Product Data Sheet

Product Name: Cetuximab (C225)
Cat. No.: GC34217

Chemical Properties

Cas. No.	205923-56-4		
SMILES	[Cetuximab]		
Formula	$C_{6484}H_{10042}N_{1732}O_{2023}S_{36}$	M.Wt	145543.35
Solubility		Storage	Store at 4°C, do not freeze
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	Cetuximab (C225)		

Protocol

Cell experiment [1]:

Cell lines	SCC1 and UM-SCC-22B cells
Preparation Method	The toxicity of cetuximab to SCC1 and UM-SCC-22B cells was determined by MTT assay. At 72 h or 120 h after treatment with different doses of cetuximab (ranging from 0.1 nM to 0.5 μM), the culture medium was replaced and 50 μl of 1.0 mg/ml sterile filtered 3-(4, 5-dimethylthiazol-2-yl)-2, 5-diphenyl tetrazolium bromide (MTT; Sigma) was added to each well.
Reaction Conditions	0.1 nM to 0.5 μM at 72 h or 120 h

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Applications	Treatment with cetuximab produced only modest inhibition of cell proliferation on SCC1 cells in vitro as determined by MMT assay.

Animal experiment [2]:

Animal models	BALB/c (nu/nu) female nude mice
Preparation Method	Xenografts were established in female nude mice (BALB c[nu/nu]) by subcutaneous injection of head and neck squamous cell carcinoma cell lines a UT-SCC-14 and b UT-SCC-2. Cetuximab (1 mg/injection) was administered by intraperitoneal injection at day 10, 13 and 16. The tumour size was recorded at an interval of 2–3 days, n = 10-14.
Dosage form	1 mg, i.p.
Applications	Cetuximab treatment showed reduction in the nuclear accumulation of HIF-1α, while the overall HIF-1α expression was not significantly altered. And after cetuximab treatment a downregulation of CAIX was only found in UT-SCC-14 xenografts. Cetuximab treatment affects the tumour growth and the tumour partial oxygen pressure as measured by LiPc EPR oximetry.

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References:

[1]. Niu G, et al. Cetuximab-based immunotherapy and radioimmunotherapy of head and neck squamous cell carcinoma. Clin Cancer Res. 2010 Apr 1;16(7):2095-105.

[2]. Gustafsson H, et al. EPR Oximetry of Cetuximab-Treated Head-and-Neck Tumours in a Mouse Model. Cell Biochem Biophys. 2017 Dec;75(3-4):299-309.

Background

Cetuximab is a chimeric monoclonal antibody generated from fusion of the variable region of the murine anti-EGFR monoclonal antibody M225 and the human IgG1 constant region. It produced antibody retains high affinity and specificity to EGFR and reduces immunogenicity.[1] Cetuximab bound with high affinity to FcγRI (EC50 = 0.13 nM) and FcγRIIIa (EC50 = 6 nM). It effectively induced ADCC across multiple tumor cell lines.[4] Treatment with 100?μg/ml cetuximab for 24h enhances the cytotoxicity effect of RSL3 treatment on KRAS mutant CRC cells.[2]

In vitro experiment indicated it that radiation enhances cetuximab (0.5 μg/ml)-mediated ADCC and activation of NK cells.[3] Treatment with 20 μg/mL cetuximab inhibited the proliferation of the parental UMSCC1 cell line (UMSCC1-P) ,while the three HNSCC cetuximab-resistant clones (C2, C5, and C11) were completely refractory to cetuximab.[6]

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In vivo experiment it shown that cetuximab (13?mg/kg, s.c.) enhances the inhibitory effects of RSL3 and RSL3-induced ferroptosis.[2] In vivo, after i.v. injection of 4 doses of 10 mg/kg body-weight demonstrated that cetuximab markly inhibited tumor growth in SCC1 tumor bearing mice.[5] In vivo experiment it illustrated that cetuximab-treated (50 mg/kg, i.p.) tumors showed delayed growth, when mice were inoculated with the NSCLC H226 cell line individually with 2x106 tumor cells in the dorsal flank.[6]

References:

[1]. Xiong HQ, et al. Cetuximab, a monoclonal antibody targeting the epidermal growth factor receptor, in combination with gemcitabine for advanced pancreatic cancer: a multicenter phase II Trial. J Clin Oncol. 2004 Jul 1;22(13):2610-6.
[2]. Yang J, et al. Cetuximab promotes RSL3-induced ferroptosis by suppressing the Nrf2/HO-1 signalling pathway in KRAS mutant colorectal cancer. Cell Death Dis. 2021 Nov 13;12(11):1079.
[3]. Jin WJ, et al. Tumor-Specific Antibody, Cetuximab, Enhances the In Situ Vaccine Effect of Radiation in Immunologically Cold Head and Neck Squamous Cell Carcinoma. Front Immunol. 2020 Nov 12;11:591139.
[4]. Patel D, et al. IgG isotype, glycosylation, and EGFR expression determine the induction of antibody-dependent cellular cytotoxicity in vitro by cetuximab. Hum Antibodies. 2010;19(4):89-99.
[5]. Niu G, et al. Cetuximab-based immunotherapy and radioimmunotherapy of head and neck squamous cell carcinoma. Clin Cancer Res. 2010 Apr 1;16(7):2095-105.
[6]. Iida M, et al. Targeting the HER Family with Pan-HER Effectively Overcomes Resistance to Cetuximab. Mol Cancer Ther. 2016 Sep;15(9):2175-86.

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