CD84/SLAMF5 Protein, Human (HEK293, His-Avi)

Cat. No.:	HY-P78418
Synonyms:	Hly9-beta; CD84; SLAMF5; LY9B; CD84 molecule; DKFZp781E2378; Hly9-β
Species:	Human
Source:	HEK293
Accession:	Q9UIB8 (K22-G225)
Gene ID:	8832
Molecular Weight:	40-55 kDa

PROPERTIES

Appearance	Lyophilized powder.
Formulation	Lyophilized from a 0.22 μm filtered solution of PBS, pH 7.4. Normally 5% trehalose is added as protectant before lyophilization.
Endotoxin Level	<1 EU/μg, determined by LAL method.
Reconsititution	It is not recommended to reconstitute to a concentration less than 100 μg/mL in ddH_2O.
Storage & Stability	Stored at -20°C for 2 years. After reconstitution, it is stable at 4°C for 1 week or -20°C for longer (with carrier protein). It is recommended to freeze aliquots at -20°C or -80°C for extended storage.
Shipping	Room temperature in continental US; may vary elsewhere.

DESCRIPTION

Background

The CD84/SLAMF5 protein serves as a self-ligand receptor within the signaling lymphocytic activation molecule (SLAM) family, engaging in homo- or heterotypic cell-cell interactions that modulate the activation and differentiation of diverse immune cells, contributing to the intricate regulation and interconnection of both innate and adaptive immune responses. The protein's activities are finely tuned by the presence or absence of small cytoplasmic adapter proteins, including SH2D1A/SAP and/or SH2D1B/EAT-2. CD84/SLAMF5 can mediate natural killer (NK) cell cytotoxicity, dependent on both SH2D1A and SH2D1B. It enhances proliferative responses of activated T-cells, and SH2D1A/SAP appears not to be required for this process. Homophilic interactions amplify interferon gamma/IFNG secretion in lymphocytes and induce platelet stimulation via a SH2D1A-dependent pathway. The protein may serve as a marker for hematopoietic progenitor cells. Essential for prolonged T-cell:B-cell contact, optimal T follicular helper function, and germinal center formation, CD84/SLAMF5 in germinal centers contributes to maintaining B-cell tolerance and preventing autoimmunity. In mast cells, it negatively regulates high-affinity immunoglobulin epsilon receptor signaling, independently of SH2D1A and SH2D1B but implicating FES and PTPN6/SHP-1. In macrophages, it positively regulates LPS-induced MAPK phosphorylation and NF-kappaB activation, modulating LPS-induced cytokine secretion. Additionally, CD84/SLAMF5 positively regulates macroautophagy in primary dendritic cells through the stabilization of IRF8 and inhibits TRIM21-mediated proteasomal degradation of IRF8. The protein forms homodimers via its extracellular domain and interacts with CD48 molecules from

other cells in a head-to-tail dimeric arrangement. It also interacts with SH2 domain-containing proteins, including SH2D1A/SAP and SH2D1B/EAT-2, and tyrosine-protein phosphatases PTPN6/SHP-1 and PTPN11/SHP-2 via its phosphorylated cytoplasmic domain, with the latter interaction blocked by SH2D1A. CD84/SLAMF5 further interacts with INPP5D/SHIP1 through its phosphorylated ITSM domains.

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