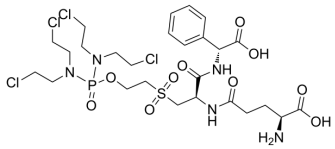
Canfosfamide

Cat. No.:	HY-16124
CAS No.:	158382-37-7
Molecular Formula:	$C_{26}H_{40}Cl_4N_5O_{10}PS$
Molecular Weight:	787.47
Target:	DNA-PK; Apoptosis
Pathway:	Cell Cycle/DNA Damage; PI3K/Akt/mTOR; Apoptosis
Storage:	Please store the product under the recommended conditions in the Certificate of Analysis.



BIOLOGICAL ACTIVITY

Description	Canfosfamide (TLK-286, TER286) is a glutathione analogue prodrug that is activated by glutathione S-transferase P1-1 and induces apoptosis. Canfosfamide also inhibits the catalytic kinase activity of DNA-dependent protein kinase (DNA-PK). Canfosfamide produces an anticancer alkylating agent and a glutathione derivative after activation. Canfosfamide can be used to research malignancies[1][2][3].
IC_{50} & Target	IC_{50}: ~1 μM (DNA-PK)[3]

In Vitro

Canfosfamide (TLK-28) inhibit the catalytic kinase activity of purified DNA-dependent protein kinase (DNA-PK) with an IC_{50} value of ~1 μM, but causes minimal direct damage to DNA[2].
Canfosfamide (TER286) inhibits MCF-7, NIH-3T3 and M7609 in a dose-dependent manner[3].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Cell Proliferation Assay[3]

Cell Line:	MCF-7, NIH-3T3 and M7609
Concentration:	0-100 μM
Incubation Time:	Co-incubated for 2 h then removed and incubated for 5 days in MCF-7; co-incubated for 10 days in NIH-3T3; co-incubated for 48 h in M7609
Result:	Inhibited these cancer cell lines in a dose-dependent manner.

In Vivo

Canfosfamide (TER286) exhibits more effective antitumor effect in more frequent administration[3].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	BALB/c nude mice (s.c. with tumors of M7609, MX-1 human breast tumor, lung tumor[3]
Dosage:	150 mg/kg for M7609 xenografts; 400 mg/kg or 200 mg/kg for other xenografts
Administration:	i.v., single dosage for M7609 xenograft; i.p., single dosage for other xenografts; i.p., daily for 5 days
Result:	The best response to TER286 was for the MX-1 human breast tumor treated with an

	aggressive regimen (daily for 5 days), under which nearly all of the tumors were either severely growth inhibited or substantially regressed.

REFERENCES

[1]. Dourado DF, et al. Mechanism of glutathione transferase P1-1-catalyzed activation of the prodrug canfosfamide (TLK286, TELCYTA). Biochemistry. 2013 Nov 12;52(45):8069-78.

[2]. Tew KD. TLK-286: a novel glutathione S-transferase-activated prodrug. Expert Opin Investig Drugs. 2005 Aug;14(8):1047-54.

[3]. Morgan AS, et al. Tumor efficacy and bone marrow-sparing properties of TER286, a cytotoxin activated by glutathione S-transferase. Cancer Res. 1998 Jun 15;58(12):2568-75.

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