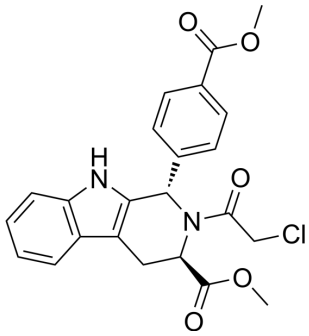
RSL3

Cat. No.:	HY-100218A
CAS No.:	1219810-16-8
Molecular Formula:	$C_{23}H_{21}ClN_2O_5$
Molecular Weight:	440.88
Target:	Glutathione Peroxidase; Ferroptosis; p62
Pathway:	Apoptosis; Metabolic Enzyme/Protease; Autophagy
Storage:	4°C, sealed storage, away from moisture * In solvent : -80°C, 2 years; -20°C, 1 year (sealed storage, away from moisture)



SOLVENT & SOLUBILITY

In Vitro

DMSO : 100 mg/mL (226.82 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	2.2682 mL	11.3410 mL	22.6819 mL
	5 mM	0.4536 mL	2.2682 mL	4.5364 mL
	10 mM	0.2268 mL	1.1341 mL	2.2682 mL

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

In Vivo

1. Add each solvent one by one: 50% PEG300 >> 50% saline
   Solubility: 20 mg/mL (45.36 mM); Suspended solution; Need ultrasonic
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: 5 mg/mL (11.34 mM); Suspended solution; Need ultrasonic
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (5.67 mM); Clear solution
4. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 2.08 mg/mL (4.72 mM); Clear solution
5. Add each solvent one by one: 10% DMF >> 90% corn oil
   Solubility: ≥ 0.56 mg/mL (1.27 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

RSL3 ((1S,3R)-RSL3) is an inhibitor of glutathione peroxidase 4 (GPX4) (ferroptosis activator), reduces the expression of GPX4 protein, and induces ferroptotic death of head and neck cancer cell. RSL3 increases the expression of p62 and Nrf2 and inactivates Keap1 in HN3-rslR cells[1].

IC_{50} & Target	Glutathione peroxidase 4[1]

In Vitro

RSL3 (0-8 μM, 72 hours) potently reduces the viability of HN3 cells, with IC_{50}s of 0.48 μM in HN3 and 5.8 μM in HN3-rslR cells, respectively[1].
RSL3 (0-8 μM, 24 hours) reduces the expression of GPX4 protein, increases the expression of p62 and Nrf2 and inactivates Keap1 in HN3-rslR cells[1].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Cell Viability Assay[1]

Cell Line:	HN3 cells, HN3-rslR cells
Concentration:	0-8 μM
Incubation Time:	72 hours
Result:	Showed IC_{50}s of 0.48 μM in HN3 and 5.8 μM in HN3-rslR cells, respectively[1].

Western Blot Analysis[1]

Cell Line:	HN3-rslR cells
Concentration:	0-8 μM
Incubation Time:	24 hours
Result:	Inhibited GPX4 expression, increased p62 and Nrf2 levels, and decreased Keap1 levels.

In Vivo

RSL3 (100 mg/kg, Intratumorally twice per week for 20 days) significantly inhibits the growth of tumor in combination with Trigonelline (HY-N0414) in mice bearing HN3R cells[1].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Ten-week-old athymic BALB/c male nude mice (nu/nu) bearing HN3R cells[1]
Dosage:	100 mg/kg in combination with trigonelline (50 mg/kg)
Administration:	Intratumorally twice per week for 20 days
Result:	Significantly reduced the volume of tumor combined with trigonelline in mice.

CUSTOMER VALIDATION

- Cell Discov. 2022 May 3;8(1):40.
- Adv Mater. 2024 Mar 24:e2401384.
- J Hematol Oncol. 2023 May 3;16(1):46.
- Cancer Discov. 2023 Apr 3;CD-22-0411.
- Nat Cancer. 2022 Apr;3(4):471-485.

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REFERENCES

[1]. Shin D, et al. Nrf2 inhibition reverses resistance to GPX4 inhibitor-induced ferroptosis in head and neck cancer. Free Radic Biol Med. 2018 Dec;129:454-462.

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