Human B7-H4
Alexa Fluor® 647-conjugated Antibody

Monoclonal Mouse IgG_{2A} Clone # 973816
Catalog Number: FAB65761R
100 µg

DESCRIPTION

Species Reactivity	Human
Specificity	Detects human B7-H4 in direct ELISAs.
Source	Monoclonal Mouse IgG_{2A} Clone # 973816
Purification	Protein A or G purified from hybridoma culture supernatant
Immunogen	Mouse myeloma cell line NS0-derived recombinant human B7-H4 Phe29-Ala258 Accession # Q7Z7D3
Conjugate	Alexa Fluor 647 Excitation Wavelength: 650 nm Emission Wavelength: 668 nm
Formulation	Supplied 0.2 mg/mL in a saline solution containing BSA and Sodium Azide. *Contains <0.1% Sodium Azide, which is not hazardous at this concentration according to GHS classifications. Refer to the Safety Data Sheet (SDS) for additional information and handling instructions.

APPLICATIONS

Please Note: *Optimal dilutions should be determined by each laboratory for each application. General Protocols are available in the Technical Information section on our website.*

	Recommended Concentration	Sample
Flow Cytometry	0.25-1 µg/10^6 cells	HEK293 Human Cell Line Transfected with Human B7-H4 and eGFP

PREPARATION AND STORAGE

Shipping	The product is shipped with polar packs. Upon receipt, store it immediately at the temperature recommended below.
Stability & Storage	**Protect from light. Do not freeze.** • 12 months from date of receipt, 2 to 8 °C as supplied.

BACKGROUND

B7-H4, also known as VTCN1, B7x and B7S1, is a 50-80 kDa glycosylated member of the BTN/MOG family of immunomodulatory protein (1, 2). Mature human B7-H4 consists of a 235 amino acid (aa) extracellular domain (ECD) with one Ig-like V-set domain and one Ig-like C2-set domain, a 21 aa transmembrane segment, and a 2 aa cytoplasmic tail (3-5). Within the ECD, human B7-H4 shares 90% aa sequence identity with mouse and rat B7-H4. It shares 22%-28% aa sequence identity with human B7-1, B7-2, B7-H1, B7-H2, B7-H3, and PD-L2. Alternate splicing of human B7-H4 generates an additional isoform that lacks the first Ig-like domain. 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). Following binding to activated T cells, B7-H4 serves as a co-inhibitor of the T cell response. This is accomplished by reverse signaling that can induce either cell cycle arrest, or apoptosis in B7-H4 expressing cells (3-5, 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).

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