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Product Data Sheet

Recombinant Mouse IL-12 p40 Homodimer (carrier-free)

Catalog # / Size: 573102 / 10 µg
573104 / 25 µg
573106 / 100 µg
573108 / 500 µg

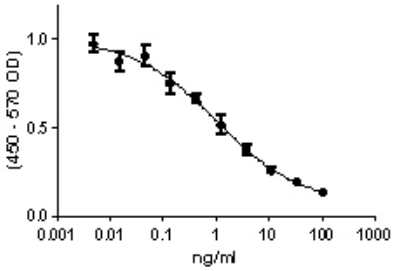
Source: Mouse IL-12 p40 homodimer, amino acids Met23-Ser335 (Accession # NM_008352), was expressed in insect cells.

Molecular Mass: The 313 amino acid recombinant protein has a predicted molecular mass of 35.8 kD. The DTT-reduced protein migrates at approximately 40 kD and the non-reduced protein migrates at approximately 75 kD by SDS-PAGE. The N-terminal amino acid is Met.

Purity: >98%, as determined by Coomassie stained SDS-PAGE.

Endotoxin Level: Less than 0.01 ng per µg cytokine as determined by the LAL method.

Activity: ED_{50} = 1- 4 ng/ml corresponding to a specific activity of 1.0 - 0.25 x 10^6 units/mg, as determined by the dose dependent inhibition of IL-12-dependent IFNγ production.



Mouse IL-12 p40 homodimer is able to inhibit IL-12-dependent IFNγ production.

Preparation: 10-100 µg sizes are bottled at 100 µg/mL. 500 µg and larger sizes are bottled at the concentration indicated on the vial.

Formulation: 0.22 µm filtered protein solution is in 20 mM Tris-HCl, pH 8.0, 0.1 M NaCl

Storage: Unopened vial can be stored at -20°C for six months or at -70°C for one year. For maximum results, quick spin vial prior to opening. Stock solutions should be prepared at no less than 10 µg/mL in **sterile** buffer containing carrier protein such as 1% BSA or HSA or 10% FBS. Stock solution can be stored for one month at 4°C or up to three months at -20°C to -70°C. **Avoid repeated freeze/thaw cycles.**

Applications:

Applications: Bioassay

Application References:
1. Wang X, *et al.* 1999. *Eur. J. Immunol.* 29:2007.
2. Walter JM, *et al.* 2001. *J. Exp. Med.* 193:339.
3. Russell TD, *et al.* 2003. *J. Immunol.* 171:6866.
4. Mikols CL, *et al.* 2006. *Am. J. Respir. Crit. Care* 174:461.
5. Gunsten S, *et al.* 2008. *Immunology* 126:500.
6. Jana M, *et al.* 2009. *Glia* 57:1553.
6. Yabu M, *et al.* 2010. *Int Immunol.* 23:29. PubMed

Description: IL-12 and IL-23 share the p40 subunit, which heterodimerizes respectively with IL-12 p35 or IL-23 p19 subunits to form IL-12 or IL-23. IL-12 p40 exists as a monomer and as a homodimer (IL-12 p80). IL-12 induction is relevant in asthmatic airway inflammation. IL-12 expression can be induced by mouse parainfluenza type I (Sendai) virus and its source is airway epithelial cells. In that experimental model, IL-12 induction is followed by excessive expression of IL-12 p40 that could be further enhanced in IL-12 p35-deficient mice. Overexpression of IL-12 p80 causes macrophage accumulation and contributes to airway inflammation and consequent morbidity during viral bronchitis. Amplified epithelial IL-12 p40 expression and augmented concentrations of BAL fluid IL-12 p40 (but not IL-12 p70) has been detected in asthmatic subjects. It has been demonstrated that p80, but not IL-12 or p40, induces macrophage chemotaxis that is independent of IL-12 and mediated through the cytoplasmic tail of IL-12b1. Additional studies with transgenic mice suggest that overexpression of IL-12 p80 prior to a viral infection increases the number of resident airway macrophages, and this primes the host for a protective response against a lethal respiratory viral infection. In addition, it has been suggested that p80 functions as a competitive antagonist of IL-12 p70. Mouse Con A-activated splenocytes display identical binding affinities for p80 and IL-12, and in these cells p80 competitively inhibited IL-12 binding and IL-12-dependent proliferation. Furthermore, p80 is able to inhibit IL-12-dependent IFNγ production in freshly isolated splenocytes.

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