Thapsigargin

Cat. No.: HY-13433

CAS No.: 67526-95-8

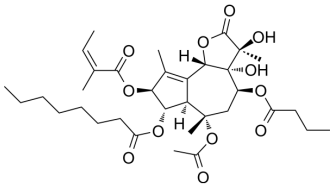
Molecular Formula: $C_{34}H_{50}O_{12}$

Molecular Weight: 650.75

Target: Calcium Channel; Apoptosis; SARS-CoV

Pathway: Membrane Transporter/Ion Channel; Neuronal Signaling; Apoptosis; Anti-infection

Storage: -20°C, sealed storage, away from moisture and light
* In solvent : -80°C, 6 months; -20°C, 1 month (sealed storage, away from moisture and light)



SOLVENT & SOLUBILITY

In Vitro

DMSO : 50 mg/mL (76.83 mM; Need ultrasonic)

H_2O : < 0.1 mg/mL (ultrasonic;warming;heat to 60°C) (insoluble)

Preparing Stock Solutions

Concentration \ Solvent \ Mass	1 mg	5 mg	10 mg
1 mM	1.5367 mL	7.6834 mL	15.3669 mL
5 mM	0.3073 mL	1.5367 mL	3.0734 mL
10 mM	0.1537 mL	0.7683 mL	1.5367 mL

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

In Vivo

1. Add each solvent one by one: 10% DMSO >> 90% saline
   Solubility: 5 mg/mL (7.68 mM); Suspended solution; Need ultrasonic
2. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 2.08 mg/mL (3.20 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.08 mg/mL (3.20 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Thapsigargin, an endoplasmic reticulum (ER) stress inducer, is an inhibitor of microsomal Ca^{2+}-ATPase. Thapsigargin efficiently inhibits coronavirus (HCoV-229E, MERS-CoV, SARS-CoV-2) replication in different cell types[1][2][3][4][5].

IC_{50} & Target

Ca^{2+}-ATPase[1]

In Vitro

Thapsigargin (0.001- 1 μM; for 2 and 4 days) arrests cell proliferations in MH7A human rheumatoid arthritis synovial cells in a time- and dose-dependent manner[2].

Thapsigargin (0.001- 1 μM; for 2 and 4 days) induces cell apoptosis in MH7A cells in a time- and dose-dependent manner[2].
Thapsigargin (0.001- 1 μM; for 2 and 4 days) impairs mTOR activity and leads to cyclin D1 expressions in MH7A cells[2].
Thapsigargin inhibits Ca^{2+} entry into human neutrophil granulocytes[1].
Thapsigargin inhibits the carbachol-evoked $[Ca^{2+}]$i-transients with (IC_{50}=0.353 nM) or without (IC_{50}=0.448 nM) a KCl-prestimulation, but an additional small component, with a much lower sensitivity (IC_{50}=4814 nM), is observed in the absence of a KCl-prestimulation. In contrast, the KCl-evoked $[Ca^{2+}]$i-transients displayed only one component with a very low sensitivity to Thapsigargin in both absence (IC_{50}=3343 nM) and presence (IC_{50}=6858 nM) of a carbachol-prestimulation[3].
Thapsigargin also phosphorylate p38 MAPK by Ca^{2+} influx through SOCE, leading to suppression of TNF-α-induced NF-κB phosphorylation[6].

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

Cell Proliferation Assay[2]

Cell Line:	MH7A human rheumatoid arthritis synovial cells
Concentration:	0.001, 0.1, and 1 μM
Incubation Time:	For 2 and 4 days
Result:	Arrested cell proliferations in a time- and dose-dependent manner.

Apoptosis Analysis[2]

Cell Line:	MH7A human rheumatoid arthritis synovial cells
Concentration:	0.001, 0.1, and 1 μM
Incubation Time:	For 2 and 4 days
Result:	Induces cell apoptosis in a time- and dose-dependent manner.

Western Blot Analysis[2]

Cell Line:	MH7A human rheumatoid arthritis synovial cells
Concentration:	0.001, 0.1, and 1 μM
Incubation Time:	For 2 and 4 days
Result:	Impairs mTOR activity and leads to cyclin D1 expressions

In Vivo

Thapsigargin (Injection; 0.25 ug/g, 0.5 ug/g and 1 ug/g; 24 hours) significant increases of 2 to 5-fold in chemokine and pro-inflammatory expression. Thapsigargin is more sensitive to inducing a systemic immune response[4].

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

Animal Model:	Male Balb/c mice (20-25 g)[4]
Dosage:	0.25 ug/g, 0.5 ug/g and 1 ug/g
Administration:	Injection; 24 hours
Result:	Increased of 2 to 5-fold in chemokine and pro-inflammatory expression.

CUSTOMER VALIDATION

- Nat Immunol. 2023 Dec 7.
- ACS Nano. 2024 Jan 10.
- ACS Nano. 2021 Jun 22;15(6):10640-10658.
- Nat Commun. 2023 Nov 1;14(1):6982.
- Adv Sci (Weinh). 2022 Oct 10;e2203831.

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REFERENCES

[1]. Junsuke Uwada, et al. Store-operated calcium entry (SOCE) contributes to phosphorylation of p38 MAPK and suppression of TNF-α signalling in the intestinal epithelial cells. Cell Signal. 2019 Nov;63:109358.

[2]. Geiszt M, et al. Thapsigargin inhibits Ca^{2+} entry into human neutrophil granulocytes. Biochem J. 1995 Jan 15;305 (Pt 2):525-8.

[3]. Wang H, et al. Effects of thapsigargin on the proliferation and survival of human rheumatoid arthritis synovialcells. ScientificWorldJournal. 2014 Feb 9;2014:605416.

[4]. Garavito-Aguilar ZV, et al. Differential thapsigargin-sensitivities and interaction of Ca2+ stores in human SH-SY5Y neuroblastoma cells. Brain Res. 2004 Jun 18;1011(2):177-86.

[5]. Abdullahi A, et al. Modeling Acute ER Stress in Vivo and in Vitro. Shock. 2017 Apr;47(4):506-513.

[6]. Mohammed Samer Shaban, et al. Inhibiting coronavirus replication in cultured cells by chemical ER stress. bioRxiv 2020.08.26.266304;

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