

DESCRIPTION

Species Reactivity	Human
Specificity	Detects human B7-H4 in direct ELISAs.
Source	Monoclonal Mouse IgG _{2A} Clone # 973815
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 0.2mg/ml in 1X PBS with RDF1 and 0.09% 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.

ELISA Optimal dilution of this antibody should be experimentally determined.

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).

PRODUCT SPECIFIC NOTICES

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