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Lieferung & Zahlungsart

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Zuschläge

- Mindermengenzuschlag
- Trockeneiszuschlag
- Gefahrgutzuschlag
- Expressversand

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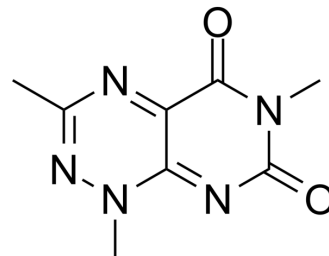
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3-Methyltoxoflavin

Cat. No.:	HY-111117		
CAS No.:	32502-62-8		
Molecular Formula:	C ₈ H ₉ N ₅ O ₂		
Molecular Weight:	207.19		
Target:	PDI		
Pathway:	Cell Cycle/DNA Damage; 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 : 50 mg/mL (241.32 mM; ultrasonic and warming and heat to 60°C)

Concentration	Solvent	Mass		
		1 mg	5 mg	10 mg
Preparing Stock Solutions	1 mM	4.8265 mL	24.1324 mL	48.2649 mL
	5 mM	0.9653 mL	4.8265 mL	9.6530 mL
	10 mM	0.4826 mL	2.4132 mL	4.8265 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: 5 mg/mL (24.13 mM); Clear solution; Need ultrasonic
- Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
Solubility: 5 mg/mL (24.13 mM); Clear solution; Need ultrasonic
- Add each solvent one by one: 10% DMSO >> 90% corn oil
Solubility: ≥ 2.5 mg/mL (12.07 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

3-Methyltoxoflavin is a potent Protein disulfide isomerase (PDI) inhibitor, with an IC₅₀ of 170 nM.

IC₅₀ & Target

IC₅₀: 170 nM (PDI)^[1].

In Vitro

3-Methyltoxoflavin is a potent Protein disulfide isomerase (PDI) inhibitor, with an IC₅₀ of 170 nM. 3-Methyltoxoflavin is toxic in a panel of human glioblastoma cell lines. From the screen, 3-Methyltoxoflavin emerges as the most cytotoxic inhibitor of PDI. Bromouridine labeling and sequencing (Bru-seq) of nascent RNA reveals that 3-Methyltoxoflavin induces Nrf2

antioxidant response, ER stress response, and autophagy. Specifically, 3-Methyltoxoflavin upregulates heme oxygenase 1 and SLC7A11 transcription and protein expression and represses PDI target genes such as TXNIP and EGR1. Interestingly, 3-Methyltoxoflavin-induced cell death does not proceed via apoptosis or necrosis, but by a mixture of autophagy and ferroptosis^[1].

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

PROTOCOL

Cell Assay^[1]

The colon cancer cells are seeded in duplicate in 96-well plates at 7000-10000 cells per well. For the combination therapies, NAC is added to the well at the same time as 3-Methyltoxoflavin (35G8) (24 hours after plates are seeded), and Z-VAD-FMK and Necrostatin-1 are added to the well 1 hour prior to 3-Methyltoxoflavin addition. Cell growth inhibition is assessed by the cell viability rate. Cell viability is determined with the MTT assay. U87MG cells are seeded at 5000 cells per well in 96-well plates. Deferoxamine is added to cells in a five-point, three-fold dilution series from 400 μ M. 3-Methyltoxoflavin is added immediately after in a five-point, three-fold dilution series from 100 μ M. Cells are incubated with compounds for 12 hours^[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- Inflammation. 2023 Dec 6.

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REFERENCES

[1]. Kyani A, et al. Discovery and Mechanistic Elucidation of a Class of Protein Disulfide Isomerase Inhibitors for the Treatment of Glioblastoma. ChemMedChem. 2018 Jan 22;13(2):164-177.

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

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