Proteins





Product Data Sheet

LILRB4/CD85k/ILT3 Protein, Human (Biotinylated, 141a.a, HEK293, His-Avi)

Cat. No.: HY-P701018

Synonyms: HM18; ILT3; ILT-3; LILRB4; LIR5; CD85K

Species: Human HEK293 Source:

Accession: AAH26309.1 (G119-E259)

Gene ID: 11006 **Molecular Weight:** 32-45 kDa

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| Appearance | Solution. |
|---------------------|--------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| Formulation | Supplied as a 0.22μm filtered solution of PBS, pH 7.4. |
| Endotoxin Level | <1 EU/μg, determined by LAL method. |
| Reconsititution | N/A. |
| Storage & Stability | Stored at -80°C for 1 year. It is stable at -20°C for 3 months after opening. It is recommended to freeze aliquots at -80°C for extended storage. Avoid repeated freeze-thaw cycles. |
| Shipping | Shipping with dry ice. |

DESCRIPTION

Background

LILRB4/CD85k/ILT3, an inhibitory receptor intricately involved in immune regulation, plays a crucial role in down-regulating immune responses. It serves as a receptor for FN1 and integrin ITGAV/ITGB3, exerting inhibitory effects on IgE-mediated mast cell activation and KITLG/SCF-mediated mast cell activation. Through its interaction with ITGAV/ITGB3, LILRB4/ILT3 further inhibits antibody production by memory and marginal zone B cells, likely by suppressing their differentiation into plasma cells. This multifaceted receptor extends its inhibitory influence to diverse immune functions, such as suppressing IFNG production by CD8 T cells, CD4 T cells, and natural killer cells, as well as inhibiting antigen presentation by dendritic cells to T cells, preventing T cell activation. Additionally, LILRB4/ILT3 effectively inhibits lipopolysaccharide-mediated neutrophil-dependent vascular injury and contributes to the suppression of the allergic inflammatory response by impeding the infiltration of neutrophils and eosinophils while preventing mast cell degranulation. Its interactions, particularly when tyrosine phosphorylated, with SH2 domain-containing phosphatases PTPN6/SHP-1 and PTPN11/SHP-2 enhance the inhibition of mast cell activation.

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