

## Human non-lytic IL-21/mouse Fc Fusion Protein

**CATALOG#:** MF-22021  
**QUANTITY:** 10 µg  
**MOLECULAR STRUCTURE:**

**LOT#:**  
**CONCENTRATION:** 100 µg/ml  
A soluble 90 kd dimeric fusion protein consisting of the extracellular (126 aa) domain of human IL-21 fused to mutant mouse IgG2a Fc.

**TRANSFECTANT CELL LINE:**  
**STORAGE CONDITIONS:**

NS1 cells  
Store stock solution at <-20<sup>0</sup>C. Store working solution at 4<sup>0</sup>C. Freeze/Thawing is not recommended.

**PRODUCT STABILITY:**

Product should retain for at least one year after shipping date when stored at <-20<sup>0</sup>C and the working solution should retain for at least one week at 4<sup>0</sup>C.

**FORMULATION:** IL-21/Fc is supplied as a frozen liquid comprised of 0.22 µm sterile-filtered PBS (PH 7.4, 50 mM Sodium Phosphate, 100 mM Potassium Chloride, 150 mM NaCl) and containing no preservatives.

**PRODUCTION:** Human IL-21/Fc fusion protein was purified from serum free tissue culture supernatant of NS1 transfectants. Purity was >98% by SDS-PAGE. The endotoxin level is ≤0.2 EU per µg of IL-21/Fc.

**ACTIVITY RANGE:**

The ED50, determined by the dose-dependent costimulation of IL-21/Fc on splenic leukocyte proliferation triggered by suboptimal dose of soluble anti-CD3 (0.2 µg/ml), is 0.2 -2 µg/ml.

**INFORMATION:** IL-21 and its receptor have recently been cloned. IL-21 appears to be a new member of the IL-2/IL-15 cytokine family, being most homologous to IL-15. Recent evidence suggests that IL-21 plays a supportive role in the proliferation of activated lymphocytes and NK cells.<sup>(1-3)</sup> Human IL-21/Fc fusion protein was made by genetically fused human IL-21 to mouse mutant Fcγ2a. This fusion protein possesses both biological function of IL-21 and prolonged circulating half-life determined by Fc domain. Mutations to the complement (C1q) and FcγR I binding sites of the Fcγ2a fragment render IL-21/Fc incapable to direct antibody directed cytotoxicity (ADCC) and complement directed cytotoxicity (CDC)<sup>(4)</sup>.

Reference:

1. Ishida, Y., Y. Agata, K. Shibahara, and T. Honjo. 1992. Induced expression of PD-1, a novel member of the immunoglobulin gene superfamily, upon programmed cell death. *Embo J* 11:3887.
2. Latchman, Y., C. R. Wood, T. Chernova, D. Chaudhary, M. Borde, I. Chernova, Y. Iwai, A. J. Long, J. A. Brown, R. Nunes, E. A. Greenfield, K. Bourque, V. A. Boussiotis, L. L. Carter, B. M. Carreno, N. Malenkovich, H. Nishimura, T. Okazaki, T. Honjo, A. H. Sharpe, and G. J. Freeman. 2001. PD-L2 is a second ligand for PD-1 and inhibits T cell activation. *Nat Immunol* 2:261.
3. Freeman, G. J., A. J. Long, Y. Iwai, K. Bourque, T. Chernova, H. Nishimura, L. J. Fitz, N. Malenkovich, T. Okazaki, M. C. Byrne, H. F. Horton, L. Fouser, L. Carter, V. Ling, M. R. Bowman, B. M. Carreno, M. Collins, C. R. Wood, and T. Honjo. 2000. Engagement of the PD-1 immunoinhibitory receptor by a novel B7 family member leads to negative regulation of lymphocyte activation. *J Exp Med* 192:1027.
4. Zheng, X. X., A. W. Steele, P. Nickerson, W. Steurer, J. SteFcer, and T. B. Strom. 1995. Administration of Non-Cytolytic IL-10/Fc in LPS-induced septic shock and allogeneic islet transplantation murine animal models. *J. Immunol.* 154:559

**\*This Product is intended for Laboratory Research use only.**

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