein S1-derived peptide (LQPRTFLL) expressed on the surface of lung epithelial cells, inducing NK cell exhaustion and dampening antiviral immune surveillance (PubMed:32859121).

Storage

Shipped at conditions	Ambient - Can Ship with Ice
Appropriate short-term storage conditions	+4°C
Appropriate long-term storage conditions	+4°C
Storage information	Do Not Freeze

Product promise

Tested

We have tested this species and application combination and it works. It is covered by our product promise.

Expected

We have not tested this specific species and application combination in-house, but expect it will work. It is covered by our product promise.

Predicted

This species and application combination has not been tested, but we predict it will work based on strong homology. However, this combination is not covered by our product promise.

Not recommended

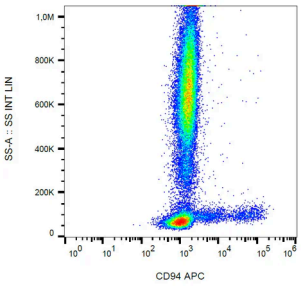
We do not recommend this combination. It is not covered by our product promise.

We are dedicated to supporting your work with high quality reagents and we are here for you every step of the way should you need us.

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1 product image



Flow Cytometry - APC Anti-CD94 antibody [HP-3D9] (ab328589)

CD94 Flow Cytometry staining of human peripheral blood using mouse Anti-CD94 antibody

Flow cytometry analysis (surface staining) of CD94 in human peripheral blood with anti-CD94 (HP-3D9) APC.

Please note: All products are 'FOR RESEARCH USE ONLY. NOT FOR USE IN DIAGNOSTIC OR THERAPEUTIC PROCEDURES'.