

DESCRIPTION

Species Reactivity	Human
Specificity	Detects human GPR158 in direct ELISAs.
Source	Monoclonal Mouse IgG _{2B} Clone # 1027651
Purification	Protein A or G purified from hybridoma culture supernatant
Immunogen	Human embryonic kidney cell HEK293-derived human GPR158 protein Ala24-Gln411 Accession # Q5T848
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 GPR158 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

G-protein coupled receptor 158 (GPR158) is a receptor belonging to the Class C GPCR family. It lacks the extracellular Venus flytrap module characteristic of the known members of that family and instead contains two other elements that are not typical of the class: a calcium-binding EGF-like domain and a leucine repeat region (1, 2). The mature extracellular domain of human GPR158 contains 393 amino acids (aa) and shares 89% identity with both mouse and rat GPR158. GPR158 is expressed at the highest level in the brain, but also in a variety of other tissues including retina, spleen, liver and lung (3). GPR158 was originally identified in functional screens linked with biological stress and has been implicated in the osteocalcin effect on cognitive processes in the brain (4, 5), and glaucoma and cancer in the periphery (4, 6).

References:

1. Jingami, H. *et al.* (2003) *Curr. Opin. Neurobiol.* **13**:271.
2. Bjarnadóttir, T.K *et al.* (2005) *Gene*. **362**:70.
3. Orlandi, C. *et al.* (2012) *J. Cell Biol.* **197**:711.
4. Itakura, T. *et al.* (2019) *J. Ocul. Pharmacol. Ther.* **35**:203.
5. Khirman, L. *et al.* (2017) *J. Exp. Med.* **214**:2859.
6. Fenner, A. (2015) *Nat. Rev. Urol.* **12**:182.

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