Human α-2A Adrenergic R/ADRA2A Alexa Fluor® 488-conjugated Antibody

Monoclonal Mouse IgG_1 Clone # 1006614

Catalog Number: FAB10129G

100 µg

DESCRIPTION

Species Reactivity	Human
Specificity	Detects human α-2A Adrenergic R/ADRA2A in direct ELISAs.
Source	Monoclonal Mouse IgG_1 Clone # 1006614
Purification	Protein A or G purified from hybridoma culture supernatant
Immunogen	Synthetic peptide containing Human α-2A Adrenergic R/ADRA2A
Conjugate	Alexa Fluor 488 Excitation Wavelength: 488 nm Emission Wavelength: 515-545 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 ADRA2A 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

Alpha-2-adrenergic receptors, including ADRA2A, are members of the G protein-coupled receptor superfamily. They include 3 highly homologous subtypes: alpha2A, alpha2B, and alpha2C. These receptors have a critical role in regulating neurotransmitter release from sympathetic nerves and from adrenergic neurons in the central nervous system. Studies in mouse revealed that both ADRA2A and ADRA2C subtypes were required for normal presynaptic control of transmitter release from sympathetic nerves in the heart and from central noradrenergic neurons. The ADRA2A subtype inhibited transmitter release at high stimulation frequencies, whereas the ADRA2C subtype modulated neurotransmission at lower levels of nerve activity.

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