

# Product Data Sheet

## Alexa Fluor® 647 anti-human CD186 (CXCR6)

**Catalog # / Size:** 2275505 / 25 tests

**Clone:** TG3/CXCR6

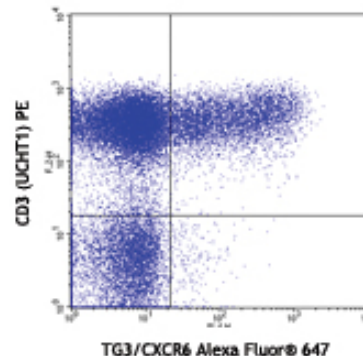
**Isotype:** Mouse IgG2b,  $\kappa$

**Reactivity:** Human

**Preparation:** The antibody was purified by affinity chromatography, and conjugated with Alexa Fluor® 647 under optimal conditions.

**Formulation:** Phosphate-buffered solution, pH 7.2, containing 0.09% sodium azide and 0.2% (w/v) BSA (origin USA).

**Storage:** The antibody solution should be stored undiluted between 2°C and 8°C, and protected from prolonged exposure to light. **Do not freeze.**



Human peripheral blood mononuclear cells stimulated with IL-2 for 9 days and then stained with CD3 (UCHT1) PE and TG3/CXCR6 Alexa Fluor® 647

## Applications:

**Applications:** FC - Quality tested

**Recommended Usage:** Each lot of this antibody is quality control tested by immunofluorescent staining with flow cytometric analysis. For flow cytometric staining, the suggested use of this reagent is 5  $\mu$ l per million cells or 5  $\mu$ l per 100  $\mu$ l of whole blood. It is recommended that the reagent be titrated for optimal performance for each application.

\* Alexa Fluor® 647 has a maximum emission of 668 nm when it is excited at 633nm / 635nm.

**Application References:**

1. Walker LJ., *et al.* 2012. *Blood*. 119:422. PubMed.
2. Havenith SH, *et al.* 2012. *Int Immunol*. 24:625. PubMed.
3. Tang XZ, *et al.* 2013. *J. Immunol*. 190:3142. PubMed.

**Description:** CXCR6 is a chemokine receptor that binds CXCL16. It is expressed in Th1 inflammatory diseases such as rheumatoid arthritis (3) and Grave's disease (4) among others. Th1 cells isolated from the bronchoalveolar lavage of patients with sarcoidosis and T-cell alveolitis coexpressed CXCR3 and CXCR6. The CXCR6 ligand CXCL16 was abundantly expressed by macrophages infiltrating sarcoid tissue and/or forming the granuloma core (5). Recent data showed that CXCR6 is expressed on polymorphonuclear neutrophils in pancreatic carcinoma and in acute localized bacterial infections. CXCR6 is not constitutively expressed on PMN, but is up-regulated under inflammatory conditions and mediates migration of CXCR6-positive PMN (6). Fetus-derived trophoblasts can attract T cells, gamma delta T cells, and monocytes by producing CXCL16 and interaction with CXCR6 on these cells, leading to forming a specialized immune milieu at the maternofetal interface (7).

**Antigen References:**

- 1) Kim CH, *et al.* 2001. *J Clin Invest* 107:595.
- 2) Nanki T, *et al.* 2005. *Arthritis Rheum* 52:3004.
- 3) Van der Vort R *et al.* 2005. *Arthritis Rheum* 52:1381.
- 4) Aust G *et al.* 2005. *Eur J Endocrin* 152:635-643.
- 5) Agostini C, *et al.* 2005. *Am j Respir Crit Care Med* 172:1290.
- 6) Gaida MM, *et al.* 2008. *Clin Exp Immunol*.
- 7) Huang J, *et al.* 2008. *J. Immunol*. 180:2367.

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