Trilexium

Cat. No.: HY-157800

CAS No.: 1983180-82-0

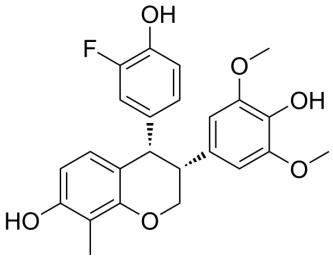
Molecular Formula: $C_{24}H_{23}FO_6$

Molecular Weight: 426.43

Target: Apoptosis; PARP; Caspase; Microtubule/Tubulin

Pathway: Apoptosis; Cell Cycle/DNA Damage; Epigenetics; Cytoskeleton

Storage: -20°C, protect from light
* In solvent : -80°C, 6 months; -20°C, 1 month (protect from light)



SOLVENT & SOLUBILITY

In Vitro

DMSO : 300 mg/mL (703.52 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	2.3451 mL	11.7253 mL	23.4505 mL
	5 mM	0.4690 mL	2.3451 mL	4.6901 mL
	10 mM	0.2345 mL	1.1725 mL	2.3451 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: ≥ 5 mg/mL (11.73 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 5 mg/mL (11.73 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Trilexium (TRX-E-009-1) is a third-generation of benzopyran structurally related to TRX-E-002-1 (HY-114250). Trilexium increases p21 protein expression and induces apoptosis. Trilexium depolymerizes microtubules. Trilexium shows broad anti-cancer activity[1][2].

In Vitro

Trilexium shows activity against a broad range of cancer types. Of the 240 cell lines assessed in the Eurofin's Oncopanel, 10 cell lines shows IC_{50} > 30 μM, of the remaining 230 cell lines, the average IC_{50} is 0.428 μM[1].
Trilexium (300 nM, 4 h) disrupts microtubule networks in Hela cells[1].
Trilexium (1-5 μM) leads to increased protein expression of p21, c-PARP, and c-Caspase 3 in HSJD-DIPG007 cells[2].
Trilexium (0-2 μM) restores H3K27 trimethylation and increases H3K27 acetylation in HSJD-DIPG007 cells[2].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

In Vivo

Trilexium (5-60 mg/kg, IV daily for 15 days) significantly reduces tumour volume in vivo[1].
Trilexium (80 mg/kg, IV daily for 5 days) disrupts microtubules in vivo[1].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	C57/BL6 mice (n = 8 mice per group, subcutaneous mouse xenograft model of BRAF mutant (BRAFV600E) melanoma (A375))[1]
Dosage:	5 and 60 mg/kg
Administration:	IV (tail vein injection), daily for 15 days
Result:	Resulted in a tumour growth inhibition at 60 mg/kg. Significantly improved survival at 60 mg/kg.

Animal Model:	A375 xenografts mice[1].
Dosage:	80 mg/kg
Administration:	IV, daily for 5 days
Result:	Resulted in a loss of the long microtubule filaments in favour of shorter filaments and dispersed tubulin staining.

REFERENCES

[1]. Stevenson AJ, et al. Mechanism of action of the third generation benzopyrans and evaluation of their broad anti-cancer activity in vitro and in vivo. Sci Rep. 2018 Mar 23;8(1):5144.

[2]. Ehteda A, et al. Microtubule-Targeting Combined with HDAC Inhibition Is a Novel Therapeutic Strategy for Diffuse Intrinsic Pontine Gliomas. Mol Cancer Ther. 2023 Dec 1;22(12):1413-1421.

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