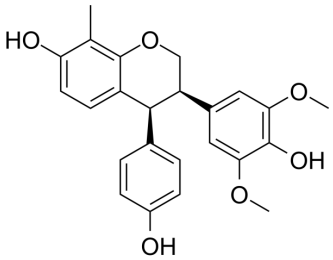
Cantrixil

Cat. No.:	HY-114250		
CAS No.:	2135511-22-5		
Molecular Formula:	$C_{24}H_{24}O_6$		
Molecular Weight:	408.44		
Target:	Apoptosis		
Pathway:	Apoptosis		
Storage:	Powder	-20°C	3 years
		4°C	2 years
	In solvent	-80°C	6 months
		-20°C	1 month



BIOLOGICAL ACTIVITY

Description

Cantrixil (TRX-E-002-1), an active enantiomer of TRX-E-002, is a second-generation super-benzopyran (SBP) compound. Cantrixil increases phosphorylated c-Jun levels resulting in caspase-mediated apoptosis in ovarian cancer cells. Cantrixil has potent pan anti-cancer activity against a broad range of cancer phenotypes[1][2].

In Vitro

TRX-E-002-1 shows broad cytotoxic activity against ovarian, prostate and lung cancer cells, with IC_{50} values of ≤0.1 μM (SK-OV-3, JAM, OVCAR-3 cells: IC_{50}=0.023-0.065 μM; DU145, PC3; C4-2B cells: IC_{50}=0.014-0.096 μM; A549 cells: IC_{50}=0.058 μM). Activity in pancreatic and colorectal cancer cells and glioblastoma cells are more variable[1].
Cantrixil (0.2 μM; 2-24 hours) shows high levels of phosphorylated c-Jun (p-c-Jun) and low levels of phosphorylated-ERK (p-ERK)[2].
Cantrixil (2.45 μM; 2-24 hours) induces a significant increase in both caspase-3/7 and caspase-9 activity at 16 and 24 hours[2].

TRX-E-002-1 inhibits multiple cytochrome P450 drug-metabolizing enzymes, including CYP2C9, CYP2C8, CYP2C19, CYP2B6, CYP3A4, CYP2D6, CYP2A6 and CYP1A2. IC_{50} values ranges from 1.5 to 75 μM (612-30,600 ng/mL)[1].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Western Blot Analysis[2]

Cell Line:	Ovarian cancer stem cells (OCSCs)
Concentration:	0.2 μM
Incubation Time:	2, 4, 8, 16, 24 hours
Result:	Showed higher levels of phosphorylated c-Jun (p-c-Jun) and lower levels of phosphorylated-ERK (p-ERK). Showed a time-dependent increase in p-c-Jun accompanied by a time-dependent increase in total c-Jun.

In Vivo

TRX-E-002-1 (100 mg/kg/day; IP; for 13-14 days) significantly inhibits tumour growth in disseminated ovarian cancer mouse model[1].
TRX-E-002-1 (100 mg/kg/day; IP; for 4 weeks) inhibits tumour growth and reduces terminal tumour burden by 77% in the recurrent ovarian cancer mouse model[1].
TRX-E-002-1 (100 mg/kg/day; IP; for 18 days) significantly reduces terminal pancreatic tumour burden in a mouse model of

pancreatic cancer (human Panc-1 pancreatic tumour cells implanted orthotopically into female NOD-SCID mice)[1]. TRX-E-002-1 (100 mg/kg; IP) has a $T_{1/2}$ of 2.5 hours, a C_{max} of 8355 ng/mL and an $AUC_{0-\infty}$ of 40600 ng•h/mL[1].

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

Animal Model:	Disseminated ovarian cancer mouse model[1]
Dosage:	100 mg/kg (dissolved in 20% SBECD)
Administration:	IP; once daily; for 13-14 days
Result:	Significantly inhibited tumour growth and reduced excised tumour weight at termination by 50-72%.

Animal Model:	Male female Sprague-Dawley rats[1]
Dosage:	100 mg/kg (Pharmacokinetic Analysis)
Administration:	IP
Result:	Had a $T_{1/2}$ of 2.5 hours, a C_{max} of 8355 ng/mL and an $AUC_{0-\infty}$ of 40600 ng•h/mL.

REFERENCES

[1]. Muhammad Wasif Saif, et al. Pharmacology and toxicology of the novel investigational agent Cantrixil (TRX-E-002-1). Cancer Chemother Pharmacol. 2017 Feb;79(2):303-314.

[2]. Ayesha B Alvero, et al. TRX-E-002-1 Induces c-Jun-Dependent Apoptosis in Ovarian Cancer Stem Cells and Prevents Recurrence In Vivo. Mol Cancer Ther. 2016 Jun;15(6):1279-90.

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