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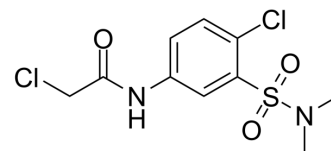
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GSTO1-IN-1

Cat. No.:	HY-111530
CAS No.:	568544-03-6
Molecular Formula:	C ₁₀ H ₁₂ Cl ₂ N ₂ O ₃ S
Molecular Weight:	311.18
Target:	Gutathione S-transferase
Pathway:	Metabolic Enzyme/Protease
Storage:	Powder -20°C 3 years 4°C 2 years In solvent -80°C 2 years -20°C 1 year



SOLVENT & SOLUBILITY

In Vitro

DMSO : ≥ 300 mg/mL (964.07 mM)
 * "≥" means soluble, but saturation unknown.

	Solvent Concentration	Mass	1 mg	5 mg	10 mg
Preparing Stock Solutions	1 mM		3.2136 mL	16.0679 mL	32.1357 mL
	5 mM		0.6427 mL	3.2136 mL	6.4271 mL
	10 mM		0.3214 mL	1.6068 mL	3.2136 mL

Please refer to the solubility information to select the appropriate solvent.

In Vivo

- Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
Solubility: ≥ 2.5 mg/mL (8.03 mM); Clear solution
- Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
Solubility: ≥ 2.5 mg/mL (8.03 mM); Clear solution
- Add each solvent one by one: 10% DMSO >> 90% corn oil
Solubility: ≥ 2.5 mg/mL (8.03 mM); Clear solution

BIOLOGICAL ACTIVITY

Description	GSTO1-IN-1 is a potent glutathione S-transferase omega 1 (GSTO1) inhibitor with an IC ₅₀ of 31 nM.
IC ₅₀ & Target	IC ₅₀ : 31 nM (GSTO1) ^[1]
In Vitro	GSTO1-IN-1 (C1-27) potently inhibits GSTO1 enzyme activity with an IC ₅₀ value of 31 nM. GSTO1-IN-1 also potently competes with 5-chloromethylfluorescein diacetate (CMFDA) for binding to recombinant protein, as well as endogenous GSTO1 in the

milieu of a soluble proteome. HCT116 cells treated with GSTO1-IN-1 also show a decrease in cell viability in a dose-dependent manner. GSTO1-IN-1 inhibits the clonogenic survival of HCT116 cells at sub-micromolar concentrations^[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

In Vivo

To test whether GSTO1-IN-1 had in vivo efficacy, its effects are evaluated in a human colon cancer cell line xenograft model. GSTO1-IN-1 (20-45 mg/kg) is administered as a single agent to nude mice bearing HCT116 xenografts. After 5 weeks of treatment, tumor growth is significantly inhibited in GSTO1-IN-1-treated mice compared with the vehicle-treated group ($P < 0.05$). GSTO1-IN-1 treatment is generally well tolerated by mice up to 45 mg/kg, with no overt signs of toxicity and no significant variations in average body weights throughout the duration of the study^[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

PROTOCOL

Cell Assay ^[1]

Cell proliferation is assessed by a MTT assay. Cancer cells H630, HT29 and HCT116 are seeded in 96-well microtitre plates and, after overnight attachment, treated with GSTO1 inhibitors (e.g., GSTO1-IN-1; 0.1, 1, 10 and 100 μ M). After 72 h, MTT solution (3 mg/mL; 20 mL) is added to each well and cells are incubated for 3 h at 37°C. After incubation, media from each well is removed and the dark blue formazan crystals formed by live cells are dissolved in DMSO (150 mL per well). The absorbance intensity is measured at 570 nm on a microplate reader. Cell viability after 24 h treatment is assessed using ApoTox-Glo triplex assay. At least three independent dose-response experiments with each concentration tested in triplicate are performed for each cell line^[1].

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Administration ^[1]

Mice^[1]

In the pilot study, HCT116 cells (1×10^6) in exponential phase are injected subcutaneously into the left flank of 8- to 10-week-old female nude mice (25-30 g). The perpendicular diameters of the tumors are measured three times weekly using standard calipers and tumour volumes are calculated. Tumors are allowed to grow to a volume of 50 mm³ and mice are randomized into control (n=5) and GSTO1-IN-1 (n=3) treatment groups. GSTO1-IN-1 is administered intraperitoneally (20 mg/kg per day) for the first 2 weeks on a 5 days on/2 days off schedule. The dose is then increased to 25 mg/kg per day for the next 23 days and further escalated by 5 mg/kg per day to a final dose of 45 mg/kg for the remaining duration of treatment. Tumor volumes and body weights are measured three times weekly to monitor tumor burden and weight loss during treatment. At the end of the experiment, animals are killed and tumor, kidney and liver are collected, fixed and paraffin embedded for histology^[1].

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- J Agric Food Chem. 2024 Mar 5.
- bioRxiv. June 28, 2021.

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REFERENCES

[1]. Ramkumar K, et al. Mechanistic evaluation and transcriptional signature of a glutathione S-transferase omega 1 inhibitor. Nat Commun. 2016 Oct 5;7:13084.

Caution: Product has not been fully validated for medical applications. For research use only.

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