ATAGENIX LABORATORIES

Catalog Number:ATMP00024HU

Recombinant Human ICOS protein ,C- Fc Tag

Product Details

Summary

English name	Recombinant Human ICOS protein ,C- Fc Tag
Purity	>90% as determined by SDS-PAGE
Endotoxin level	<1.0 EU per μg of the protein as determined by the LAL method.
Construction	A DNA sequence encoding the human ICOS(Met1~ Lys140) was fused with the C-terminal Fc Tag
Accession #	Q9Y6W8
Host	Mammalian cells
Species	Homo sapiens (Human)
Predicted Molecular Mass	39.99kDa
Formulation	Supplied as solution form in PBS, pH7.5 or lyophilized from PBS, pH7.5.
Shipping	In general, proteins are provided as lyophilized powder/frozen liquid. They are shipped out with dry ice/blue ice unless customers require otherwise.
Stability &Storage	Use a manual defrost freezer and avoid repeated freeze thaw cycles. Store at 2 to 8 °C for one week . Store at -20 to -80 °C for twelve months from the date of receipt.
Reconstitution	Reconstitute in sterile water for a stock solution.A copy of datasheet will be provided with the products, please refer to it for details.

Background

Background	Inducible T-cell costimulator (ICOS) is also known as Activation-inducible lymphocyte immunomediatory molecule (AILIM), CD278, which belongs to the CD28 family of immune costimulatory receptors consisting of CD28, CTLA-4 and PD-1. ICOS enhances all basic T-cell responses to a foreign antigen, namely proliferation, secretion of lymphokines, up-regulation of molecules that mediate cell-cell interaction, and effective help for antibody secretion by B-cells. CD278 / ICOS prevents the apoptosis of pre-activated T-cells and also plays a critical role in CD40-mediated class switching of immunoglobin isotypes.
Alternative Names	ICOS,CD278,AILIM,Inducible T-cell costimulator

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References

Louis, Macedo, Bailly, Lau, Ramaswami, Marrari, Landsittel, Chang, Chandran, Fadakar, Yamada, Chalasani, Randhawa, Zeevi, Singh, Lefaucheur, Metes (2020) Coordinated Circulating T Follicular Helper and Activated B Cell Responses Underlie the Onset of Antibody-Mediated Rejection in Kidney Transplantation Journal of the American Society of Nephrology : JASN ()

Frontier progress

Although antibody-mediated rejection (ABMR) has been long recognized as a leading cause of allograft failure after kidney transplantation, the cellular and molecular processes underlying the induction of deleterious donor-specific antibody (DSA) responses remain poorly understood. Using high-dimensional flow cytometry, in vitro assays, and RNA sequencing, we concomitantly investigated the role of T follicular helper (TFH) cells and B cells during ABMR in 105 kidney transplant recipients. There were 54 patients without DSAs; of those with DSAs, ABMR emerged in 20 patients, but not in 31 patients. We identified proliferating populations of circulating TFH cells and activated B cells emerging in blood of patients undergoing ABMR. Although these circulating TFH cells comprised heterogeneous phenotypes, they were dominated by activated (ICOS+PD-1+) and early memory precursor (CCR7+CD127+) subsets, and were enriched for the transcription factors IRF4 and c-Maf. These circulating TFH cells produced large amounts of IL-21 upon stimulation with donor antigen and induced B cells to differentiate into antibody-secreting cells that produced DSAs. Combined analysis of the matched circulating TFH cell and activated B cell RNA-sequencing profiles identified highly coordinated transcriptional programs in circulating TFH cells and B cells among patients with ABMR, which markedly differed from those of patients who did not develop DSAs or ABMR. The timing of expansion of the distinctive circulating TFH cells and activated B cells paralleled emergence of DSAs in blood, and their magnitude was predictive of IgG3 DSA generation, more severe allograft injury, and higher rate of allograft loss. Patients undergoing ABMR may benefit from monitoring and therapeutic targeting of TFH cell-B cell interactions.