Product Data Sheet

Product Name: Carlumab
Cat. No.: GC68832

Chemical Properties

Cas. No.	915404-94-3	
Formula	M.Wt	114.87 kDa
Solubility	Storage	Store at -80°C for 2 years.
General tips	For obtaining a higher solubility , please warm the tube at 37 ℃ and shake it in the ultrasonic bath for a while.Stock solution can be stored below -20℃ for several months.	
Shipping Condition	Evaluation sample solution : ship with blue ice All other available size: ship with RT , or blue ice upon request.	
Structure	Carlumab	

Protocol

Cell experiment [1]:

Cell lines	Human peritoneal mesothelial cells (HPMCs) and PMA-differentiated THP-1 macrophages
Preparation Method	HPMCs were cultured in M199 medium supplemented with 10% FBS. To simulate EPS conditions, HPMCs in the lower chamber of a Trans-well system were treated with 0.1% SHS and 1μg/mL LPS. Carlumab (30μg/mL) or an anti-IgG control was added to the HPMCs 30 minutes prior to the EPS stimulation. PMA-differentiated THP-1 macrophages were seeded in the upper chamber.
Reaction Conditions	30μg/mL; pretreatment for 30min.

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Applications	Pretreatment with Carlumab significantly attenuated the migration of macrophages towards the EPS-stimulated mesothelial cells in the co-culture system.

Animal experiment [2]:

Animal models	Female BALB/c nude mice (aged 4-5 weeks) bearing KLE cell xenograft tumors.
Preparation Method	KLE cells were transplanted into the forelimb armpit of mice to establish a xenograft model. Mice were administered physiological saline, Carlumab, the ERRα inhibitor XCT790, or a combination of Carlumab (20mg/kg) and XCT790 via intraperitoneal injection for 29 days.
Dosage form	20mg/kg; i.p.; for 29 days.
Applications	Carlumab administration, either alone or in combination with XCT790, significantly reduced tumor volume, decreased serum CCL2 levels, and reduced the infiltration of CD163+ and CD115+ M2-type macrophages within the tumor tissue.

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References:
[1] Sun J, Sun Y, Guo D, et al. Targeting STING to disrupt macrophage-mediated adhesion in encapsulating peritoneal sclerosis. Commun Biol. 2025 Aug 23;8(1):1266.
[2] Ma J, Mao X, Ren Y, et al. Antagonism of estrogen-related receptor-α inhibits mitochondrial oxidative phosphorylation and reduces M2 macrophage infiltration in endometrial cancer. J Immunother Cancer. 2025 Sep 29;13(9):e012521.

Background

Carlumab is a humanized, high-affinity monoclonal antibody that targets CCL2[1-2]. Carlumab exerts its effects by specifically binding to and neutralizing CCL2, thereby inhibiting the recruitment and migration of monocytes/macrophages to the tumor microenvironment and reducing angiogenesis. Carlumab has been investigated in research related to solid tumors such as prostate cancer, ovarian cancer, and glioblastoma, as well as idiopathic pulmonary fibrosis[3-4].

In vitro, human peritoneal mesothelial cells were pretreated with Carlumab (30μg/mL) for 30 minutes, followed by stimulation for 24 hours with a medium containing 0.1% SHS (containing chlorhexidine) and 1μg/mL LPS. Carlumab significantly attenuated the migration of THP-1-derived macrophages towards the injured site in the co-culture system[5]. Endometrial cancer cells were treated with Carlumab (5μg/mL) for 24 hours. This treatment was able to neutralize tumor cell-secreted CCL2, significantly inhibiting the migration of THP-1-derived macrophages towards the tumor cells[6].

In vivo, Carlumab (20mg/kg) was administered via intraperitoneal injection to BALB/c

nude mice bearing KLE cell xenograft tumors for 29 days. Carlumab treatment effectively reduced tumor volume, decreased serum CCL2 levels, and reduced the infiltration of CD163+ and CD115+ M2-type macrophages within the tumor tissue[6].

References:

[1] Brana I, Calles A, LoRusso PM, et al. Carlumab, an anti-C-C chemokine ligand 2 monoclonal antibody, in combination with four chemotherapy regimens for the treatment of patients with solid tumors: an open-label, multicenter phase 1b study. Target Oncol. 2015 Mar;10(1):111-23.
[2] Raghu G, Martinez FJ, Brown KK, et al. CC-chemokine ligand 2 inhibition in idiopathic pulmonary fibrosis: a phase 2 trial of carlumab. Eur Respir J. 2015 Dec;46(6):1740-50.
[3] Fetterly GJ, Aras U, Meholick PD, et al. Utilizing pharmacokinetics/pharmacodynamics modeling to simultaneously examine free CCL2, total CCL2 and carlumab (CNTO 888) concentration time data. J Clin Pharmacol. 2013 Oct;53(10):1020-7.
[4] Wang C, Wang L, Li Q, et al. Computational Drug Discovery in Ankylosing Spondylitis-Induced Osteoporosis Based on Data Mining and Bioinformatics Analysis. World Neurosurg. 2023 Jun;174:e8-e16.
[5] Sun J, Sun Y, Guo D, et al. Targeting STING to disrupt macrophage-mediated adhesion in encapsulating peritoneal sclerosis. Commun Biol. 2025 Aug 23;8(1):1266.
[6] Ma J, Mao X, Ren Y, et al. Antagonism of estrogen-related receptor-α inhibits mitochondrial oxidative phosphorylation and reduces M2 macrophage infiltration in endometrial cancer. J Immunother Cancer. 2025 Sep 29;13(9):e012521.

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