

DESCRIPTION

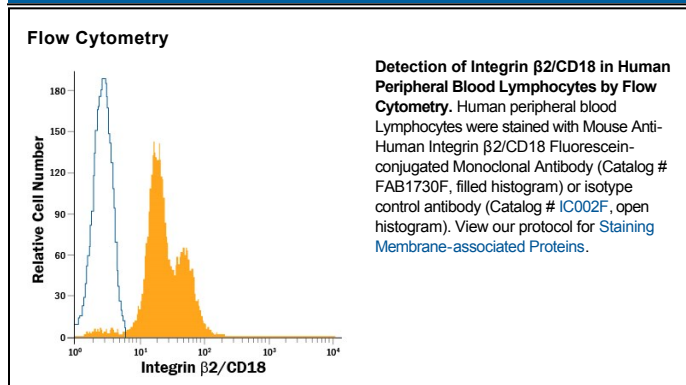
Species Reactivity	Human
Specificity	Detects human Integrin β 2/CD18 in direct ELISAs and Western blots. In direct ELISAs, no cross-reactivity with recombinant human Integrin β 1 or recombinant mouse Integrin α 5 is observed.
Source	Monoclonal Mouse IgG ₁ Clone # 212701
Purification	Protein A or G purified from hybridoma culture supernatant
Immunogen	Mouse myeloma cell line NS0-derived recombinant human Integrin β 2/CD18 Gln23-Asn700 Accession # AAA59490
Conjugate	Fluorescein Excitation Wavelength: 488 nm Emission Wavelength: 515-545 nm (FITC)
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	10 μ L/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

Integrin α X β 2, also called CD11c/CD18, p150/95 or complement receptor type 4 (CR4), is one of four β 2 integrins. The non-covalent heterodimer of 150 kDa α X/CD11c and 95 kDa β 2/CD18 integrin subunits is expressed on macrophages, dendritic cells and hairy cell leukemias, with lower amounts on other myeloid cells and activated B, NK and some cytotoxic T cells (1-7). Like other integrins, α X β 2 has multiple activation states (3). In the presence of divalent cations and "inside-out" signaling, α X β 2 is fully active and extended. The α X vWFA or I-domain, which contains the adhesion sites, forms the N-terminal head region with the α X beta-propeller and the β 2 vWFA domain (1, 8). In the inactive state, the heterodimer flexes in the center at the α X thigh and calf domains and β 2 I-EGF domains, impeding access to adhesion sites (1). The 1088 aa human α X/CD11c ECD shares 70-76% aa sequence identity with mouse, rat and canine α X while the 678 aa human β 2/CD18 ECD shares 81-83% aa sequence identity with mouse, rat, cow, dog, goat, sheep, and pig β 2. Potential α X isoforms containing 719 and 725 aa (as compared to full-length 1163 aa α X) lack the vWFA domain and the N-terminus. Active α X β 2 shares some adhesion partners with α M β 2/CD11b/CD18, including complement opsonin fragment iC3b, ICAMs, vWF and fibrinogen, and is expressed on many of the same cells (4-11). However, α M β 2 activity is often constitutive, while α X β 2 activity requires cell activation (4-7). α X β 2 also binds osteopontin, Thy-1, plasminogen, heparin, and proteins with abnormally exposed acidic residues (11-16). The adhesion events are important for proliferation, degranulation, chemotactic migration, and phagocytosis of complement-opsonized particles (5, 6, 9, 11, 12, 16). Mutations of β 2, especially in the vWFA domain, cause leukocyte adhesion deficiency (LAD-1) and susceptibility to bacterial infections (17).

References:

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