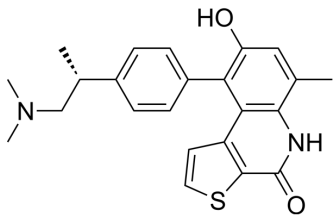
OTS964

Cat. No.:	HY-19718
CAS No.:	1338542-14-5
Molecular Formula:	$C_{23}H_{24}N_2O_2S$
Molecular Weight:	392.51
Target:	TOPK; CDK; Apoptosis
Pathway:	Cell Cycle/DNA Damage; Apoptosis
Storage:	Please store the product under the recommended conditions in the Certificate of Analysis.



BIOLOGICAL ACTIVITY

Description

OTS964 is an orally active, high affinity and selective TOPK inhibitor with an IC_{50} of 28 nM[1]. OTS964 is also a potent inhibitor of the cyclin-dependent kinase CDK11, which binds to CDK11B with a K_d of 40 nM[2].

IC_{50} & Target

TOPK	CDK11B
28 nM (IC_{50})	40 nM (Kd)

In Vitro

OTS964 (10 nM; 48 hours) suppresses cancer cell proliferation[1].
OTS964 (10 nM; 48 hours) increases cancer cell death[1].
OTS964 (0.1-2 μM; 24 and 48 hours) increases the expression of LC3-II and decreases the expression of P62, both in a dose-dependent manner[3].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Cell Proliferation Assay[1]

Cell Line:	LU-99 cells
Concentration:	10 nM
Incubation Time:	48 hours
Result:	Suppressed cancer cell proliferation.

Apoptosis Analysis[1]

Cell Line:	LU-99 cells
Concentration:	10 nM
Incubation Time:	48 hours
Result:	Increased cancer cell death.

Western Blot Analysis[3]

Cell Line:	Hs683 cells, H4 cells

Concentration:	0.1, 1, 2 μM
Incubation Time:	24 and 48 hours
Result:	Increased the expression of LC3-II and decreased the expression of P62, both in a dose-dependent manner.

In Vivo

OTS964 (intravenously; 40 mg/kg on days 1, 4, 8, 11, 15, and 18) makes tumors shrinking even after the treatment and finally revealing complete regression[1].
OTS964 (oral administration; 50 or 100 mg/kg/day for 2 weeks) achieves complete tumor regression[1].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Nude mice bearing LU-99 lung cancer cells[1]
Dosage:	40 mg/kg
Administration:	Intravenously; on days 1, 4, 8, 11, 15, and 18
Result:	The tumors continued shrinking even after the treatment and finally revealed complete regression.

Animal Model:	Nude mice bearing LU-99 lung cancer cells[1]
Dosage:	50 or 100 mg/kg
Administration:	Oral administration; once every day for 2 weeks
Result:	Achieved complete tumor regression.

CUSTOMER VALIDATION

- Nature. 2022 Sep;609(7928):829-834.
- Cell. 2021 Jun 10;184(12):3143-3162.e32.
- Adv Sci (Weinh). 2024 Feb 2:e2308496.
- J Eur Acad Dermatol Venereol. 2023 Dec 22.
- Cell Death Dis. 2019 Aug 5;10(8):583.

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REFERENCES

[1]. Matsuo Y , et al. TOPK inhibitor induces complete tumor regression in xenograft models of human cancerthrough inhibition of cytokinesis. Sci Transl Med. 2014 Oct 22;6(259):259ra145.

[2]. Lin A, et al. Off-target toxicity is a common mechanism of action of cancer drugs undergoing clinical trials. Sci Transl Med. 2019 Sep 11;11(509).

[3]. Lu H, et al. TOPK inhibits autophagy by phosphorylating ULK1 and promotes glioma resistance to TMZ. Cell Death Dis. 2019 Aug 5;10(8):583.

Caution: Product has not been fully validated for medical applications. For research use only.

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