Human CD59 Alexa Fluor® 350-conjugated Antibody

Recombinant Monoclonal Rabbit IgG Clone # 2491C
Catalog Number: FAB1987U
100 µg

DESCRIPTION

Species Reactivity	Human
Specificity	Detects human CD59 in direct ELISAs.
Source	Recombinant Monoclonal Rabbit IgG Clone # 2491C
Purification	Protein A or G purified from cell culture supernatant
Immunogen	Mouse myeloma cell line, NS0-derived recombinant human CD59 Leu26-Asn102 Accession # P13987
Conjugate	Alexa Fluor 350 Excitation Wavelength: 346 nm Emission Wavelength: 442 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	Human PBMC lymphocytes

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

CD59, also known as membrane attack complex inhibition factor (MACIF) and Protectin, is an approximately 20 kDa GPI-anchored glycoprotein that is an important regulator of the complement system in blood. The complement system triggers innate immune responses to immune complexes, MBL-opsonized microorganisms, and apoptotic cells through the classical, lectin, and alternative pathways. One major consequence of complement activation is the assembly of a membrane attack complex (MAC) composed of one molecule each of complement proteins C5b, C6, C7, and C8 (C5b-8) followed by the incorporation of multiple copies of C9 (C5b-9). Membrane insertion of the MAC results in formation of a cytolytic pore in the target cell (1). CD59, which is widely expressed on healthy cells, binds to both C8 and C9 and shields them from complement-mediated lysis. It inhibits MAC pore formation by blocking C5b-8 complex membrane insertion and the incorporation of C9 molecules (2-4). The binding of CD59 to C8 and C9 is species-selective, and this contributes to the restricted ability of MACs to lyse cells of other species (5). The cytoprotective function of CD59 plays a variety of roles in pathology. It limits tissue damage and inflammation following ischemia/reperfusion injury (6, 7). It also protects against the development of atherosclerosis and abdominal aortic aneurysms (8, 9). Its protectiveness can be inactivated by diabetes-induced glycation, leading to increased MAC deposition and hemolytic anemia (10). In contrast, CD59 can be exploited to promote red cell lysis; it functions as a cellular receptor for the bacterial pore-forming toxin Intermedilysin (11). CD59 can be incorporated into several enveloped viruses such as hepatitis C virus where it limits the destruction of virus particles (12). Aside from its complement regulatory functions, CD59 limits the activation of T cells following their interaction with antigen presenting cells (13), but it promotes NK cell activation through association with NKp30 and NKp46 (14). In mouse, gene duplication has given rise to two related proteins, CD59a and CD59b. Mature human CD59 shares 37%, 43%, and 44% amino acid sequence identity with mouse CD59a, mouse CD59b, and rat CD59, respectively (15).

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