

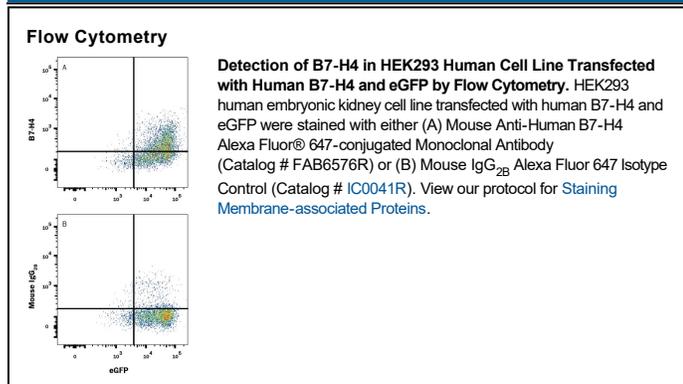
DESCRIPTION	
Species Reactivity	Human
Specificity	Stains human B7-H4 transfectants but not irrelevant transfectants in flow cytometry.
Source	Monoclonal Mouse IgG _{2B} Clone # 935317
Purification	Protein A or G purified from hybridoma culture supernatant
Immunogen	NS0 mouse myeloma cell line transfected with human B7-H4 Phe29-Ser258 Accession # Q7Z7D3
Conjugate	Alexa Fluor 647 Excitation Wavelength: 650 nm Emission Wavelength: 668 nm
Formulation	Supplied in a saline solution containing BSA and Sodium Azide. See Certificate of Analysis for details. *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	5 µL/10 ⁶ cells	See Below

DATA



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. <ul style="list-style-type: none"> ● 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 two Ig-like V-type domains, 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 (2-5, 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, and is correlated 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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