Product Data Sheet

Product Name: Disitamab vedotin
Cat. No.: GC64039

Chemical Properties

Cas. No.	2136633-23-1	
Formula	M.Wt	144.15 kDa(Approximately)
Solubility	Storage	Store at -20°C,protect from light
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	Disitamab vedotin	

Protocol

Cell experiment [1]:

Cell lines	HT1376 cells
Preparation Method	HT1376 cells were grown in DMEM medium supplemented with 10% fetal bovine serum, 100U/ml penicillin, and 0.1mg/ml streptomycin in 5% CO_2 at 37°C. 5×10^3 HT1376 cells/ml were seeded into 96-well microplates for 24h. The wells were replenished with fresh complete medium containing different concentrations of Disitamab vedotin (0, 1, 10, 50, 100, and 200μg/ml) for 48h. Then, the cell viability was detected.
Reaction Conditions	0, 1, 10, 50, 100, and 200μg/ml; 48h

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Applications	Disitamab vedotin treatment significantly decreased the cell viability of HT1376 cells in a dose-dependent manner.

Animal experiment [2]:

Animal models	BALB/c nude mice
Preparation Method	BALB/c nude mice (6 weeks old) were kept in temperature-controlled (21°C-25°C) and humidity-maintained (40%-70%) rooms with a 12-h light/dark cycle. Water and food were provided freely. 5×10^6 HCT116 cells suspended in 100μl PBS were injected subcutaneously into the right flank of mice. When the tumor volume reached 100 to 150mm^3, the mice were randomized into 2 groups and injected intravenously with Disitamab vedotin (5mg/kg) for 3 weeks. Tumor sizes and body weight were recorded twice a week, and tumor volumes were determined according to the formula: tumor volume (mm^3)=length×$(width)^2\times0.5$.
Dosage form	5mg/kg; once a week for 3 weeks; i.v.
Applications	Disitamab vedotin treatment significantly inhibited tumor growth in the HCT116 mouse xenograft model.

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References:
[1] Li J, Shan K, Huang W, et al. The combination treatment of RC48 and STAT3 inhibitor acts as a promising therapeutic strategy for basal bladder cancer[J]. Frontiers in immunology, 2025, 15: 1432586.
[2] Liu H, Zhou D, Liu D, et al. Synergistic antitumor activity between HER2 antibody-drug conjugate and chemotherapy for treating advanced colorectal cancer[J]. Cell Death & Disease, 2024, 15(3): 187.

Background

Disitamab vedotin is an antibody-drug conjugate (ADC) targeting HER2 protein that is comprised of hertuzumab coupling monomethyl auristatin E (MMAE) via a cleavable linker [1]. Disitamab vedotin can kill tumor cells by targeting HER2-protein on the surface of tumor cells, as well as releasing small molecules in lysosomes after endocytosis[2]. Disitamab vedotin has been widely used to inhibit the growth of various drug-resistant cancer cells and tumors in xenograft mouse models[3].

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In vitro, Disitamab vedotin treatment for 48 hours significantly inhibited the growth of HT1376 cells, SW780 cells and RT4 cells was, with IC_{50} values of 158.3μg/ml, 155.7μg/ml and 132.2μg/ml, respectively[4]. Treatment with 50μg/ml Disitamab vedotin for 48 hours impeded the transition from G1 phase to S phase in A549 cells, increased the apoptosis rate and decreased the expression of FOXA1 protein[5]. Treatment with 100μg/ml Disitamab vedotin for 48 hours activated the cGAS-STING pathway in HCT116 cells, inducing cell cycle arrest[6].

In vivo, Disitamab vedotin treatment via weekly intravenous administration of 2.5mg/kg for 3 weeks significantly reduced the tumor volume in the CRC054 xenograft mouse model, without showing any obvious organ toxicity[7]. A single intravenous injection of 5mg/kg of Disitamab vedotin can inhibit tumor growth in the L-JIMT-1 lung metastasis mouse model and reduce vascular volume[8].

References:
[1] Shi F, Liu Y, Zhou X, et al. Disitamab vedotin: a novel antibody-drug conjugates for cancer therapy[J]. Drug Delivery, 2022, 29(1): 1335-1344.
[2] Jiang J, Li S, Shan X, et al. Preclinical safety profile of disitamab vedotin: a novel anti-HER2 antibody conjugated with MMAE[J]. Toxicology letters, 2020, 324: 30-37.
[3] Pourjamal N, Le Joncour V, Vereb G, et al. Disitamab vedotin in preclinical models of HER2-positive breast and gastric cancers resistant to trastuzumab emtansine and trastuzumab deruxtecan[J]. Translational oncology, 2025, 53: 102284.
[4] Li J, Shan K, Huang W, et al. The combination treatment of RC48 and STAT3 inhibitor acts as a promising therapeutic strategy for basal bladder cancer[J]. Frontiers in immunology, 2025, 15: 1432586.
[5] Zhao M, Zhang N, Wang Y, et al. FOXA1, induced by RC48, regulates HER2 transcription to enhance the tumorigenic capacity of lung cancer through PI3K/AKT pathway[J]. Journal of Cancer, 2024, 15(18): 5863.
[6] Wu X, Xu L, Li X, et al. A HER2-targeting antibody-MMAE conjugate RC48 sensitizes immunotherapy in HER2-positive colon cancer by triggering the cGAS-STING pathway[J]. Cell Death & Disease, 2023, 14(8): 550.
[7] Liu H, Zhou D, Liu D, et al. Synergistic antitumor activity between HER2 antibody-drug conjugate and chemotherapy for treating advanced colorectal cancer[J]. Cell Death & Disease, 2024, 15(3): 187.

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[8] Pourjamal N, Yazdi N, Halme A, et al. Comparison of trastuzumab emtansine, trastuzumab deruxtecan, and disitamab vedotin in a multiresistant HER2-positive breast cancer lung metastasis model[J]. Clinical & Experimental Metastasis, 2024, 41(2): 91-102.

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