

DESCRIPTION

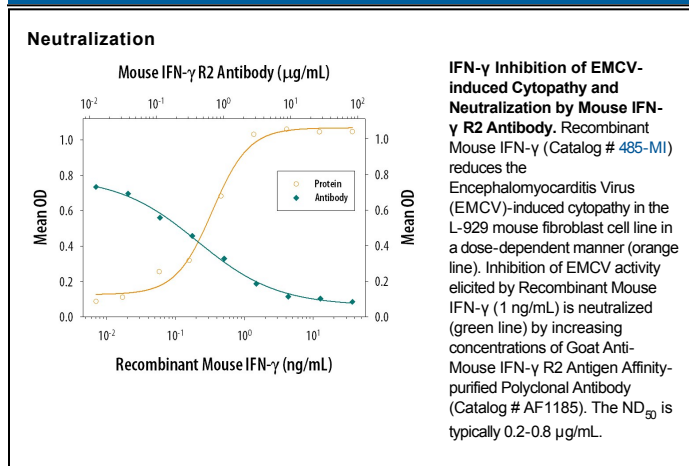
Species Reactivity	Mouse
Specificity	Detects mouse IFN- γ R2 in direct ELISAs and Western blots. In Western blots, approximately 5% cross-reactivity with recombinant human IFN- γ R2 is observed and less than 1% cross-reactivity with recombinant mouse (rm) IFN- γ R1 and rmIFN- α/β R2 is observed.
Source	Polyclonal Goat IgG
Purification	Antigen Affinity-purified
Immunogen	Mouse myeloma cell line NS0-derived recombinant mouse IFN- γ R2 Ser21-Val243 Accession # NP_032364
Endotoxin Level	<0.10 EU per 1 μ g of the antibody by the LAL method.
Formulation	Lyophilized from a 0.2 μ m filtered solution in PBS with Trehalose. See Certificate of Analysis for details. *Small pack size (-SP) is supplied as a 0.2 μ m filtered solution in PBS.

APPLICATIONS

Please Note: Optimal dilutions should be determined by each laboratory for each application. *General Protocols* are available in the *Technical Information* section on our website.

	Recommended Concentration	Sample
Western Blot	0.1 μ g/mL	Recombinant Mouse IFN- γ R2
Neutralization	Measured by its ability to neutralize IFN- γ R2-mediated inhibition of EMCV-induced cytopathy in the L-929 mouse fibroblast cell line. The Neutralization Dose (ND ₅₀) is typically 0.2-0.8 μ g/mL in the presence of 1 ng/mL Recombinant Mouse IFN- γ .	

DATA



PREPARATION AND STORAGE

Reconstitution	Reconstitute at 0.2 mg/mL in sterile PBS.
Shipping	The product is shipped at ambient temperature. Upon receipt, store it immediately at the temperature recommended below. *Small pack size (-SP) is shipped with polar packs. Upon receipt, store it immediately at -20 to -70 °C
Stability & Storage	Use a manual defrost freezer and avoid repeated freeze-thaw cycles. <ul style="list-style-type: none"> • 12 months from date of receipt, -20 to -70 °C as supplied. • 1 month, 2 to 8 °C under sterile conditions after reconstitution. • 6 months, -20 to -70 °C under sterile conditions after reconstitution.

BACKGROUND

IFN- γ R2 (Interferon gamma receptor 2; also called IFN- γ R β IFN- γ RII, or AF1) is a 60-64 kDa type I transmembrane glycoprotein that is a member of the class II cytokine receptor family of molecules (1). It is widely expressed as part of a preassembled cell surface multimeric complex. In the absence of IFN- γ , the complex contains two each of IFN- γ R1, R2 and Jak1 molecules (2). Binding of IFN- γ to IFN- γ R1 recruits Jak2 to IFN- γ R2 and initiates phosphorylation, STAT1 binding, conformational changes, and transcriptional regulation, which mainly inhibits proliferation and/or promotes apoptosis (2, 3). Mouse IFN- γ R2 cDNA encodes 332 amino acids (aa), including a signal sequence (aa 1-27), an extracellular region (ECD, aa 28-243) with two fibronectin type III domains, a transmembrane sequence (aa 244-264) and a cytoplasmic tail (aa 265-332) (1, 2). Within the ECD, mouse IFN- γ R2 shares 80% aa sequence identity with rat IFN- γ R2, and 49-55% with human, canine, porcine and bovine IFN- γ R2. IFN- γ R1 and R2 must be from the same species for receptor complexes to be active, and human IFN- γ is not active on the mouse IFN- γ receptor complex (1, 2). IFN- γ R1 is essential for ligand binding and is more constitutively expressed, while IFN- γ R2 is essential for signaling, and its more limited expression controls cell response to IFN- γ (2, 3). For example, mouse T cell IFN- γ R2 is down-regulated during differentiation to subtypes such as Th1 which produce IFN- γ . (3, 4) This allows expansion of activated cells without growth arrest due to paracrine response to IFN- γ . Following expansion, IFN- γ R2 is re-expressed to limit the immune reaction (5). IFN- γ signaling mediates control of intracellular pathogens such as mycobacteria (3, 4, 6). In humans, deficiency of IFN- γ R2 or other IFN- γ pathway molecules causes the MSMD (mendelian susceptibility to mycobacterial diseases) syndrome (6-8).

References:

1. Hemmi, S. *et al.* (1994) *Cell* **76**:803.
2. Krause, C.D. *et al.* (2006) *Cell Res.* **16**:55.
3. Haring, J. S. *et al.* (2005) *J. Immunol.* **174**:6791.
4. Tau, G.Z. *et al.* (2000) *J. Exp. Med.* **192**:977.
5. Foulds, K.E. *et al.* (2008) *J. Immunol.* **180**:842.
6. Rosenzweig, S.D. *et al.* (2004) *J. Immunol.* **173**:4000.
7. Filipe-Santos, O. *et al.* (2006) *Semin. Immunol.* **18**:347.
8. Zhang, S-Y. *et al.* (2008) *Immunol. Rev.* **226**:29.