

Human Bradykinin RB2/BDKRB2 Alexa Fluor® 350-conjugated Antibody

Monoclonal Mouse IgG₁ Clone # 471902 Catalog Number: FAB9434U

100 µg

DESCRIPTION		
Species Reactivity	Human	
Specificity	Detects human RB2/BDKRB2 in direct ELISAs and Western blots.	
Source	Monoclonal Mouse IgG ₁ Clone # 471902	
Purification	Protein A or G purified from hybridoma culture supernatant	
Immunogen	NS0 mouse myeloma cell line transfected with human RB2/BDKRB2 Met1-Gln391 Accession # P30411	
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. 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	0.25-1 μg/10 ⁶ cells	HEK293 Human Cell Line Transfected with Human Bradykinin RB2/BDKRB2 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.		

BACKGROUNE

Bradykinin RB2 (BDKRB2) is a receptor for bradykinin. The 9 aa bradykinin peptide elicits many responses including vasodilation, edema, smooth muscle spasm and pain fiber stimulation. BDKRB2 expression is widespread in normal smooth muscle tissue and neurons. BDKRB2 associates with G proteins that stimulate a phosphatidylinositol-calcium second messenger system. BDKRB2 forms a complex with PECAM1 and GNAQ and interacts with PECAM1. Aging cardiac endothelial cells gradually lose their capacity to express BDKRB2. This loss appears to be parallel with a loss of the angiogenic potential of the aging cells.

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