## Technical Data Sheet

# Biotin Rat Anti-Mouse NKG2A/C/E

#### **Product Information**

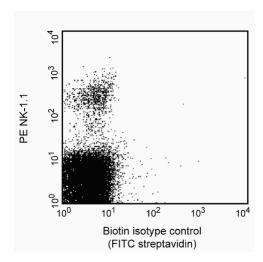
**Material Number:** 550519 Size: 0.1 mg 0.5 mg/mlConcentration: 20d5 Clone:

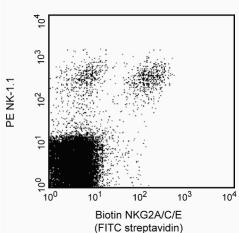
Transfected cell line Immunogen: Rat (LEW) IgG2a, ĸ Isotype: Reactivity: QC Testing: Mouse

Storage Buffer: Aqueous buffered solution containing ≤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) concensus 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 biotin-conjugated rat IgG2a isotype control mAb R35-95 (Cat. no. 553928, Left panel), biotin-conjugated mAb 20d5 (Right panel), and PEconjugated anti-mouse NK-1.1 mAb PK136 (Cat. no. 557391), followed by Streptavidin-FITC (Cat. no. 554060). The NK1.1+NKG2+ and NK1.1 +NKG2- subsets are detected. Flow cytometry was performed on a BD FACSCalibur™ flow cvtometry svstem.

## **Preparation and Storage**

The monoclonal antibody was purified from tissue culture supernatant or ascites by affinity chromatography. The antibody was conjugated with FITC under optimum conditions, and unreacted FITC was removed. Store undiluted at 4°C and protected from prolonged exposure to light. Do not freeze.

### **Application Notes**

Application

Flow cytometry Routinely Tested

# **BD Biosciences**

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# **Suggested Companion Products**

Catalog Number	Name Name	Size	Clone	
553928	Biotin Rat IgG2a κ Isotype Control	0.25 mg	R35-95	
557391	PE Mouse Anti-Mouse NK-1.1	0.1 mg	PK136	
554060	FITC Streptavidin	0.5 mg	(none)	

### **Product Notices**

- Since applications vary, each investigator should titrate the reagent to obtain optimal results.
- Please refer to www.bdbiosciences.com/pharmingen/protocols for technical protocols.
- 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.

#### References

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Vance RE, Jamieson AM, Cado D, Raulet DH. Implications of CD94 deficiency and monoallelic NKG2A expression for natural killer cell development and repertoire formation. Proc Natl Acad Sci U S A. 2002; 99(2):868-873.(Biology)

Vance RE, Jamieson AM, Raulet DH. Recognition of the class lb molecule Qa-1(b) by putative activating receptors CD94/NKG2C and CD94/NKG2E on mouse natural killer cells. *J Exp Med.* 1999; 190(12):1801-1812.(Immunogen)

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