Product Data Sheet

Product Name: Trastuzumab (Anti-Human HER2, Humanized Antibody)
Cat. No.: GC34215

Chemical Properties

Cas. No.	180288-69-1		
SMILES	[Trastuzumab]		
Formula	$C_{6470}H_{10012}N_{1726}O_{2013}S_{42}$	M.Wt	145145.09
Solubility		Storage	-20 ℃, avoid multiple freeze-thaw cycles.
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			

Protocol

Cell experiment [1]:

Cell lines	Breast cancer HER2+ cells BT474 and SKBR3
Preparation Method	Cells were seeded on 48-wells plates and were treated 24h later with indicated doses of each drug (MZ1 and trastuzumab), alone or in combination. Cell medium was replaced at 72h with MTT solution for 45min at 37℃.
Reaction Conditions	25-100nM MZ1, 10nM trastuzumab, 25-100nM MZ1+trastuzumab

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Applications	MZ1 showed the highest antiproliferative effect. Trastuzumab combined with MZ1 induced a more profound antiproliferative effect than the administration of single agents in both BT474 and SKBR3 cells. A synergistic interaction between trastuzumab and MZ1 in BT474 cells and an additive interaction in SKBR3 cells was observed.

Animal experiment [2]:

Animal models	Female BALB/c nude mice, 4-6 weeks
Preparation Method	Xenografts were used for the experiments once the tumor volume reached about 150-200mm^3. Mice were randomly assigned to different groups as follows: (i) vehicle; (ii) trastuzumab 10mg/kg twice weekly of intraperitoneal injection; (iii) AZD5438 20mg/kg daily by oral gavage; (iv) AZD5438+trastuzumab, for 3 weeks. Experiments were ended once the tumor volume surpassed 1500mm3 or mouse weight loss reached 20%.
Dosage form	10mg/kg trastuzumab, 20mg/kg AZD5438

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Applications	Trastuzumab exerted antitumor effects in ERBB2-positive AFPGC PDX models. Statistically significant differences were also present in the tumor volume between the group treated with AZD5438 combined with trastuzumab and those treated with AZD5438 or trastuzumab alone in ERBB2 and CCNE1 co-amplified PDX models, but the results were not observed in CCNE1 non-amplified PDX models.

References:

[1]. Noblejas-López MDM, Nieto-Jiménez C, et al. MZ1 co-operates with trastuzumab in HER2 positive breast cancer. J Exp Clin Cancer Res. 2021 Mar 19;40(1):106.

[2]. Lu J, Ding Y, et al. Whole-exome sequencing of alpha-fetoprotein producing gastric carcinoma reveals genomic profile and therapeutic targets. Nat Commun. 2021;12(1):3946. Published 2021 Jun 24. doi:10.1038/s41467-021-24170-0

Background

Trastuzumab is a fully humanized monoclonal antibody directed at HER2 which binds the external domain of the receptor and exerts its action via a combination of antibody-dependent cytotoxicity, reduced shedding of the extracellular domain, inhibition of

dimerization and possibly receptor downregulation[1,2]. Trastuzumab is used as a standard treatment for breast and metastatic gastric cancer when the cancer cells overexpress HER2[3].

Trastuzumab combined with MZ1 significantly decreased cell proliferation, the formation of three-dimensional structures and cellular invasion compared to either of the drugs alone[4]. Trastuzumab treated HER2-overexpressing breast cancer cell lines results in induction of p27KIP1 and the Rb-related protein, p130, which in turn significantly reduces the number of cells undergoing S-phase. A number of other phenotypic changes are observed in vitro as a consequence of trastuzumab binding to HER2-overexpressing cells[5]. Trastuzumab-dendrimer-fluorine drug delivery system is a new form of trastuzumab to treat breast cancer cells in vitro. The potential of Trastuzumab-dendrimer-fluorine drug delivery system is more efficient than trastuzumab alone[6]

Trastuzumab causes a significant growth inhibition of the outgrowth of macroscopic JIMT-1 xenograft tumors in both nude and SCID mice[7]. The administration of MZ1 and trastuzumab induced a reduction in tumor progression, while individual treatments failed to do so[4]

References:
[1]. Ning G, Zhu Q, et al. A novel treatment strategy for lapatinib resistance in a subset of HER2-amplified gastric cancer. BMC Cancer. 2021;21(1):923. Published 2021 Aug 16. doi:10.1186/s12885-021-08283-9
[2]. Okines AF, Cunningham D. Trastuzumab: a novel standard option for patients with HER-2-positive advanced gastric or gastro-oesophageal junction cancer. Therap Adv Gastroenterol. 2012 Sep;5(5):301-18.
[3]. Sarosiek T, Morawski P. Trastuzumab and its biosimilars [Trastuzumab and its biosimilars]. Pol Merkur Lekarski. 2018 May 25;44(263):253-257. Polish.
[4]. Noblejas-López MDM, Nieto-Jiménez C, et al. MZ1 co-operates with trastuzumab in HER2 positive breast cancer. J Exp Clin Cancer Res. 2021 Mar 19;40(1):106.
[5]. Sliwkowski MX, Lofgren JA, et al. Nonclinical studies addressing the mechanism of action of trastuzumab (Herceptin). Semin Oncol. 1999 Aug;26(4 Suppl 12):60-70.
[6]. Bartusik-Aebisher D, Chrzanowski G, et al. An analytical study of Trastuzumab-dendrimer-fluorine drug delivery system in breast cancer therapy in vitro. Biomed Pharmacother. 2021 Jan;133:111053.

[7]. Barok M, Isola J, et al. Trastuzumab causes antibody-dependent cellular cytotoxicity-mediated growth inhibition of submacroscopic JIMT-1 breast cancer xenografts despite intrinsic drug resistance. Mol Cancer Ther. 2007 Jul;6(7):2065-72.

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