Recombinant Mouse B7-H4

Catalog Number: 2154-B7

DESCRIPTION

Source	Mouse myeloma cell line, NS0-derived Phe29-Pro258, with a C-terminal 10-His tag Accession # Q7TSP5
N-terminal Sequence Analysis	Phe29
Predicted Molecular Mass	26.6 kDa

SPECIFICATIONS

SDS-PAGE	45-55 kDa, reducing conditions
Activity	Measured by its ability to inhibit anti-CD3 antibody induced IL-2 secretion in human T lymphocytes. The presence of Recombinant Mouse B7-H4 at 10 µg/mL inhibited the anti-CD3 response by >30%. **Optimal dilutions should be determined by each laboratory for each application.**
Endotoxin Level	<0.10 EU per 1 µg of the protein by the LAL method.
Purity	>95%, by SDS-PAGE visualized with Silver Staining and quantitative densitometry by Coomassie® Blue Staining.
Formulation	Lyophilized from a 0.2 µm filtered solution in PBS. See Certificate of Analysis for details.

PREPARATION AND STORAGE

Reconstitution	Reconstitute at 100 µg/mL in sterile PBS.
Shipping	The product is shipped at ambient temperature. Upon receipt, store it immediately at the temperature recommended below.
Stability & Storage	**Use a manual defrost freezer and avoid repeated freeze-thaw cycles.** ● 12 months from date of receipt, -20 to -70 °C as supplied. ● 1 month, 2 to 8 °C under sterile conditions after reconstitution. ● 3 months, -20 to -70 °C under sterile conditions after reconstitution.

BACKGROUND

B7-H4, also known as B7x and B7S1, is a 50-80 kDa glycosylated member of the B7 family of immune co-stimulatory proteins (1, 2). Mature mouse B7-H4 consists of a 230 amino acid (aa) extracellular domain (ECD) with one Ig-like V-set domain and one Ig-like C2-set domain which is followed by a hydrophobic C-terminal region (3-5). Within the ECD, mouse B7-H4 shares 90% and 99% aa sequence identity with human and rat B7-H4, respectively. It shares 21%-29% aa sequence identity with mouse B7-1, B7-2, B7-H1, B7-H2, B7-H3, and PD-L2. B7-H4 is expressed on the surface of activated lymphocytes, macrophages, monocytes, dendritic cells, epithelial cells, and bone marrow-derived mesenchymal stem cells (4-8). Its binding to activated T cells dampens T cell responses and induces cell cycle arrest in the T cell (3-5). Reverse signaling can induce either cell cycle arrest or apoptosis in the B7-H4 expressing cell (9, 10). B7-H4 is up-regulated in several carcinomas in correlation with tumor progression and metastasis (2, 7, 11, 12). A soluble form of B7-H4 is elevated in the serum of ovarian cancer, renal cell carcinoma, and rheumatoid arthritis patients, also in correlation with advanced disease status (13-15). Soluble B7-H4 functions as a decoy molecule that blocks the inhibitory influence of B7-H4 on immune activation (15). Despite evidence for the involvement of B7-H4 in immune regulation, mice deficient in its expression do not show significant immune deficiencies, suggesting compensation by other molecules *in vivo* (16).

References:

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