

Technical Data Sheet

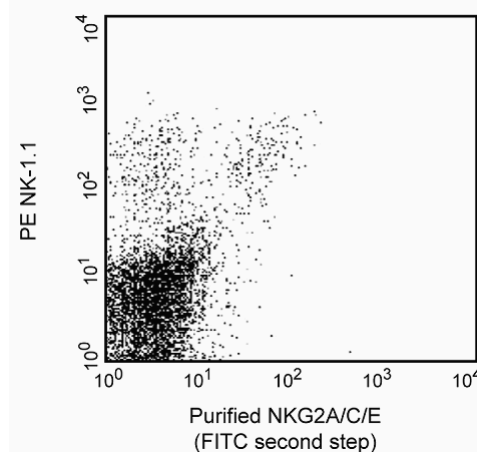
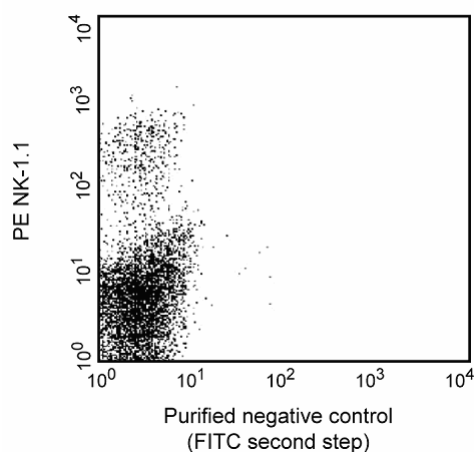
Purified Rat Anti-Mouse NKG2A/C/E

Product Information

Material Number:	550518
Size:	0.1 mg
Concentration:	0.5 mg/ml
Clone:	20d5
Immunogen:	Transfected cell line
Isotype:	Rat (LEW) IgG2a, κ
Reactivity:	QC Testing: Mouse
Storage Buffer:	Aqueous buffered solution containing $\leq 0.09\%$ sodium azide.

Description

The 20d5 antibody reacts with NKG2A, C, and E on a subset of NK and NK-T cells in most strains tested (eg, AKR/J, BALB/c, C3H/He, C57BL/6, CBA/J, DBA/1, FVB/N, 129/Sv, NOD, SWR, and most DBA/2 substrains, but not DBA/2J). The NKG2 molecules are a family of lectin-like receptors that form heterodimers with CD94 on the surface of NK cells. DBA/2J mice do not express CD94, and the lack of CD94 is responsible for the absence of NKG2 expression in this substrain. NKG2 receptors are also expressed on CD8+ T lymphocytes activated *in vivo* and *in vitro*. The heterodimers of CD94 with NKG2A, C, or E recognize Qa-1, a nonclassical MHC class I antigen, presenting the Qdm peptide. Studies of CD94/NKG2 heterodimers on human NK cells have demonstrated that the NKG2 components mediate signal transduction for the receptor, with NKG2A being inhibitory and NKG2C being stimulatory. The CD94/NKG2E heterodimer is also thought to be stimulatory. The mouse NKG2A molecule contains two intracytoplasmic sequences that resemble the ITIM (Immunoreceptor Tyrosine- based Inhibitory Motif) consensus sequence. *NKG2A* transcripts have been shown to be up to 20-fold more abundant than *NKG2C* and *NKG2E* mRNA in NK cells of adult mice. The CD94/NKG2 receptors show increased expression on neonatal NK cells compared to the Ly-49 MHC class I receptors, suggesting that CD94/NKG2 receptors and their ligand, Qa-1, may play a role in maintenance of self-tolerance in developing NK cells. The 20d5 antibody is useful for identification of NK cells expressing functional CD94/NKG2 receptors, in contrast to the non-functional CD94 expressed alone, and it blocks the binding of Qdm-complexed Qa-1b tetramers to CD94/NKG2-transfected CHO cells.



Expression of NKG2A/C/E on mouse splenic NK cells. C57BL/6 splenocytes were simultaneously stained with PE-conjugated anti-mouse NK-1.1 mAb PK136 (Cat. no. 557391) and purified mAb 20d5 (Right panel), followed by FITC-conjugated anti-rat IgG2a mAb RG7/1.30 (Cat. no. 553896). The NK1.1+NKG2+ and NK1.1+NKG2- subsets are detected. Flow cytometry was performed on a BD FACSCalibur™ flow cytometry system.

Preparation and Storage

The monoclonal antibody was purified from tissue culture supernatant or ascites by affinity chromatography.

Store undiluted at 4°C.

Application Notes

Application

Flow cytometry	Routinely Tested
Blocking	Reported

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Suggested Companion Products

Catalog Number	Name	Size	Clone
557391	PE Mouse Anti-Mouse NK-1.1	0.1 mg	PK136
553896	FITC Mouse Anti-Rat IgG2a	0.5 mg	RG7/1.30
553927	Purified Rat IgG2a, κ Isotype Control	0.5 mg	R35-95

Product Notices

1. Since applications vary, each investigator should titrate the reagent to obtain optimal results.
2. Please refer to www.bdbiosciences.com/pharming/en/protocols for technical protocols.
3. Caution: Sodium azide yields highly toxic hydrazoic acid under acidic conditions. Dilute azide compounds in running water before discarding to avoid accumulation of potentially explosive deposits in plumbing.
4. Sodium azide is a reversible inhibitor of oxidative metabolism; therefore, antibody preparations containing this preservative agent must not be used in cell cultures nor injected into animals. Sodium azide may be removed by washing stained cells or plate-bound antibody or dialyzing soluble antibody in sodium azide-free buffer. Since endotoxin may also affect the results of functional studies, we recommend the NA/LE (No Azide/Low Endotoxin) antibody format, if available, for in vitro and in vivo use.

References

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- Vance RE, Kraft JR, Altman JD, Jensen PE, Raulet DH. Mouse CD94/NKG2A is a natural killer cell receptor for the nonclassical major histocompatibility complex (MHC) class I molecule Qa-1(b). *J Exp Med.* 1998; 188(10):1841-1848. (Biology)
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