

DESCRIPTION	
<b>Species Reactivity</b>	Mouse
<b>Specificity</b>	Stains mouse CCR8 transfectants but not irrelevant transfectants in flow cytometry.
<b>Source</b>	Monoclonal Rabbit IgG Clone # 1055C
<b>Purification</b>	Protein A or G purified from cell culture supernatant
<b>Immunogen</b>	HEK293 human embryonic kidney cell line transfected with mouse CCR8 Accession # P56484
<b>Conjugate</b>	Alexa Fluor 647 Excitation Wavelength: 650 nm Emission Wavelength: 668 nm
<b>Formulation</b>	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		
<b>Please Note:</b> Optimal dilutions should be determined by each laboratory for each application. <i>General Protocols</i> are available in the <i>Technical Information</i> section on our website.		
	<b>Recommended Concentration</b>	<b>Sample</b>
<b>Flow Cytometry</b>	0.25-1 µg/10 <sup>6</sup> cells	HEK293 human embryonic kidney cell line transfected with mouse CCR8 and eGFP

PREPARATION AND STORAGE	
<b>Shipping</b>	The product is shipped with polar packs. Upon receipt, store it immediately at the temperature recommended below.
<b>Stability &amp; Storage</b>	<b>Protect from light. Do not freeze.</b> <ul style="list-style-type: none"> <li>12 months from date of receipt, 2 to 8 °C as supplied.</li> </ul>

**BACKGROUND**

CCR8 (C-C chemokine Receptor 8; also known as CD198) is a 41-43 kDa member of the GPCR #1 family of transmembrane proteins. Mouse CCR8 is expressed on vascular smooth muscle cells, monocytes, eosinophils, peritoneal macrophages, thymocytes, CD8<sup>+</sup> T cells, Langerhans cells and neurons. CCL1/TCA3 and vMIP-1 are known agonists for CCR8. Mouse CCR8 is a 7-transmembrane protein that is 353 amino acids (aa) in length. It contains a 33 aa N-terminal extracellular domain plus a 50 aa C-terminal cytoplasmic tail. In mouse, CCR8 is N- and possibly O-glycosylated, and known to be sulfated on Tyr14 and 15. The unusual nature of these posttranslational modifications may lead to anomalous migration in SDS-PAGE. There are two potential isoforms, one that shows a deletion of aa 103-163, and another that shows a Met substitution for aa 125-166. Over aa sequences 1-33 and 92-105 collectively, mouse CCR8 shares 64% and 85% aa identity with human and rat CCR8, respectively.

**PRODUCT SPECIFIC NOTICES**

This product is provided under an agreement between Life Technologies Corporation and R&D Systems, Inc, and the manufacture, use, sale or import of this product is subject to one or more US patents and corresponding non-US equivalents, owned by Life Technologies Corporation and its affiliates. The purchase of this product conveys to the buyer the non-transferable right to use the purchased amount of the product and components of the product only in research conducted by the buyer (whether the buyer is an academic or for-profit entity). The sale of this product is expressly conditioned on the buyer not using the product or its components (1) in manufacturing; (2) to provide a service, information, or data to an unaffiliated third party for payment; (3) for therapeutic, diagnostic or prophylactic purposes; (4) to resell, sell, or otherwise transfer this product or its components to any third party, or for any other commercial purpose. Life Technologies Corporation will not assert a claim against the buyer of the infringement of the above patents based on the manufacture, use or sale of a commercial product developed in research by the buyer in which this product or its components was employed, provided that neither this product nor any of its components was used in the manufacture of such product. For information on purchasing a license to this product for purposes other than research, contact Life Technologies Corporation, Cell Analysis Business Unit, Business Development, 29851 Willow Creek Road, Eugene, OR 97402, Tel: (541) 465-8300. Fax: (541) 335-0354.