Rat IgG1 kappa Isotype Control (eBRG1), PerCP-eFluor™ 710, eBioscience™

Product Details	
Size	25 µg
Host/Isotype	Rat / IgG1, kappa
Class	Control
Type	Isotype Control
Clone	eBRG1
Conjugate	PerCP-eFluor™ 710
Excitation/Emission Max	482/708 nm
Form	Liquid
Concentration	0.2 mg/mL
Purification	Affinity chromatography
Storage buffer	PBS, pH 7.2
Contains	0.09% sodium azide
Storage conditions	4° C, store in dark, DO NOT FREEZE!
RRID	AB_1944422

Applications	Tested Dilution	Publications
Flow Cytometry (Flow)	Assay-Dependent	0 Publication
Control (Ctrl)	Assay-Dependent	-

Product Specific Information

Description: The monoclonal rat IgG1, kappa is useful as an isotype control immunoglobulin.

Applications Reported: PerCP-eFluor® 710 Rat IgG1 Isotype Control has been reported for use in flow cytometric analysis.

Applications Tested: PerCP-eFluor® 710 Rat IgG1 Isotype Control has been tested by flow cytometric analysis of mouse splenocytes and normal human peripheral blood cells. Isotype control should be used at the same concentration as experimental antibody.

PerCP-eFluor® 710 can be used in place of PE-Cy5, PE-Cy5.5 or PerCP-Cy5.5. PerCP-eFluor® 710 emits at 710 nm and is excited with the blue laser (488 nm). Please make sure that your instrument is capable of detecting this fluorochrome. For a filter configuration, we recommend using the 685 LP dichroic mirror and 710/40 band pass filter, however the 695/40 band pass filter is an acceptable alternative.

Our testing indicates that PerCP-eFluor® 710 conjugated antibodies are stable when stained samples are exposed to freshly prepared 2% formaldehyde overnight at 4°C, but please evaluate for alternative fixation protocols.

Excitation: 488 nm; Emission: 710 nm; Laser: Blue Laser.

Filtration: 0.2 µm post-manufacturing filtered.

2 References

VprBP Is Required for Efficient Editing and Selection of Ig+ B Cells, but Is Dispensable for Ig+ and Marginal Zone B Cell Maturation and Selection. J Immunol (2015)

Chronic thoracic spinal cord injury impairs CD8+ T-cell function by up-regulating programmed cell death-1 expression. J Neuroinflammation (2014)