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Product datasheet for TP724252

Cynomolgus IL9 Protein, hFc Tag

Product data:

Product Type:	Recombinant Proteins
Description:	Cynomolgus IL9 Protein, hFc Tag
Expression Host:	HEK293
Tag:	C-Human Fc
Predicted MW:	The protein has a predicted molecular mass of 40.0 kDa after removal of the signal peptide.
Purity:	The purity of the protein is greater than 95% as determined by SDS-PAGE and Coomassie blue staining.
Reconstitution Method:	Lyophilized from sterile PBS, pH 7.4. Normally 5 % - 8% trehalose is added as protectants before lyophilization.
Storage:	Store at -20°C to -80°C for 12 months in lyophilized form. After reconstitution, if not intended for use within a month, aliquot and store at -80°C (Avoid repeated freezing and thawing). Lyophilized proteins are shipped at ambient temperature.
Stability:	12 months from date of despatch
Summary:	Interleukin 9, also known as IL-9, is a cytokine (cell signaling molecule) belonging to the group of interleukins. IL-9 is a cytokine that acts as a regulator of a variety of hematopoietic cells. This cytokine stimulates cell proliferation and prevents apoptosis. It functions through the interleukin 9 receptor (IL-9R), which activates different signal transducer and activator (STAT) proteins and thus connects this cytokine to various biological processes. Genetic studies on a mouse model of asthma demonstrated that this cytokine is a determining factor in the pathogenesis of bronchial hyperresponsiveness. IL-9 is a key molecule that affects the differentiation of TH17 cells and Treg function. IL-9 predominantly produced by TH17 cells synergizes with TGF-β1 to differentiate naive CD4 T cells into TH17 cells, while IL-9 secretion by TH17 cells is regulated by IL-23. Interestingly, IL-9 enhances the suppressive functions of FoxP3 CD4 Treg cells in vitro, and the absence of IL-9 signaling weakens the suppressive activity of nTregs in vivo, leading to an increase in effector cells and worsening of experimental autoimmune encephalomyelitis. The mechanism of IL-9 effects on TH17 and Tregs is through activation of STAT3 and STAT5 signaling. Our findings highlight the role of IL-9 as a regulator of pathogenic versus protective mechanisms of immune responses.

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