Human α-2B Adrenergic R/ADRA2B Alexa Fluor® 750-conjugated Antibody

Monoclonal Mouse IgG_1 Clone # 491613

Catalog Number: FAB10324S

100 µg

DESCRIPTION

Species Reactivity	Human
Specificity	Detects human α-2B Adrenergic R/ADRA2B in direct ELISAs.
Source	Monoclonal Mouse IgG_1 Clone # 491613
Purification	Protein A or G purified from ascites
Immunogen	NS0 mouse myeloma cell line transfected with human α-2B Adrenergic R/ADRA2B Accession # P18089
Conjugate	Alexa Fluor 750 Excitation Wavelength: 749 nm Emission Wavelength: 775 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 ADRA2B 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 ADRA2B, are members of the G protein-coupled receptor superfamily. They include 3 highly homologous subtypes: alpha2A, alpha2B, and alpha2C that have a critical role in regulating neurotransmitter release from sympathetic nerves and from adrenergic neurons in the central nervous system. ADRA2B was observed to associate with eIF-2B, a guanine nucleotide exchange protein that functions in regulation of translation. A polymorphic variant of ADRA2B was identified to have decreased G protein-coupled receptor kinase-mediated phosphorylation and desensitization; this polymorphic form is also associated with reduced basal metabolic rate in obese subjects and may therefore contribute to the pathogenesis of obesity.

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