

An Affymetrix Company

Mouse IL-22 Recombinant Protein

Catalog Number: 14-8221

Also known as: Interleukin-22, IL-TIF

RUO: For Research Use Only. Not for use in diagnostic procedures.

Product Information

Contents: Mouse IL-22 Recombinant Protein

REF Catalog Number: 14-8221

Handling Conditions: For best recovery, quick-spin vial prior to opening. Use in a

sterile environment

Source: E. coli expressed amino acids Leu34-Val179 of mature mouse IL-22

accession # NM_016971

Molecular Mass: The protein is

methionylated at the N-terminal and has a predicted molecular mass of 16,771. The DTT reduced protein migrates as a 15 kDa polypeptide on SDS-PAGE. The non-reduced protein migrates as a 13 kDa polypeptide. **Purity:** > 98% as determined by SDS-PAGE.

Endotoxin: Less than 0.01 ng/ug cytokine as

determined by the LAL assay.

Bioactivity: Measured by induction of IL-10 production by Colo205 cells. The ED50 for

this is typically below 100 pg/mL,

corresponding to a specific activity of greater

than 1.0 x 10e7 U/mg.

Formulation: Sterile liquid; 20 mM sodium phosphate, pH 7.2, 0.3 M NaCl, 1.0% BSA. 0.22 um filtered.



LOT

Temperature Limitation: Store at less than or

equal to -70°C.

Batch Code: Refer to vial
Use By: Refer to vial



IL-22, also known as IL-10-related T-cell derived inducible factor, is an alpha helical cytokine and is considered a member of the IFN-IL-10 family, which includes IL-19, IL-20, IL-24, IL-26, IL-28, IL-29, and the type I and II interferons. IL-22 is produced mainly by activated T and NK cells. No other immune cells (resting or activated) or non-immune cells have been found to produce IL-22. Amongst the T cells, in mice Th1 and Th17 cells appear to be the primary producers of IL-22. IL-22 acts by engaging the heterodimeric receptor complex consisting of primary receptor IL-22R1 and accessory receptor IL-10R2. IL-22R1 also binds IL-20 and IL-24; IL-10R2 also binds IL-10, IL-27, IL-28, and IL-29. Binding of IL-22 to its receptor complex induces signal transduction, particularly via the JAK-STAT pathway. In addition to the cell surface IL-22R1/IL-10R2 complex, a soluble single chain IL-22 receptor termed IL-22BP has been found to antagonize IL-22 binding and signaling. IL-22 appears not to directly influence immune cells; major targets of the cytokine appear to be nonimmune cells, such as cells of the skin, digestive and respiratory system, as well as hepatocytes, and keratinocytes.

IL-22 has been described as an effector cytokine of the Th17 lineage. Along with IL-17A and IL-17F, IL-22 regulates genes associated with innate immunity of the skin. IL-17A, IL-17F and IL-22 are all coexpressed by Th17 cells, however, differentially regulated. Note that TGFb, which is required for IL-17A production, inhibits IL-22 production. The effects of IL-22 include induction of acute phase reactants and antimicrobial proteins, as well as increasing the mobility of keratinocytes. IL-22 has been reported to mediate IL-23-induced acanthosis and dermal inflammation through activation of STAT3.

Applications Reported

The recombinant mouse IL-22 has been reported useful for bioassay.

Applications Tested

This recombinant IL-22 has been tested in bioassay for induction of IL-10 production by Colo205 cells. The ED50 for this effect is typically below 100 pg/ml, corresponding to a specific activity of greater than 1.0 x 10E7 U/mg.



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References

Wolk K, Witte E, Hoffmann U, Doecke WD, Endesfelder S, Asadullah K, Sterry W, Volk HD, Wittig BM, Sabat R. IL-22 induces lipopolysaccharide-binding protein in hepatocytes: a potential systemic role of IL-22 in Crohn's disease. J Immunol. 2007 May 1;178(9):5973-81.

Zheng Y, Danilenko DM, Valdez P, Kasman I, Eastham-Anderson J, Wu J, Ouyang W. IL-22, a Th17 cytokine, mediates IL-23 induced dermal inflammation and acanthosis. ure. 2007 Feb 8;445(7128):648-51.

Liang SC, Tan XY, Luxenberg DP, Karim R, Dunussi-Joannopoulos K, Collins M, Fouser LA.. IL-22 and IL-17 are coexpressed by Th17 cells and cooperatively enhance expression of antimicrobial peptides. J Exp Med. 2006 Oct 2;203(10):2271-9.

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