Valrubicin

Cat. No.: HY-13772

CAS No.: 56124-62-0

Molecular Formula: $C_{34}H_{36}F_3NO_{13}$

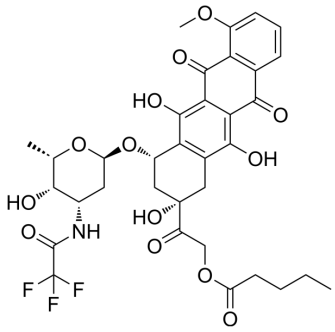
Molecular Weight: 724

Target: PKC; Antibiotic

Pathway: Epigenetics; TGF-beta/Smad; Anti-infection

Storage:

Powder	-20°C	3 years
	4°C	2 years
In solvent	-80°C	6 months
	-20°C	1 month



SOLVENT & SOLUBILITY

In Vitro

DMSO : 125 mg/mL (172.65 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	1.3812 mL	6.9061 mL	13.8122 mL
	5 mM	0.2762 mL	1.3812 mL	2.7624 mL
	10 mM	0.1381 mL	0.6906 mL	1.3812 mL

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

In Vivo

1. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 2.17 mg/mL (3.00 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Valrubicin is a chemotherapy agent, inhibits TPA- and PDBu-induced PKC activation with IC_{50}s of 0.85 and 1.25 μM, respectively, and has antitumor and antiinflammatory activity.

IC_{50} & Target

TPA-activated PKC	PDBu-activated PKC
0.85 μM (IC_{50})	1.25 μM (IC_{50})

In Vitro

Valrubicin (AD 32) is a chemotherapy agent, inhibits TPA- and PDBu-induced PKC activation with IC_{50}s of 0.85 and 1.25 μM, respectively. Valrubicin inhibits the binding of [^{3}H]PDBu to PKC. Therefore, Valrubicin competes with the tumor promoter for the PKC binding site and prevents the latter from both interacting with the phospholipid and binding to PKC[1].
Valrubicin shows cytotoxic activity against squamous cell carcinoma (SCC) cell line colony formation, with IC_{50}s and IC_{90}s of 8.24 ± 1.60 μM and 14.81 ± 2.82 μM for UMSCC5 cells, 15.90 ± 0.90 μM, 29.84 ± 0.84 μM for UMSCC5/CDDP‡ cells, and 10.50 ± 2.39 μM, 19.00 ± 3.91 μM for UMSCC10b cells, respectively. Moreover, Valrubicin in combination with radiation enhances the

	cytotoxicity[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	Valrubicin (3, 6, or 9 mg) reduces tumor growth at week 3 by intratumoral jection in hamster. Valrubicin (6 mg) combined with minimally cytotoxic irradiation (150, 250, or 350 cGy) causes significant tumor shrinkage in hamster[2]. Valrubicin (0.1 μg/μL) significantly reduces the number of infiltrating neutrophils in biopsies challenged with TPA at 24 h and attenuates chronic inflammation in mice. Valrubicin also decreases the expression levels of inflammatory cytokines in the acute model [3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

PROTOCOL

Cell Assay [2]	UMSCC5 cells exposed to Valrubicin (2 μM for 3 h), a single dose of radiation (400 cGy), or the combined treatment are cultured for a further 12, 24, or 48 hours. At these times, the cells are collected by trypsinization (0.25%), washed in phosphate-buffered saline (PBS), and fixed at 5×10^6 cells/mL with 95% ethanol. Cells are incubated with ribonuclease (50 μg; 70-90 Kunitz units/mg for 30 min), and the resulting pellet resuspended in and incubated with propidium iodide (0.05 mg/mL for 10 min). The DNA content of the samples is determined by flow cytometry according to standard technique[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
Animal Administration [2]	Hamsters[2] Hamsters with cheek pouch tumors of 100 mm^2 are randomly assigned to one of five treatment groups. Momentarily anesthetized animals each receives once a week × 3 injections (27 g × 0.5-inch needle: 0.1 mL administered slowly to the base of the lesion) of Valrubicin (3, 6, or 9 mg) or drug vehicle (Cremophor: alcohol;1:1 by volume; NCl diluent 12). A further group of animals receives anesthesia but no direct tumor treatment (control). Individual tumor sizes are measured with calipers at weekly intervals for 4 weeks, at which time the animals are sacrificed[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

REFERENCES

[1]. Chuang LF, et al. Activation of human leukemia protein kinase C by tumor promoters and its inhibition by N-trifluoroacetyladriamycin-14-valerate (AD 32). Biochem Pharmacol. 1992 Feb 18;43(4):865-72.

[2]. Wani MK, et al. Rationale for intralesional valrubicin in chemoradiation of squamous cell carcinoma of the head and neck. Laryngoscope. 2000 Dec;110(12):2026-32.

[3]. Hauge E, et al. Topical valrubicin application reduces skin inflammation in murine models. Br J Dermatol. 2012 Aug;167(2):288-95.

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