## **Product Data Sheet**

## Alexa Fluor<sup>®</sup> 488 anti-mouse CD16.2 (FcγRIV)

Catalog # / Size:	1347615 / 25 μg 1347620 / 100 μg
Clone:	9E9
Isotype:	Hamster IgG
Immunogen:	FCγR4 ââ,¬â€œEC domain fusion with IgG1 Fc
<b>Reactivity:</b>	Mouse
Preparation:	The antibody was purified by affinity chromatography and conjugated with Alexa Fluor® 488 under optimal conditions.
Formulation:	Phosphate-buffered solution, pH 7.2, containing 0.09% sodium azide.
<b>Concentration:</b>	0.2

## **Applications:**

Applications:	Flow Cytometry
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 $\leq 0.5$ microg per million cells in 100 microL volume. It is recommended that the reagent be titrated for optimal performance for each application.
	st Alexa Fluor $ m I$ 488 has a maximum emission of 519 nm when it is excited at 488 nm.
Application Notes:	Additional reported applications (for the relevant formats of this clone) include: blocking of FcyRIV function1 and inhibition of immune complex binding <sup>1,2</sup> . The LEAF <sup>TM</sup> or Ultra-LEAF <sup>TM</sup> purified antibody (Endotoxin < EU/microg, Azide-Free, 0.2 µm filtered) is recommended for functional assays ( <u>contact our custom solutions</u> <u>team</u> ).
Application References:	1. Mancardi DA, <i>et al.</i> 2008. <i>J. Clin. Invest</i> 118:3738. (FC, Block) 2. Nimmerjahn F, <i>et al.</i> 2005. <i>Immunity</i> 23:41.
Description:	FcyRIV, also known as CD16.2, is an intermediate-affinity activating receptor for IgG2a and IgG2b. CD16.2 is the mouse homolog of human FcyRIIIA. CD16.2 is a low-affinity IgE receptor for all allotypes and the ligation of FcyRIV by antigen-IgE immune complexes promotes macrophage-mediated phagocytosis and is involved in lung inflammation.
Antigen References:	1. Mechetina LV, <i>et al.</i> 2002. <i>Immunogenetics</i> 54:463-8. 2. Nimmerjahn F, <i>et al.</i> 2005. <i>Immunity</i> 23:41-51. 3. Seeling M, <i>et al.</i> 2013. <i>Proc. Natl. Acad. Sci.</i> 110:10729.

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