

## DESCRIPTION

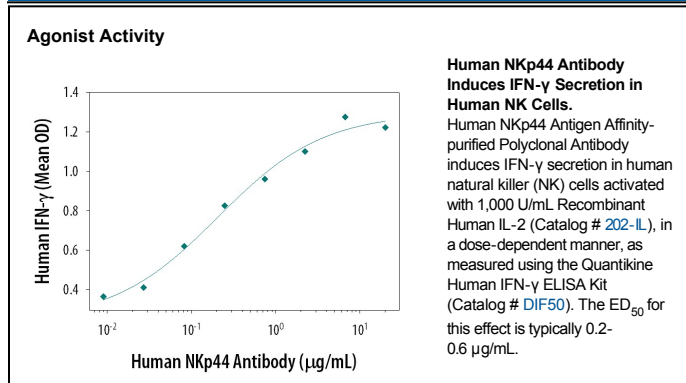
<b>Species Reactivity</b>	Human
<b>Specificity</b>	Detects human NKp44/NCR2 in direct ELISAs and Western blots. In direct ELISAs and Western blots, less than 1% cross-reactivity with recombinant human (rh) NKp30, rhNKp46, and rhNKp80 is observed.
<b>Source</b>	Polyclonal Goat IgG
<b>Purification</b>	Antigen Affinity-purified
<b>Immunogen</b>	Mouse myeloma cell line NS0-derived recombinant human NKp44/NCR2 Gln22-Pro190, predicted Accession # O95944
<b>Endotoxin Level</b>	<1.5 EU per 1 µg of the antibody by the LAL method.
<b>Formulation</b>	Lyophilized from a 0.2 µm filtered solution in PBS with Trehalose. See Certificate of Analysis for details. *Small pack size (-SP) is supplied as a 0.2 µm 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
<b>Western Blot</b>	0.1 µg/mL	Recombinant Human NKp44/NCR2 Fc Chimera (Catalog # 2249-NK)
<b>Agonist Activity</b>	0.2-0.6 µg/mL	See Below

## DATA



## PREPARATION AND STORAGE

<b>Reconstitution</b>	Reconstitute at 0.2 mg/mL in sterile PBS.
<b>Shipping</b>	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
<b>Stability &amp; Storage</b>	<b>Use a manual defrost freezer and avoid repeated freeze-thaw cycles.</b> <ul style="list-style-type: none"> <li>● 12 months from date of receipt, -20 to -70 °C as supplied.</li> <li>● 1 month, 2 to 8 °C under sterile conditions after reconstitution.</li> <li>● 6 months, -20 to -70 °C under sterile conditions after reconstitution.</li> </ul>

## BACKGROUND

NKp44, along with NKp30 and NKp46, constitute a group of receptors termed "Natural Cytotoxicity Receptors" (NCR) (1). These receptors are expressed almost exclusively by NK cells and play a major role in triggering NK-mediated killing of most tumor cell lines. No rodent ortholog to NKp44 has been identified. Human NKp44, also known as NCR2, is a 44 kDa type I transmembrane glycoprotein that is characterized by the presence of one extracellular V-like immunoglobulin domain (2). It is synthesized as a 276 amino acid (aa) precursor that contains a 21 aa signal sequence, a 171 aa extracellular region, a 21 aa transmembrane segment and a 63 aa cytoplasmic tail. Alternate splicing in both the cytoplasmic tail and extracellular region generates multiple isoforms of unknown significance. The Ig-like region is unaffected. A physical association with the ITAM-bearing accessory protein, DAP12, occurs via a charged residue in the NKp44 transmembrane domain. Ligation of NKp44 with a specific antibody results in phosphorylation of DAP12 (3) and activation of target cell lysis in a redirected killing assay (4). NKp44 is absent from resting NK cells but is up-regulated upon activation with IL-2. Activation-induced expression occurs in the CD56<sup>dim</sup> CD16<sup>+</sup> NK subset that accounts for more than 85% of NK cells found in peripheral blood and spleen, as well as the CD56<sup>bright</sup> CD16<sup>-</sup> NK subset that constitutes the majority of NK cells in lymph node and tonsil (5). Studies with neutralizing antibodies reveal that NKp44 is partially responsible for triggering lytic activity against several tumor cell types (2, 6). Blocking any of the individual NCRs results in partial inhibition of tumor cell lysis, but nearly complete inhibition of lysis is observed if all three receptors are blocked simultaneously (6). NKp44 has also been implicated in recognition of virus-infected cells through its capacity to bind to viral hemagglutinins (7).

## References:

1. Moretta, L. and A. Moretta (2004) *EMBO J.* **23**:255.
2. Cantoni, C. *et al.* (1999) *J. Exp. Med.* **189**:787.
3. Augugliaro, R. *et al.* (2003) *Eur. J. Immunol.* **33**:1235.
4. Vitale, M. *et al.* (1998) *J. Exp. Med.* **187**:2065.
5. Ferlazzo, G. *et al.* (2004) *J. Immunol.* **172**:1455.
6. Pende, D. *et al.* (1999) *J. Exp. Med.* **190**:1505.
7. Arnon, T. *et al.* (2001) *Eur. J. Immunol.* **31**:2680.