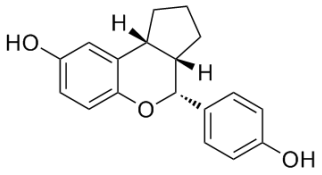
Erteberel

Cat. No.:	HY-18295
CAS No.:	533884-09-2
Molecular Formula:	$C_{18}H_{18}O_3$
Molecular Weight:	282.33
Target:	Estrogen Receptor/ERR
Pathway:	Others
Storage:	Please store the product under the recommended conditions in the COA.



BIOLOGICAL ACTIVITY

Description	Erteberel (LY500307) is a potent and selective estrogen receptor beta (**ERβ**) inhibitor with **K_i** and **EC_{50}** of 1.54 nM and 3.61 nM, respectively[1]. Anti-tumor activities[2].

IC_{50} & Target	ERβ 1.54 nM (Ki)	ERβ 3.61 nM (EC50)

In Vitro

Treatment with Erteberel (0.25-10 μM, 72 hours) significantly reduces the proliferation of GBM cells with no activity on normal astrocytes in vitro[2].

Erteberel promotes apoptosis of GBM cells. Erteberel modulated several pathways related to apoptosis, cell cycle, and DNA damage response[2].

Erteberel (0-1000 μM) sensitizes GBM cells to several FDA-approved chemotherapeutic drugs including cisplatin, lomustine and temozolomide[2].

Cell Viability Assay[2]

Cell Line:	U87, U251,T98G and normal astrocytes
Concentration:	0.25, 0.5, 1, 2, 4, 6, 8, and 10 μM
Incubation Time:	72 h
Result:	Treatment with Erteberel significantly reduces the viability of various GBM cell lines in adose-dependent manner. In contrast, viability of normal astrocytes is not affected at the tested doses, suggesting that Erteberel has tumor cell–specific activity[2].

In Vivo

Erteberel (5 mg/Kg body weight/day, oral, 28 days) treatment significantly reduces tumor growth and promotes apoptosis of GBM tumors in an orthotopic model[2].

Erteberel (5 mg/Kg body weight/day, oral, 40-50 days) treatment improves the overall survival of tumor-bearing mice in the GL26 syngeneic glioma model[2].

Animal Model:	Athymic mice (5-7 weeks) inoculated with OVCAR-3 cells[2]
Dosage:	5mg/Kg body weight
Administration:	Oral, daily for 28 days
Result:	Immunohistochemical analysis reveals that Erteberel treatment significantly reduces the number of proliferation marker Ki-67-positive cells and increases the number of TUNEL-positive apoptotic cells[2].

REFERENCES

[1]. Zhao L, et al. Pharmacological activation of estrogen receptor beta augments innate immunity to suppress cancer metastasis. Proc Natl Acad Sci U S A. 2018 Apr 17;115(16):E3673-E3681.

[2]. Sareddy GR, et al. Selective Estrogen Receptor β Agonist LY500307 as a Novel Therapeutic Agent for Glioblastoma. Sci Rep. 2016 Apr 29;6:24185.

Tel: 609-228-6898 Fax: 609-228-5909 E-mail: tech@MedChemExpress.com
Address: 1 Deer Park Dr, Suite Q, Monmouth Junction, NJ 08852, USA